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A TEXT-BOOK

OF THE

PRACTICE OF MEDICINE

BY

JAMES M. ANDERS, M.D., PH.D., LL.D.
Professor of the Practice of Medicine and of Clinical Medicine in the Medico-Chirurgical College,
Philadelphia; Attending Physician to the Medico-Chirurgical and
Samaritan Hospitals, Philadelphia, Etc.

ILLUSTRATED

FIFTH EDITION, THOROUGHLY REVISED

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PREFACE TO THE FIFTH EDITION.

It is especially gratifying to the author to be called on for another revision of this work within less than a year since the fourth edition was issued. In the preparation of this edition, the original purpose to make the book pre-eminently practical has been the sole point of vantage, and to this end theoretic considerations have been, as in previous issues, subordinated. The most thorough and extensive changes have been made in connection with the large group of Infectious Diseases. No pains have been spared to present modern views, derived from clinical experience and critical bedside observation, as well as newly discovered scientific facts, tempered, it is hoped, with commendable conservatism. Especial care has been bestowed upon the etiology, including bacteriology, inductive diagnosis, and the details of treatment, in the belief that these phases of the subjects treated form the groundwork for an intelligent and successful pursuit of the science and art of medicine.

The etiology and mode of transmission of Malaria and of Yellow Fever have been almost entirely rewritten. Certain affections of growing importance, as Diphtheritic Dysentery and Parasitic Hemoptysis, have been recast and more fully discussed. A few new articles have been introduced; for example, Fatty Infiltration of the Heart, Streptococcus Pneumonia, and Acute Diffuse Interstitial Nephritis. Among leading infections that have received careful and thorough revision are Typhoid Fever, Malaria, Cerebro-spinal Meningitis, Lobar Pneumonia, Influenza, Variola, Chronic Tuberculosis, and Hydrophobia. The entire work, moreover, has been carefully scrutinized and brought into harmony with the most recent developments in practical medicine.

The original plan of the work, based on the systematic, orderly, and convenient arrangement of the individual themes and their subdivisions, has been retained and in numerous places further perfected. Thus changed and improved, it is hoped that the present issue will be found deserving of no less professional favor than that enjoyed by the previous ones.

Dr. Joseph Sailer has carefully revised the section on Nervous Diseases. My best thanks are due to Dr. L. Napoleon Boston and Dr. W. Z. Anders for kind aid rendered, as well as to the publishers for courtesies extended and the efficient manner in which they have executed their work.

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PREFACE.

THIS work is meant to introduce the student to the present state of our knowledge of the practice of medicine in general and of the diagnosis, differential diagnosis, and treatment of disease in particular. The historic development of the subjects treated has been either briefly given or intendedly omitted, since this scarcely falls within the scope of a practical treatise on medicine. Although the book as a whole is submitted to the critical judgment of a learned profession, it may be pardonable to emphasize, provisionally, a few features pertaining to the mode of treating the separate subjects, or the arrangement of the material under the latter—to indicate some of the more salient lineaments, so to speak, in the general design. Since in medical schools it is taught from a separate chair, the pathology (special) of the individual affections has almost invariably been taken up before the etiology; from this point the student will find the story of each affection a continuous one. The practitioner, however, must ever aim to associate the clinical symptoms with the morbid lesions.

Under special etiology the bacteriology has been prominently mentioned, since we owe to it the rapid progress that is being made in the study of the causation of disease.

The differential diagnosis has in many instances been tabulated—an ear-mark that I confidently believe will be found especially helpful. It may be stated that not less than fifty-six diagnostic tables are scattered throughout the work, and that by far the greater number of these are my own.

Such formulæ have been introduced into the text, and only such, as a more or less extended experience has shown to be possessed of real therapeutic importance. Whilst these, and all additional points relating to the treatment of the single affections, may serve as guides, particularly to the beginner, I fully appreciate how often the practising physician is placed in a position in which he is compelled to form a therapeutic judgment for himself. Whenever the dosage is stated, the metric equivalent is placed in parentheses, the number of grams being stated in round numbers (ʒj—4.0; ℥j—32.0) in order to render it of greater practical value. In all instances, however, in which this would involve an important difference in quantity the exact decimal figures are given. A considerable variation from the usual classification of diseases may be observed, but this is accounted for in the text wherever it occurs.

Preference has been given to the modern orthography and terminology, not only because it is more euphonious, but also because of its adoption by the standard lexicographers.

I have gleaned without stint from medical literature with a view to bringing the book up to date, and if I have failed to give full credit in every instance, my grateful acknowledgments are here due and are cheerfully made. The chief results of my personal experience and observation, extending over a period of two decades, and derived from both hospital and private practice, will also be found upon these pages.

I wish to thank Prof. W. C. Hollopeter, who has written some of the articles upon the diseases of children, as measles, chicken-pox, mumps, whooping-cough, and the acute diarrheas, and who has kindly aided in the preparation of those upon diphtheria and scarlatina.

My cordial thanks are due also to Dr. C. L. Furbush for kind aid in preparing some of the illustrations, to Doctors Robert N. Willson, Howard S. Anders, and Geo. W. Pfromm for valuable assistance while the work was passing through the press, and to Dr. A. M. Davis for preparing the index.

JAMES M. ANDERS.

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PART I.

INFECTIOUS DISEASES.

TYPHOID FEVER.

(*Enteric Fever; Abdominal Typhus; Ileo-typhoid; Nerven Fieber.*)

Definition.—An acute infectious disease of which the definitive cause is the specific bacillus of Eberth. It is characterized, pathologically, by hyperplasia and sloughing of Peyer's patches; and clinically by its slow, insidious onset, peculiar temperature-curve, swelling of the spleen, rose-colored spots, diarrhea, tympanites, sero-reaction, and a liability to certain complications (intestinal hemorrhage, peritonitis, etc.). The disease has an average duration of from three to four weeks.

History.—Although known beyond the reach of tradition, typhoid fever was clearly distinguished from typhus at a comparatively recent date. Louis of Paris in 1829 proposed the term *typhoide*, but it remained for Gerhard of Philadelphia to discriminate typhoid from typhus fever as the result of his own precise clinical observations. His account of the disease was ably corroborated by the writings of E. Hale and James Jackson, Sr. (1838, 1839). Later, Shattuck of Boston and Jenner of London made important contributions to the subject. Shattuck's experiments on typhus and typhoid fevers at the London Fever Hospital in England, and Alfred Stillé's studies of the former affection in Dublin and Naples, and of the latter in Paris, increased greatly our knowledge of these diseases. As a result of the labors of the above-mentioned American authors the true nature and identity of typhoid fever were appreciated in America at an earlier day than in either France or England.

Briefly, the decade from 1840 to 1850 witnessed, on the one hand, the overthrow of erroneous notions concerning the similarity of typhoid and typhus fevers, and, on the other, the establishment of their points of dissimilarity.

Pathology.—The lesions produced by typhoid fever may conveniently be divided into two groups: (1) Primary lesions, due to the direct effect of the special bacillus upon the lymph-follicles of the intestines, the mesenteric and other lymph-glands, and the spleen. (2) Secondary lesions, due chiefly to the long-continued fever and to secondary infection, for the occurrence of which the essential lesions of typhoid fever furnish the golden opportunity.

(1) **The primary morbid changes** in the Peyer's patches and solitary glands of the intestines are divided, usually, into four stages:

(*a*) **The Stage of Infiltration.**—The lymph-follicles become engorged

of Peyer generally presents several ulcers of irregular outline separated by strips of mucous membrane. Rarely, the entire plaque is implicated, in which case a large oval ulcer is the result, and at the lower end of the ileum the ulcers often coalesce until they almost encircle the bowel. The ulcers of the solitary glands assume a rounded form. The character of the floor of the ulcer will vary with the character of the intestinal coat which forms its base, though usually it is clean and smooth. The edges are usually irregular, engorged, soft, and frequently overhanging. In the lower segment of the ileum ulcers may be numerous, whilst in other portions of the gut Peyer's plaques may be merely hyperemic. In about 25 per cent. of the cases the typhoid ulcers are found in the large intestines—*i. e.* in the cecum and colon. Perforation of the large bowel is exceedingly rare. Exceptionally, the appendix is the seat of ulcer, and in a recent case of my own that came to autopsy perforation had occurred during the fourth week of the disease. Localized abscesses have been found under these circumstances.

Hemorrhage usually results from erosion of a vessel—an accident which is occasioned by the separation of the sloughs—but small bleedings may take place from the swollen, hyperemic edges of an ulcer. *Perforation* of the bowel occurs in a small percentage of cases (about 6 per cent.). In the majority of instances it is attributed to a perforative necrosis; hence it is that the sloughs are usually found attached to the orifice. Perforation may also occur after the separation of the necrosed portions during the stage of ulceration. The perforations may be multiple, though they are usually single and rarely exceeding two in number. The small, deep ulcers are more apt to lead to complete perforation than larger ones, and the site of the orifice is usually somewhere in the course of the lower third of the ileum. The lesions of peritonitis are invariably present, and during the stages of necrosis and ulceration a catarrhal state of the mucosa of the small and large intestines exists. The diarrhea which usually accompanies this affection is to be ascribed chiefly to the catarrhal state of the large bowel.

(*d*) **Healing** follows promptly upon the formation of the ulcer. At first a granular tissue covers its floor. The mucous membrane is replaced, including the glandular elements and epithelial layer, and, as in the stage of necrosis and sloughing, so the healing process advances inward from the border of the ulcer. Indeed, it is this process that dislodges the necrotic crust. Occasionally, ulcers are seen extending in one direction while healing in another. The cicatrix formed by the healing of an ulcer presents a smooth and often pigmented surface.

The stages thus far described do not, strictly speaking, follow one another, since two or more may be illustrated at once by a group of ulcers occupying the same section of the intestine. Again, when death occurs during a relapse fresh ulcers are observed by the side of others that are partially healed.

The Mesenteric Glands.—Changes in the mesenteric glands occur simultaneously with those in the intestines, and those situated opposite to the lower third of the ileum, the portion of the bowel showing the most extensive ulceration, are most profoundly involved. Hyperemia, and later swelling due to cell-infiltration, are among the earliest (hyperplasia), particularly Peyer's glands in the ileum and near to the valve, and, to a lesser extent, in the lower part of the jejunum. Frequently, the solitary glands in the small intestines, the colon, and rarely the rectum, become similarly infiltrated. In about 33 per cent. of the cases the chief morbid lesions are confined to the large intestines. In mild cases a few Peyer's patches in the lower part of the ileum are alone the seat of infiltration and subsequent changes. The follicles are grayish-white in color, and may project—particularly the patches of Peyer—from 3 to 5 mm. or more. Rarely, the solitary glands, which vary in size from a mustard-seed to a large pea, become very prominent and show a bold attempt at pedunculation.

The *histologic changes* at first consist in a marked dilatation of the capillary blood-vessels, which later are more or less compressed (as a consequence of cell-infiltration), giving to the follicles their whitish, anemic appearance. The cellular elements partake of the nature of lymph-corpuscles. Some of these cells are larger and are epithelioid in character, with ten or more nuclei. The mucosa and muscularis adjacent to the glandular structures may be similarly infiltrated.

From the eighth to the tenth day the stage of infiltration terminates either in resolution or in necrosis and sloughing. The infiltrated cells may undergo granular or fatty degeneration, followed by absorption. This process—resolution—during its progress produces pitting of the swollen follicles. In consequence of these minute points of necrosis the plaques now present a characteristic reticulated appearance (plaques à surface réticulée). When resolution occurs, accompanied by destruction of the follicles, small hemorrhages may take place into the glandular structure. These hemorrhages may occasion pigmentary deposits in the follicular depressions, giving rise to the so-called "shaven-beard" appearance. Resolution, however, terminates the stage of medullary infiltration with comparative infrequency. Far more frequently the hyperplasia of the lymph-follicles ends in

(*b*) **Necrosis or Sloughing.**—In all save the milder grades of cell-infiltration the hyperplasia of the lymphatic tissue cannot subside before necrosis occurs. The latter process results partly from compression and choking of the blood-vessels by the cell-infiltration, and partly from the direct action of the typhoid bacillus, leading to so-called anemic necrosis. Thus, necrotic crusts (sloughs) are formed, which are gradually separated and cast off. While not all of the glands of Peyer which are the seat of cellular infiltration undergo subsequent necrosis, as a rule those situated in the lower portion of the ileum do, and show the process in its completest development. The depth to which the necrosis extends is quite variable. It may involve only the most superficial layers of the mucosa, or it may extend in depth till it reaches, or even perforates, the outer or serous coat; but usually this work of destruction does not dip below the submucosa or muscularis. The necrosed portions become detached—a process that proceeds from the periphery toward the center—leaving behind the typhoid ulcer. The stage of necrosis and sloughing begins between the eighth and tenth days, and ends on or about the twenty-first day.

(*c*) **Stage of Ulceration.**—The size and shape of the ulcers correspond exactly to the necrosed areas in these respects. A single gland

Vesical catarrh is still more common, and the bladder may also be the seat of diphtheritic inflammation. Rarely orchitis is encountered. On making cultures from sections of the kidneys not a few observers have been able to demonstrate the specific bacillus of typhoid, particularly in the softened areas.

In the *lungs* are found morbid lesions in nearly all cases of typhoid fever, and belonging to the essential pathologic processes is bronchitis, due to a congested and catarrhal state of the bronchial mucous membrane. The lesions of lobular pneumonia present a complicating condition in many instances; those of lobar pneumonia also may be present, though less commonly. The so-called *hypostatic congestion* is often found, but is, I think, less frequent than is supposed by many authors. *Embolic infarctions*, having their origin in thrombi occupying the right side of the heart, are sometimes present. Gangrene may also occur.

Pleurisy is sometimes, though rarely, met with. It is most frequently of the plastic variety, although empyema occurred in nearly 2 per cent. of the Munich cases.

The *larynx* and the *pharynx* may manifest changes. Ulcers have been observed on the epiglottis and posterior wall of the larynx, and I have more than once seen them on the pharynx. When situated in the larynx they may extend in depth till they reach the perichondrium, causing perichondritis, with or without edema of the larynx as an associated lesion. Typhoid bacilli have been found in the ulcers (Eichhorst). Catarrhal, or even croupous, pharyngitis may occur, and a swelling of the follicles of the pharynx and base of the tongue is to be noticed in many cases. True aphthous changes, affecting the mouth and pharynx, may be present as a secondary event. The *mucosa of the stomach* is sometimes congested, and may even ulcerate, although this is very rarely seen.

Peritonitis is always found in fatal cases in which the bowel has been perforated. The condition is a general one, save in the rare instances mentioned below, and there is usually much fibrino-purulent effusion present. Diffuse peritonitis may be present without perforation, and results sometimes from a localization of the typhoid poison in the peritoneum, from rupture of suppurating mesenteric glands, but more frequently, I think, from direct extension of intestinal inflammation to the peritoneum.

The *heart* may be the seat of morbid changes. Acute endocarditis may be a very rare complication, while pericarditis occurs relatively more often—viz. in 14 of the Munich post-mortems before mentioned. Myocarditis is a not uncommon event, the cardiac muscle exhibiting parenchymatous and, less commonly, hyaline, degeneration, and the latter change sometimes leads to sudden rupture of the muscular fibers, with a fatal result (myocardite ségmentaire). It is, however, a significant fact that in the majority of instances, even of the severest type, the cell-fibers may show slight, if any, noticeable change. Out of 48 cases, 16 showed granular or fatty degeneration, and 3 a proliferative endarteritis in the small vessels (Dewevre).

The *arteries* have, in a number of instances, been found to be the seat of two forms of arteritis (Barié): (*a*) Acute obliterating arteritis, and (*b*) Partial arteritis. These conditions may affect the smaller veschanges, and correspond with the lesions noted in the intestines (*vide supra*). The mesenteric glands exhibit great variations in size, ranging, as they do, from that of a pea to a hen's egg. Their color-appearance is a grayish-red. Resolution occurs quite commonly, but, if it does not take place, then necrosis of the central portion (due, most probably, to the same causes that produce necrosis of the intestinal lymph-follicles) occurs, and suppuration has been observed in some cases. Still other glands become hyperemic and swollen (retroperitoneal, bronchial); but these usually tend toward resolution.

The Spleen.—With rare exceptions the spleen becomes enlarged in typhoid fever. At first hyperemic, the tissue then grows soft and granular, and at times is almost diffluent on section. Infarction is not a rare occurrence and may lead to suppuration. Keen has searched the literature and found only 9 cases of abscess. In rare instances, either spontaneously or as the result of injury, a rupture of the organ may occur, and the records of 2000 post-mortems at the Munich Pathologic Institute furnish 5 cases. Perisplenitis is seen, though exceptionally.

(2) **Secondary Lesions due chiefly to the Continued Fever and to Secondary Infections.**—The lesions in other organs are of subsidiary importance, and are, for the most part, secondary in nature, though we cannot, in the present state of our knowledge, draw a sharp line of distinction between these lesions and those that are primary. Further, in connection with the clinical history of the affection I shall point out that in a small percentage of cases the initial specific lesions may be localized either in the throat or in the lungs or kidneys.

The *liver* early becomes hyperemic, and later is softer and paler than is natural. Handford has described necrotic areas, and Wagner minute lymphomata. Infarction and abscess occur in rare instances. The *gall-bladder* may show catarrhal inflammation, and rarely a croupous, diphtheritic, or ulcerative inflammation leading to perforation. Westcott has tabulated 30 cases of typhoid infection of the gall-bladder that resulted in perforation. Chiari's[1] and Flexner's[2] figures show that typhoid bacilli are found in the gall-bladder in more than 50 per cent. of the fatal cases. (*Vide* Acute Infectious Cholecystitis, p. 857.) Mesenteric abscess and perforative appendicitis may be followed by pylephlebitis. The bile is thin and pale.

The microscope reveals parenchymatous and granular degeneration. The cells contain an abundance of fat, whilst their nuclei have lost, in great part, their outline.

The *kidneys*, like the liver, exhibit parenchymatous degeneration. They are somewhat pale-looking, are cloudy on section, and slightly swollen, and under the microscope granular and fatty degeneration of the epithelial cells of the convoluted tubules is observed. More rarely the lesions are those of *acute hemorrhagic nephritis*. Small areas of round-cell infiltration may develop late in the course of typhoid, and these may present an appearance similar to lymphomata or may undergo softening and suppuration, giving rise to miliary abscesses. The mucous membrane of the pelvis of the kidney is not infrequently the seat of a mild grade of catarrh, and, rarely, of diphtheritic inflammation.

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[1] *Prag. med. Woch.*, 1893, No. 22.

[2] *Johns Hopkins Hospital Reports*, vol. v.

frequently causes fatal results without producing the lesions characteristic of typhoid in human beings, although occasionally typical typhoid ulcers have been found. The susceptibility of lower animals, though normally slight, can be increased by preliminary injections of saprophytic bacteria, this result having been observed by Alessi when he exposed animals to the gases produced by putrefying matters. It has been found that the ulcerative intestinal lesions produced by the inoculation of the bacilli or their toxins in large quantities into the blood of rabbits may also be caused by other bacteria, including the bacillus coli commune.

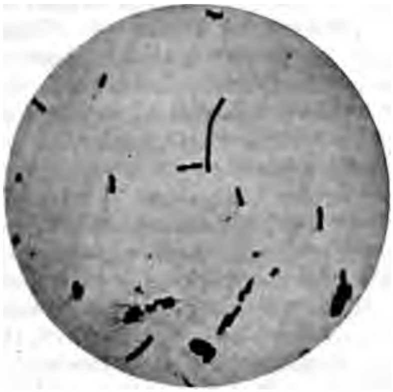
FIG. 1.—Typhoid bacilli with flagella; × 1000.

Usually, in making a *bacteriologic diagnosis* the typhoid bacillus is to be differentiated from those organisms that morphologically resemble it and present almost identical characteristics upon various culture-media. Particularly is this true of the bacterium coli commune, which can now be separated from the bacillus of Eberth, bacteriologists having pointed out the fundamental differences between these related forms. Smith and Tennant, in a study of the 1898 epidemic of typhoid fever in Belfast, failed to find typhoid bacilli in the water-supply, but were able to isolate a large number of varieties of the bacilli coli communis. Occasional races were isolated, which, when tested with the serum of typhoid patients, showed by their reactions that they possessed special relation to the typhoid disease.

The real poison of typhoid fever is most probably a ferment(?) secreted by the bacillus—*typho-toxin ;* and Brieger has extracted the latter agent, finding that it produces the fever, nervous symptoms, and the other manifestations characteristic of the affection.

Distribution in the Body.—The bacillus has been found in the intestinal tract, the lymph-glands, the contents of the intestine, the spleen, the liver, the rose-colored spots, the blood, and the bile. The bacillus typhosus is demonstrable in the stools (in about 50 per cent. of the cases), the urine (Wright and Semple), the sputa, the vomita, and the sweat. Less frequently they have been found in foci of suppuration and in exudations (pleural, endocardial, meningeal).

The Bacilli Outside the Body.—The bacilli cannot maintain a permanent existence outside the human body. From time to time, however, the conditions indispensable to the growth and development of the typhoid germs prevail, and corresponding with such periods of time more or less extensive epidemic outbreaks of the disease may occur. It is known that the typhoid bacilli may retain their vitality for from seven to fourteen days in water, disappearing from the same on account of the presence of saprophytes ; but an epidemic or an endemic of typhoid fever implies persistent contamination of the drinking-water. Multiplication of the bacilli may take place in water, in milk (very rapidly), and in the soil (where they preserve their vitality under favorable conditions for sels, particularly those of the heart, but they occur most commonly in the arteries of the lower extremities. Thrombi are found in the right chambers of the heart and in the veins—most frequently in the femoral, and less often in the cerebral sinuses.

The *voluntary muscles* undergo parenchymatous and, occasionally, a hyaline, change, though this is not a feature peculiar to typhoid fever. The latter form of degeneration does not affect the whole muscle, only certain fibers being involved, and as a rule the recti abdominis, the diaphragm, the adductors of the thigh, and the pectorals are the seats of the lesion. The parts affected are pale and possess a grayish, waxy luster. Histologically, the process implies the transformation of the muscular fibers, and especially the cement substance, into a homogeneous, pliable mass. Regeneration of the fibers thus destroyed occurs during convalescence. Hemorrhages into, and rarely abscesses in, the intermuscular tissue occur.

The *nervous system* presents no gross lesions, if we except meningitis, the latter occurring as a complication; but it is exceedingly rare, having been present in only 11 of the 2000 Munich cases. In a few instances large cerebral hemorrhages have been met with, but these are apparently coincidental, while capillary hemorrhages into the cortex may be numerous. Meningeal hemorrhages may also occur. Slight edema of the cerebral cortex has been noted. The peripheral nerves are not infrequently the seat of parenchymatous change, with or without local neuritis, and the ganglia of the trunks of the vagi exhibit an inflammatory change which Levin believes is the cause of the laryngitis, pharyngitis, pharyngolysis, and arrhythmia sometimes observed.

The *blood* shows few important alterations. The red blood-corpuscles are relatively increased during the febrile period and markedly diminished during convalescence, but the great loss of water during the former period and a reabsorption during the latter will explain these interesting facts (Henry). Leukocytosis is absent, and there is often an actual decrease in the number of leukocytes. The mononuclear forms are more numerous than in health.

Etiology.—Bacteriology.—The bacterium which is the specific cause of typhoid fever was discovered by Eberth, whose researches were later confirmed by investigations of Gaffky and others.

General Characters.—It is a short, thick bacillus, about three times as long as it is broad, with rounded ends (Fig. 1). It is motile, due to the presence of cilia on both sides, and when stained exhibits vacuolations that have been mistaken for spores. It is easily stained with all the anilin dyes.

Characteristic Growth.—Upon gelatin plates it develops in grayish translucent colonies with irregular borders and ridged surfaces. Upon agar the growth is not characteristic; upon the potato, especially if it has been rendered slightly acid, it forms a perfectly transparent growth that is only evident as a slight apparent increase of moisture upon the surface, and as offering a greater resistance to the point of the needle when scraped across it. It neither coagulates milk, liquefies gelatin, nor produces indol. The organism never forms spores. Moreover, the bacillus has no greater powers of resistance than the ordinary bacteria.

Experimental Typhoid.—Inoculated into lower animals, the bacillus

To account for this fact we have the influence of a change both of sanitary surroundings and of the habits of life. There is considerable evidence to show that the disease is on the increase in rural sections. That the predisposition to this affection may also be *inherited* I have no doubt. Most persons, however, enjoy natural immunity from the affection.

(*g*) **Intestinal Catarrh.**—I have observed cases of influenza, with marked catarrh of the gastro-intestinal tract, to be followed promptly by the symptoms of typhoid fever (*vide* Influenza).

(*h*) **Nervous Influences.**—Great mental excitement and overwork are among the predisposing nervous causes.

Immunity.—The occurrence of typhoid fever confers an approximate, though not an absolute, immunity against subsequent attacks.

In this connection two questions present themselves for consideration:

(1) **What are the methods of conveyance of the poison into the human body?**

In the first place, isolated cases and epidemics of typhoid fever are alike to be attributed to antecedent cases of the disease, and this fact presupposes that the bacillus of typhoid leaves the body of the sufferer during the attack. This it does in the stools, the urine, and occasionally in the vomitus and sputum, which are the primary sources of infection. The dejecta, which are the principal source, contain the bacillus of Eberth in great numbers, and these may be conveyed to well persons by—

(*a*) **Infected Drinking-water.**—In the vast majority of instances the poison is transmitted from those affected with the disease to those in good health through the drinking-water supply. This has been true in most extensive epidemic outbreaks in which the mode of origin has been traced. Wells, storage reservoirs, springs, and rivers may become contaminated and cause epidemic prevalence of the disease.

In the spring of 1885 a most instructive, though deplorable, epidemic occurred in Plymouth, Penna., a town of 8000 inhabitants. At first the nature of the affection was not recognized, and before it ceased to appear 1200 persons were affected, with 130 resulting deaths. This epidemic was investigated by Shakespeare and L. H. Taylor, and was found to have arisen from a single case of typhoid occurring in a house on a hill which sloped toward the water-supply of the town. This patient was ill during January, February, and March, while the ground was frozen and covered with snow, upon which the dejecta were thrown by the attendant. On March 25th there was a considerable rainfall, followed by a sudden thaw, and the water, unable to sink in the frozen earth, ran at once through the various surface channels into a brook, which in turn emptied into the reservoir. Coincidently with the thaw the patient had frequent and copious stools, and, strangely enough, for certain reasons the infected water-supply was at the same time more largely drawn upon than usual. On April 10th other cases of the disease appeared, and careful investigation showed that those citizens who obtained their water from other sources than the infected reservoir escaped the disease.

(*b*) **Infected milk** frequently conveys the poison. It may become polluted by water which has been used either to wash the cans or for diluting purposes, or the bacilli may be transferred to milk by the unclean hands of the milker. Numerous instructive epidemics, originating in infected milk, have been reported. The occurrence of numerous cases among children suggests contaminated milk.

Solid forms of food (salads, celery, fruits) may be contaminated by infected water or dust or by the fingers of the nurse or the patient. During the late Spanish-American war the typhoid bacilli may have been conveyed from the latrines directly to the victims or to the kitchens and mess-tables by swarms of flies. Vaughan[1] confirms this view, and has also observed that "officers whose mess-tents were protected by means of screens suffered proportionately less from typhoid fever than did those whose tents were not so protected." He also believes that fecal matter containing the typhoid germ may adhere to the fly, and be mechanically transported, and further suggests the possibility of the bacilli being carried in the digestive organs of the fly, and deposited with its excrement. H. W. Conn has shown that oysters while being fattened or freshened may become infected with water polluted by sewage, and Foote has shown that the typhoid bacillus will not only retain its vitality in the salt water in which the oysters are fed, but that it will live even longer in the oyster itself. Newsholme[2] attributed one-third of a total of 56 cases of typhoid to the eating of raw shell-fish.

(*c*) **Contagion or Direct Transmission.**—This necessitates direct contact with the typhoid stools, and its possibility cannot be denied. It affords a ready explanation for contraction of the disease by nurses who attend to the stools, the bed, and the body-linen of the patient, and by laundresses who are also obliged to handle the soiled clothing, and who are affected with great relative frequency.

(*d*) "**Ground-soil.**"—According to Pettenkofer's view, the typhoid poison which leaves the body of an infected person must undergo modification or development in the ground-soil before it is potent to cause the disease in question. The former great prevalence of typhoid fever in Munich was due to the great pollution of the soil (including specific pollution) modified by certain unknown conditions in the soil which correlated with the movements of the subsoil-water (Childs).

(*e*) **Sewer-gas.**—The typhoid stools, if they have not been thoroughly disinfected, may meet in sewers all the conditions favorable to the growth and propagation of the bacilli. But the recent researches of Bergey and of Abbott show that sewer gas is not decidedly deleterious, and it certainly cannot of itself cause typhoid fever.

(2) **Through what channel or channels does the bacillus enter?**

(*a*) **In the vast majority of the cases the bacilli are swallowed.** It must not be forgotten that bacilli when inhaled may find lodgement in the mouth, pharynx, etc., and then be carried along into the stomach with the next food or drink that is ingested. In the stomach they meet with the acid gastric secretions, which often destroy them. They may, however, pass into the intestinal canal, where the alkaline juices of the small intestine furnish every condition necessary for their further growth and development. They penetrate the mucosa and attack primarily the solitary follicles and Peyer's plaques. Next they invade the mesenteric glands, reaching the circulation, spleen, liver, and other organs a little later.

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[1] *Philada. Med. Journ.*, June 9, 1900.

[2] *Brit. Med. Journ.*, June 8, 1895.

(*b*) The possibility that the bacilli may reach the blood-stream through the *respiratory organs* must be conceded; and hence the added possibility that they may set up initiatory lesions either in the tonsils, lungs, or pleura, passing thence into the circulation, must also be granted. Vaughan inclines to the opinion that the bacillus may be inhaled in the infected dust by troops on the march. Complete desiccation, however, soon destroys the typhoid germ. Primary localizations may also occur in the kidneys and cerebrospinal meninges. Special clinical varieties arise from these localizations (*vide infra*), and they may or may not show the more usual intestinal lesions.

(*c*) **Typhoid Septicæmia.**—By this is meant a general infection with the bacilli without localized lesions. The special mode of infection is not clear.

(*d*) Typhoid infection predisposes the system to secondary infections with various bacilli (streptococcus, staphylococcus, bacillus coli commune, pneumococcus). The portals of entrance for these micro-organisms are various (*e. g.*, respiratory tract, lymphatics).

Clinical History.—**I. Incubation.**—The average duration of the period of incubation, or the time between the introduction of the poison into the system and the appearance of the first active symptoms, ranges from ten days to three weeks, though it sometimes lasts for a longer, and oftener, I think, for a shorter, time. During this period the patient may experience no deviation from health, but in most cases there are prodromal symptoms, such as languor, loss of appetite, nausea, headache, neuro-muscular pains in the back and limbs, a disinclination to exercise, etc. These symptoms last usually from a few days to a week or more.

II. General Symptomatology and Course.—On account of the peculiar temperature-curve in typhoid fever its course falls naturally into three periods—the stage of development; the acme or fastigium (corresponding to the height of the disease); and the stage of decline or defervescence. It is convenient to speak of the various weeks of the affection when referring to these stages. Thus, the first week represents the stage of development (stadium incrementi), the second and third weeks (in cases of average severity) the fastigium, while the fourth week in the typical form (the third week in mild cases) corresponds to the third stage (stadium decrementi) of the disease.

(*a*) **Stage of Development.**—The invasion, as a rule, is gradual, the symptoms being chilliness and feverishness, with increase in the severity of the prodromal symptoms. Typhoid fever rarely starts in with a distinct *rigor*. At or about this time nose-bleed may betray the nature of the disease. The symptoms just described are quickly followed by a prostration sufficiently well marked to compel most patients to take to their beds. From this latter event is usually dated the *onset* of the affection. It is safer, however, to regard the time of occurrence of the above-mentioned symptoms (elevation of temperature, with its attendant discomforts) as the time of onset, since many patients continue in their avocations for days after the first symptoms appear. The onset may be marked by symptoms resembling influenza (Bunce). In my experience, cases in which general pains, including backache or slight pharyngitis, are seen at the onset, are not rare.

With the progress of the initial period the symptoms usually increase in severity with considerable rapidity; the *fever* rises day by day, terrace-like, till, at the end of four or five days, the second stage, or fastigium, is reached. Anorexia is complete, thirst is great, headache rather intense, the skin hot and dry to the feel, the tongue coated, the sleep disturbed, and constipation is generally present. The patient may complain of sensations of chilliness alternating with flushings of heat, and there is a slight cough with some thoracic oppression. The *pulse* is somewhat quickened (from 90 to 110 per minute) and is full, though rarely, thus early, is it dicrotic.

The *physical signs* are not prominent. The abdomen is often slightly distended and tender; the spleen, on *palpation*, is found to be swollen.

(*b*) **Fastigium, or the second stage,** commences usually on the fourth or fifth day of the disease, and lasts, in typical cases, about two weeks. During the first week of the fastigium (the second of the disease) the general symptoms become more marked. The *fever* remains high (the evening temperature usually reaching 103° or 104° F. (40.° C.), and exhibits the continued type. The *pulse* is accelerated but not dicrotic. The headache disappears, and mental dulness and slowness are conspicuous, but there may be mild delirium, particularly at night. There is a dry *cough* and the physical signs indicate more or less extensive bronchitis. The *tongue* is coated and may become dry, the belly is somewhat swollen and tender, and diarrhea replaces constipation. The *spleen* is decidedly enlarged, and about the eighth day of the disease a number of roseate spots, which are pathognomonic, appear on the trunk. During the latter part of this week a grave or even fatal condition may be developed as a result of intense nervous or pulmonary symptoms, intestinal hemorrhage, or perforation.

During the second week of the fastigium (the third week of the disease) the marked general symptoms already noted persist in severe types of the affection. The *pulse* varies from 110 to 130, and the temperature may approach the remittent type. In addition, this period furnishes the most numerous as well as the most untoward *complications* (lobular pneumonia, hypostatic congestion of the lungs, intestinal hemorrhage, perforation, peritonitis), and in the absence of serious local complications grave general conditions may be presented. The duration of this stage varies with the severity of the type.

(*c*) **Stage of Decline or Defervescence.**—At the end of the second stage, and about the twenty-first day of the disease, in favorable cases the *fever* begins to decline, and with it the other general and local symptoms gradually disappear. This is followed by true convalescence. In protracted cases, however, the fourth week of the disease may present much the same clinical indications as the third, and these may even be intensified. Frequently an aggravated type of the *typhoid state* is now superadded, the symptoms being stupor, muttering delirium, subsultus tendinum, a rapid, feeble pulse, a dry, brown tongue, marked diarrhea, greatly swollen belly, and an involuntary discharge of feces and urine. Inflammatory complications may add to the perils of the condition.

In not a few cases the febrile period is prolonged into the fifth, and rarely into the sixth or even the seventh week, and the fever observed when defervescence is retarded presents an irregular type. I have elsewhere reported a case in which it lasted not less than seven weeks.[1] About this time recrudescences and relapses may occur in typical cases. Different epidemics of typhoid fever, however, vary so greatly in their clinical characteristics as to make it impossible to include all cases in any outline of the course of the disease that might be attempted.

III. Chief Clinical Features in Detail.—(*a*) **Course of the Fever.**—During the stage of development (the first four or five days) the temperature usually rises in "step-ladder" fashion. The evening exacerbation is on each day from a degree and a half to two degrees higher than on the preceding, and the same is true of the morning remissions. A glance at the temperature-charts (Figs. 2 and 3) will show that the morning remissions touch a level from one-half to one degree lower than the preceding evening registers. This stage is rarely met.

When the fastigium is reached, the evening temperature may be 103°, 104°, or 105° F. (39.4°–40.5° C.), and is usually thus maintained, with the slight morning remissions. The tide-like character of fever-curve seen in the initial period is absent. Often, during the latter half of the fastigium (the third or fourth week of the disease) the morning fall of temperature becomes decidedly greater. According to my own observation, the height of the fastigium is reached a day or two after its onset or at the end of the first week of the affection. Marked morning remissions are a favorable indication. On the other hand, and contrary to the general rule, the morning temperature may be higher than the evening, forming a somewhat unfavorable symptom. Morning temperatures of 104° F. (40° C.) or over are indicative of a serious type. In many instances of mild grade the evening temperature at no time exceeds 103° (39.4° C.), but oscillates between 100⅗° and 102⅗° F. (38.1°–39.2° C.). In cases of average intensity the morning remissions touch 102°–102⅗° F. (39.2° C.), and the evening exacerbations reach 104–104⅗° F. (40.3° C.). When the temperature rises above 105° F. (40.5° C.) hyperpyrexia exists. Ampugnani made studies of hourly charts from 200 cases of typhoid fever, and found the maximum temperature to occur between three and six o'clock in the afternoon, and the minimum between four and eight o'clock in the morning. The duration of the fastigium exhibits a wide range and is dependent upon a variety of conditions—*e. g.* the degree of mildness or severity of the type, the presence or absence of complications, etc. In cases of a mild character it lasts from a few days to one week; in cases of average severity, from ten days to two weeks; in the severest forms, from two to four weeks.

In typical cases the end of the fastigium marks the beginning of the last stage (that of defervescence), and during this period the temperature falls by *lysis*. Measured by days, it declines by degrees, both the morning and evening temperatures being often one or two degrees lower than on the preceding day. Thus is formed a more or less regular step-like line of descent. To this general rule there are two notable exceptions: From the beginning of the period of defervescence the morning remissions may strike the normal point, while the evening ex-

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[1] "A Case of Typhoid Fever; numerous Intestinal Hemorrhages, the Amount of Blood Lost being Seventy-eight and one-half Ounces; and Obstinate Vomiting, with Recovery," *International Clinics*, vol. i. 5th series, April, 1895, p. 29.

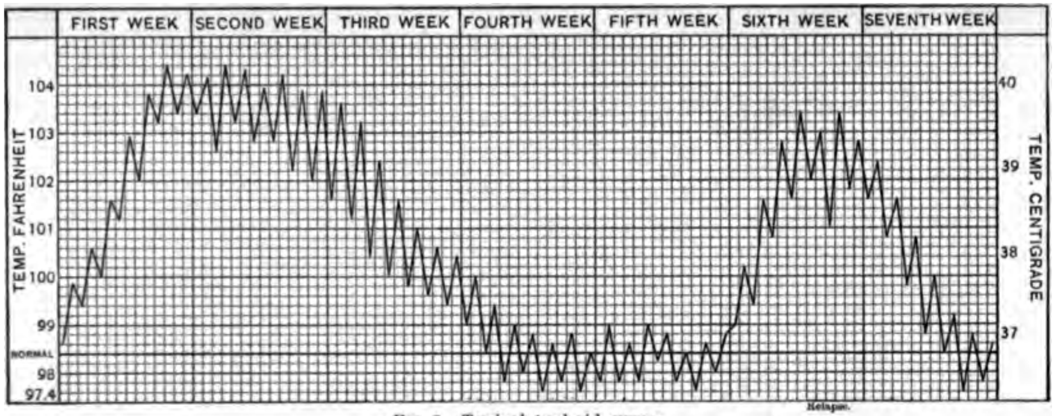

Fig. 2.—Typical typhoid curve.

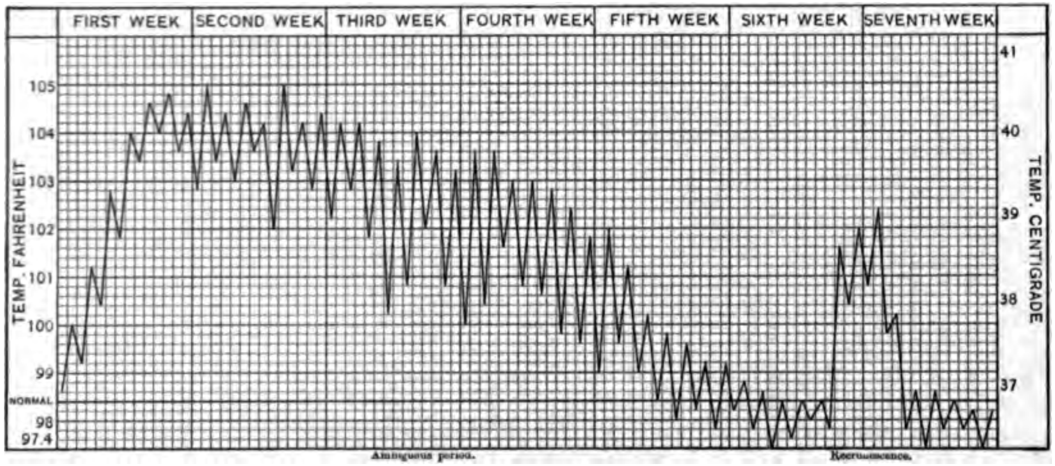

Fig. 3.—Curve in severe typhoid.

acerbations become less and less marked, until they also touch the normal. Under these circumstances the temperature-curve resembles somewhat that of the quotidian intermittents, and rarely the tertian type of curve obtains. In comparatively rare instances the morning temperature shows a deeper remission on each successive day, while the evening temperature remains high for several days, when it also declines. This period lasts from one week to ten days—a longer time than the initial stage with its ascending type of fever.

In the severe and protracted forms of typhoid fever there occurs between the second stage (fastigium) and the third stage (defervescence) another, to which Wunderlich has given the name of the "ambiguous period." This lasts from a few days to a week or more, and is characterized by a striking diurnal range of temperature, with marked irregularities. It is probable that it is sometimes produced by an auto-intoxication.

Abnormal Course of the Fever.—The pyrexial peculiarities yet to be pointed out are less usual than the foregoing, though of sufficient frequency of occurrence to demand a brief description.

The first stage varies but little from the regular course described above. A sudden elevation of temperature, however, is seen in those cases that begin with a severe rigor, accompanied by pneumonic, catarrhal, and gastro-intestinal symptoms. Pepper and Stengel have reported seven cases with acute onset, and Moore, of Dublin, states that the whole course (since 1889) has become more typhus-like than formerly.

In the lightest forms the *fastigium* may be practically absent, defervescence setting in upon the first day of the fastigium. There is also a class of cases in which, throughout the greater part of their course, the fever is distinctly intermittent or remittent, and in which careful blood-examination fails to disclose the *plasmodium malariæ.* The same characteristic marks the temperature-curve in those rare instances of typhoid fever which occur in subjects previously infected with malaria. These two classes of cases run a favorable course as a rule.

Sudden deep temporary drops in the temperature may occur during the fastigium. (1) This may take place during the early part of the fastigium without obvious cause. (2) Intestinal hemorrhage almost invariably produces a sudden, and sometimes a great, fall of temperature. Osler has reported a case in which a drop of 10° F. (5.5° C.) followed melena. The blood does not appear in the evacuations of the patient for six to twelve hours or more after the temperature has begun to fall; and hence a critical decline of temperature during the latter part of the second and the third week of the disease suggests that hemorrhage has probably taken place. (3) The occurrence of *peritonitis* is marked by a sudden and considerable fall of temperature.. (4) In the female, abortion or premature delivery occurring in the course of typhoid fever produces a decided lowering of the temperature. (5) Collapse of the circulation sometimes occurs with a notable remission of temperature—an ominous association of events, and one which I observed in two cases occurring in females in the Medico-Chirurgical Hospital. In one of these cases two such periods of collapse occurred, and in the other three, though both finally recovered under prompt and continuous stimulation. Occasionally *hyperpyrexia* is observed in typhoid fever, and most frequently just before dissolution, when the thermometer may register 108° or even 109° F. (42.7° C). A fresh rise with marked irregularity of temperature may occur during the latter part of the fastigium or the period of decline, and is often dependent upon some local complication (late pneumonia, parotitis, etc.).

The stage of defervescence is sometimes much prolonged, though most frequently there is simply a slight evening elevation (99° to 100° F.—37.7° C.), the morning temperature being normal. The causes of retarded decline are, for the most part, obscure. I believe that many of them are ascribable to a mild grade of auto-intoxication, and in my hands a mild saline laxative has been the means of cutting them short in a number of instances. An examination should, however, be made for some localized inflammatory complication, though this is not always discernible, as in the case of suppuration in the mesenteric glands, etc. Sluggish typhoid ulcers, which refuse to heal promptly and are due to the now well-known post-typhoid anemia, may act as a cause of the slow decline.

Post-typhoid Elevations of Temperature.—After both the evening and morning temperatures have become normal, fresh temporary elevations (102° or 103° F.—38.8° or 39.4° C.) frequently appear. They are, as a rule, unassociated with any other symptoms, and at the end of a few days the temperature falls rapidly to the normal. These are termed *recrudescences*, and are to be distinguished from true typhoid relapses. They are probably produced in various ways—by errors in diet, constipation, mental emotion, excitement, etc.—and there are cases in which the presence of the fever seems to be really a nervous phenomenon (Osler). It is most common in children and in persons of a decidedly nervous temperament. Certain local sequelæ may cause post-typhoid fever, such as abscess, periostitis, etc. Rarely during convalescence a sudden and marked elevation of temperature, accompanied or not by rigor, occurs, but it is usually of short duration and seldom is of serious import. I recently saw, with the attending physician, Dr. Modell, a case in which the temperature had been normal for six days, when rigors, followed by steep elevations of temperature, occurred several times and at intervals of thirty-six or forty-eight hours. These high temperatures were followed by a rapid decline to the normal, and by sweating, leaving the patient profoundly exhausted. Subsequently the convalescence was slow, but uninterrupted.

Afebrile Typhoid.—As the term indicates, typhoid fever may run a course attended with all of the characteristic symptoms save the fever. Cases of this kind are of great rarity.

(*b*) **Skin.**—The *eruption* is highly characteristic, and usually decides the diagnosis. It makes its appearance on or about the eighth day, and sometimes a little later. Occasionally it does not appear until the tenth or twelfth day of the disease. It consists of distinct, rose-colored, and slightly elevated papules, having a rounded or lenticular form and a diameter varying from one or two to three lines. The papules are almost invariably found upon the trunk, and especially upon the upper part of the abdomen and the lower part of the thorax, to which regions they may be wholly confined. They may, however, be absent from the usual seats and present elsewhere, so that the sides of the trunk, the back, and the thighs should always be inspected. They disappear upon pressure, but reappear promptly when pressure is removed. These rose-colored spots last three or four days, and appear in successive crops, each one being made up, usually, of a few spots—a half-dozen to a dozen. Rarely the eruption is abundant on the trunk, even extending to the extremities and head; but there is no direct correspondence between the extent of the eruption and the severity of the cases. Occasionally the spots are entirely absent—a condition most frequently met with in children, and less often in elderly persons.

Other eruptions are often present, and their negative diagnostic value must be kept in remembrance. Minute pearly vesicles (*sudamina*) may appear. They are limited to the abdomen, the axilla, and to the inner surface of the thighs as a rule, and are in great measure due to profuse sweating.

A scarlet-colored *erythematous eruption* sometimes appears at a comparatively early period in typhoid fever. *Urticaria* and *purpura* are rarely seen. Out of 250 cases of typhoid fever among the soldiers in the Spanish-American war treated in the Medico-Chirurgical Hospital two manifested purpuric spots. *Extensive ecchymoses* may occur, but are rare, and merely symptomatic of the hemorrhagic diathesis. Cutaneous *boils* and *abscesses* due to secondary infection with the pyogenic cocci are a comparatively frequent and late development in the course of the disease. *Peliomata typhosa* in the form of little bluish subcuticular spots (the "tâches bleuâtres" of the French writers) may appear; but they are not related specially to typhoid fever, and in a recent case of my own were undoubtedly due to pediculi. *Gangrene*, chiefly of the lower extremities, has been noted in 214 cases (Keen), and is due to the diffusion of the bacilli and their toxic products, to an obliterating endarteritis, thrombosis, or embolism.

Profuse sweats form a conspicuous symptom in many epidemics of the disease, with or without accompanying fits of chilliness or rigors, and mark the sudoral form of typhoid fever (Jaccoud). Some of these instances resemble ordinary intermittents. *Edema* of the skin is sometimes observed and is due most frequently to anemia or cachexia, though sometimes to nephritis. A local form of edema affecting the leg is not uncommon, and for this form thrombosis of the femoral vein is chiefly responsible. A peculiar "musty" odor is exhaled from the skin in some instances of typhoid fever. The patient assumes the dorsal decubitus and is exposed, particularly in cases of prolonged duration, to the danger of the formation of *bedsores*. They are most prone to occur on the nates and the heels, and, once started, they are apt to spread till they attain to large dimensions, with extensive undermining of the skin. The condition is now serious. During and after the conclusion of convalescence the hair falls out, but, fortunately, it is invariably renewed. The *nails* sometimes become roughened and brittle, while transverse pale lines or ridges can usually be observed in them, marking the impairment of nutrition during the disease (*vide* Relapse). *Jaundice*, due to a variety of causes, is a rare symptom, and generally does not come on until the middle of, or until late in, the disease (Da Costa).

(*c*) **Digestive System.**—The symptoms referable to the gastro-intestinal canal, though not very striking in most cases, are of the utmost importance and interest because of their direct connection with the pathognomonic lesions of typhoid. Beginning with the intestinal canal, and thence proceeding to the symptoms presented by the stomach, spleen, liver, throat, and mouth, will be a natural and convenient order.

At the onset of typhoid fever *constipation* is the general rule, and this may persist to the end of the illness, though more commonly a moderate diarrhea appears. Osler[1] in the Johns Hopkins Hospital, however, met an initial diarrhea in 322 out of 829 cases. During the second week of the affection the stools number, on the average, from two to four or more daily. It is only in comparatively rare instances that ten or more movements per diem occur, and the severity of the diarrhea depends largely upon the degree of catarrh, particularly of the large intestine. When, however, the ulcerative process is chiefly limited to the colon, it is an important factor in the production of the diarrhea. Indeed, in those instances—not altogether rare—in which there is urgent diarrhea of a *dysenteric character*, the ulcers are especially marked in the colon, with diphtheritic inflammation of the surrounding mucosa. Involuntary discharge of the feces may occur.

The *stools* present a characteristic yellow appearance, suggesting by their color and consistence a comparison with pea soup. They are usually either fluid or of the consistence of jelly, and are offensive and of an alkaline reaction. On standing they separate into two layers—an upper, liquid, cloudy layer, and a lower, thick yellow, sedimentary layer, in which, on macroscopic examination, remnants of food and grayish yellow fragments (necrotic crusts of Peyer's plaques) from a half to an inch in length may be detected. Microscopically, they have been found to contain undigested particles of food, epithelial débris, blood-corpuscles, crystals of triple phosphates in abundance, and innumerable bacteria. Laboratory experimentalists have been able to demonstrate the presence of the typhoid bacillus in the dejecta. *Tympanites*, mainly affecting the colon, is a common though rarely a striking feature, and cases of a quite serious nature are observed in which the abdomen presents a concavity throughout the entire illness. The latter is less unfavorable, by far, as a symptom than excessive tympanites, which interferes with both the respiration and heart action. Tympanites is apt to be most marked in serious cases which have diarrhea as a prominent symptom, though the latter may not even be present. It is due to the generation of gas from decomposing food, and to the arrest of peristaltic movements in consequence of the degeneration of the muscularis of the intestines. *Pain* is absent in the majority of cases, and when present is not intense, save in rare instances. Pressure upon the ileocecal region usually causes a *gurgling noise*, but, although this symptom is commonly present, it is not characteristic of the disease. There is generally also a slight degree of tenderness of the abdomen under pressure, most marked in the right iliac fossa, and hence, in all probability, due to the presence of ulcers in this region. Absence of tenderness, however, is not a safe indication of the absence of extensive ulceration. *Extreme sensitiveness* generally denotes peritonitis (often without perforation), although the symptom may be marked in constipation.

Intestinal hemorrhage occurs in from 4 to 7 per cent. of cases, its frequency varying with different epidemics. The hemorrhages appear almost invariably during the latter part of the second and third week, being caused by the opening of blood-vessels during the necrotic or ulcerative process. Bleedings may also take place from the soft and hyperemic edges of the ulcer (*vide supra*), and when it occurs quite early in the disease it may be due to an excessive hyperemia of the lymph-follicles. The amount may be so small as to be scarcely discernible by the naked eye, or it may be from one to two or three pints (0.5–1.5 liters), or even more. In one of my own cases the total amount of blood discharged from the bowel was nearly 5 pints (2.5 liters), and yet the patient recovered. The blood presents a dark hue, and that which is passed last may be tarry.

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[1] *Philada. Med. Journ.*, October 15, 1900.

The significance of intestinal hemorrhage, however slight, is always grave. On the other hand, recovery is possible even if the hemorrhage be copious and oft-repeated; and in general terms it may be said that death supervenes in from 30 to 40 per cent. of all cases. R. G. Curtin has recorded 60 cases, of which 46.6 per cent. died; he argues that cold applications to the skin and the necessary disturbance in giving a cold bath tend to produce melena. It occurred in more than the usual proportion of cases in the soldiers under my care during the Spanish-American War, probably owing to the fact that the men were conveyed from the various distant camps to the Medico-Chirurgical Hospital. A fatal result may occur as the direct effect of a profuse hemorrhage. When death does not follow immediately, however, the signs of collapse (more or less intense) and of anemia appear; yet intestinal hemorrhage sometimes exerts a favorable influence, stupor and delirium quickly giving place to consciousness. When typhoid fever occurs in the hemorrhagic diathesis hemorrhage occurs from all of the outlets.

Perforation, which almost invariably produces fatal diffuse peritonitis, is the accident most to be dreaded. It does not bear a fixed relation to the severity of the affection, but in the 2000 Munich cases (*vide supra*) perforation occurred in 114; and according to Fitz, who tabulated 4680 cases of typhoid fever, there is a mortality of 6.58 per cent. from perforation of the bowel. It is much more common in males than in females, and appears in a ratio of about 71 to 29. *Age* has a decided influence, the complication being most marked between ten and forty years old, whilst in children it is rare; and, though perforation may occur at any time in the course of typhoid fever, it is most common between the second and fourth weeks of the disease. In the cases analyzed by Fitz perforation was found in the ileum in 81.4 per cent., in the large intestine in 12.9 per cent., in the vermiform appendix in 2.5 per cent., and in the jejunum in 1.29 per cent. The accident is usually announced by the sudden advent of acute *pain* in the abdomen, quickly followed by the symptoms of *collapse*; and the fact that diffuse peritonitis, following perforation, may develop insidiously must be recollected. The abdominal muscles become rigid, sensitive to touch, and later tympanites develops. Fluctuation can sometimes be elicited. On *percussion* splenic and hepatic dulness is often absent, but hepatic dulness is also wanting when the distended intestines lie in front of the liver. The collapse of the circulatory system is evidenced by the pinched features, hollow cheeks, vomiting, and the small, frequent pulse.

Leucocytosis is a valuable diagnostic symptom. Wilson has emphasized the importance of an early diagnosis and the immediate resort to operative intervention.

No other complication is so grave as *peritonitis*. Its causes have been pointed out previously (*vide* p. 21), but from a clinical point of view a division of all the cases into two classes—those due to perforation and those due to other agencies—is desirable. The instances that develop independently of actual perforation are not of infrequent occurrence. They usually assume the local or circumscribed form of peritonitis, which is occasioned by direct extension of the inflammatory process from the intestinal ulcers. The condition presents corresponding areas of tenderness under gentle, and especially under prolonged, pressure. It is, however, confessedly difficult to diagnose between the intra- and extra-intestinal states, which are accompanied by sensitiveness to gentle palpation, particularly when peritoneal inflammation exists in a mild form. Generalized peritonitis may succeed to the circumscribed variety in consequence of direct extension of the inflammation.

The *mesenteric lymph-glands* may soften or suppurate (*vide* Pathology), and, as before mentioned, may be the exciting cause of a recrudescence, or they may rupture and cause diffuse peritonitis.

The Spleen.—With few exceptions the spleen is enlarged in typhoid fever, the edge usually being palpable below the margin of the ribs, on or before the commencement of the fastigium. It generally goes on increasing in size till near the beginning of the third week, and lessens during the latter part of the third and fourth weeks. In four of Osler's autopsies it weighed less than normally. Swelling of the spleen is sometimes absent after a copious intestinal hemorrhage, as well as in elderly typhoid subjects. As before mentioned, the enlargement in many cases is not demonstrable by percussion when the tympanites is excessive, but by means of careful palpation we can in most cases satisfy ourselves of its existence or non-existence, despite the great distention of the bowel. Suppurative infarcts, or softening of the spleen may start a peritonitis. Rarely, *rupture* of the organ may occur, which is manifested usually by intense pain in the splenic region. A slight swelling of the *liver* can sometimes be detected.

The Liver.—Among the least frequent complications is *jaundice* (*vide supra*), and abscess of the liver also rarely occurs.

The Stomach.—The stomach presents no characteristic symptoms. Of the *anorexia*, enough has been said, but during convalescence the appetite returns, becoming even voracious. *Nausea* and *vomiting* may occur during any stage of the disease, but are most common at the beginning. When they appear as late symptoms they are probably excited either by a typhoid ulcer or by peritonitis. Nausea is usually traceable to definite causes—either to errors in diet or to the use of irritating medicaments, but vomiting also occurs from unknown and inevitable causes. This sort of vomiting was present in the afore-mentioned case reported by myself. It may become a serious or even fatal symptom. Hiccough is a rare but serious symptom. Hematemesis has been observed, although practically unknown.

The Pharynx.—The pharynx frequently shows *catarrhal irritation*, and the patient may complain of dryness or a burning sensation in the throat. Actual *sore throat* may be present at the time of onset, associated with a diffuse *erythematous rash*, suggesting scarlatina.

The Tonsils.—There is a special form of typhoid—*tonsillo-typhoid* or *pharyngo-typhoid*—in which there appear upon the tonsils peculiar patchy elevations, whitish in color, which undergo subsequent ulceration. It is not improbable that these lesions result from the local action of the specific bacillus in an unusual situation. *Thrush*, affecting the mouth, throat, and even extending to the esophagus, not infrequently arises as a complication. The *tongue* is heavily coated, as a rule, with a yellowish-white fur; later it clears off near the edges and tip, while the center becomes dry or brown and sometimes fissured. The lips are also dry, sometimes fissured, and often covered with dry, black crusts (sordes). *Ulcerative stomatitis* may occur if the mouth be not kept clean. Under these circumstances secondary lesions evincing unpleasant and even serious symptoms may also arise in organs more or less remote from the mouth, and among these is *parotitis*, which is most probably caused by the staphylococcus or streptococcus reaching the parotid gland by way of Steno's duct. The condition is betrayed by such symptoms as pain, redness, and finally by fluctuation, with an elevation of the bodily temperature. It is a late-appearing development, and is usually unilateral, though it may be bilateral. *Suppurative otitis media*, a rarer complication, arises in a similar manner, the pathogenetic agents reaching the ear through the Eustachian tube.

(*d*) **Respiratory System.**—As pointed out in the section on Pathology, *bronchitis* is almost invariably present, but in the majority of instances the cough is slight. The condition is recognized by the existence of numerous sibilant râles. Very rarely is it a striking feature in the early stage of typhoid fever, and then, except this fact be remembered, room for error of diagnosis exists. Moreover, in cases that are improperly treated the bronchial secretions are apt to accumulate, and a well-marked bronchitis may be the result. It may be said, however, that, as a rule, bronchitis does not assume a severe type in cases which receive proper attention from the beginning, provided the patient be not unusually stupid or unconscious. When the nervous phenomena are pronounced, however, and the patient maintains the dorsal decubitus (expectorating little or nothing), bronchitis of a severe grade and affecting the smaller bronchi is almost inevitable. The occurrence of an intense generalized bronchitis is also favored by certain other conditions, such as corpulence, advanced age, and emphysema. These cases are apt to lead to lobular infiltration—*aspiration-pneumonia.*

Lobular pneumonia may take on a putrid nature and the consolidated area may become *gangrenous*. As a sequel, *pleurisy* with effusion or empyema may originate in consequence of the infiltrated lobules being contiguous to the pleura. If the lobules occupying the periphery of the lung become gangrenous, perforation of the pleura, leading to *pyopneumothorax*, may result. As pointed out by Gordinier and Lartigau,[1] in the majority of instances of typhoid pleurisies the aspirated fluid has been found to be purulent in character. Lobular pneumonia may be attended with *hurried breathing* or troublesome *cough.*

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[1] *Amer. Journ. Med. Sci.*, January, 1901.

More commonly, the *local symptoms* are either wanting or feebly marked, and this is especially true of the severer forms of lobular pneumonia, which occur in patients in whom profound nervous prostration coëxists with more or less complete unconsciousness. Sole reliance is to be placed upon the results of a *physical examination*, which even in the absence of subjective symptoms should be repeated daily. Points or surfaces of dulness, most marked near the bases of the lungs and frequently on both sides, are found on percussion. Fine moist râles, most marked toward the bottom of the thorax, form a very characteristic sign, and are heard in every direction on auscultation. In order to ensure a certain diagnosis of lobular pneumonia both the circumscribed dulness and moist râles must be found present in the same situation.

Lobar pneumonia is a not uncommon complication. In a small percentage of cases it develops early, and is most probably the result of a special concentration of the poison in the lungs, giving rise to the so-called *pneumo-typhoid fever* (*vide infra*, Varieties). These cases are often mistaken for primary lobar pneumonia. Their onset may or may not be marked by a rigor, but it is usually more gradual than that of primary lobar pneumonia. Characteristic typhoid symptoms soon follow, and at the end of the first week or thereabouts the pulmonary symptoms gradually abate, while those most characteristic of typhoid (enlarged spleen, roseate spots, etc.) occupy the foreground. Lobar pneumonia more often develops as a late complication—in the second or third week, or even during convalescence—but it is not attended by the usual phenomena (rigor, cough, rusty expectoration, intense chest-pain, etc.), and hence may be easily overlooked. The temperature may be either quite elevated or at times only moderate. The *diagnosis* is to be made from the physical signs, together with the peculiar temperature-curve, which may present marked irregularities. Pulmonary infarction and abscess of the lungs are occasional complications.

Hypostatic congestion of the lungs, due to enfeeblement of the cardiopulmonary circulation, is a comparatively frequent concomitant, appearing in the third week of the disease. It is generally bilateral, affecting the base of the lungs, and is promoted by the effects of gravitation. It is almost always associated with more or less edema of the lungs. The subjective symptoms, including fever, are usually negative, while the objective signs are those of partial or complete consolidation of the bases (defective resonance or dulness, broncho-vesicular breathing, with moist râles). *Miliary tuberculosis* rarely develops as either a complicating affection or, it may be, as a sequel. A spasmodic or jerking inspiration when pneumonia does not exist is a precursor of coma (Flint).

Laryngitis, indicated by hoarseness, is an occasional complication. The laryngeal ulcers may extend in depth to the perichondrium, and promote that grave though not necessarily fatal condition, *perichondritis* leading to necrosis of the cartilages with edema of the glottis. The symptoms of laryngeal stenosis are apt to develop.

Epistaxis appears early in a large number of cases, and is a valuable diagnostic symptom. It may also occur during the fastigium, and particularly toward the latter part, when it is of little or no diagnostic, but of grave prognostic, significance. It is apt now to be troublesome, and may even, as in a case I saw recently with Dr. I. Newton Snively, be so persistent as to lead to a fatal issue.

(*e*) **The circulatory system** presents no characteristic symptoms. The *heart-sounds* are but little affected, as a rule. In cases of asthenic type and in severe typical instances the first sound of the heart may grow quite feeble and ultimately resemble the second (embryocardia). Under these circumstances a soft systolic murmur may be faintly heard along the left border of the sternum. Among occasional *complications* presented by the heart is pericarditis, and still less frequent is endocarditis. Myocarditis is somewhat more common. The sudden development of circulatory collapse in the course of typhoid fever, as previously noted, may be due chiefly to myocardial inflammation; and there may be a brief though alarming derangement of the heart action, due to functional disturbances of the sympathetic and pneumogastric nerves.

The *pulse* is accelerated, but not, as a general rule, in proportion to the height of the temperature until late in the affection. Its average rate is from 84 to 108, but it may go much higher, and when the pulse is maintained at 130 or more for days together it is of ominous import. The temperature, moreover, may be of average height, while the pulse is normal or only slightly quickened throughout; and hence the increase in the pulse-rate cannot be due solely to the elevation of temperature. As before intimated, the extreme debility which comes on during the third week in severe cases may have, as one of its manifestations, a very rapid pulse, reaching to 160 or more (the so-called running pulse), and with or without marked irregularity. Slight *irregularity* is sometimes observed, either during the height or decline of the affection, but as a rule this soon disappears, and proves of no serious consequence. Marked temporary accelerations are often caused by undue exertion or mental excitement. The lowered arterial tension is shown by a dicrotism of the pulse—a symptom which is not characteristic of typhoid fever, however, since it is well marked in other acute infectious diseases, though less frequently. During convalescence the pulse often becomes subnormal in rate, and *brachycardia* is oftener a sequel of typhoid than of any other acute infectious disease.

Venous thrombosis occurs in 1 per cent. of all cases (Murchison). Its most frequent seat is the left femoral, and the next most frequent the right femoral vein, and it is the immediate result of cardiac weakness, except perhaps in those rare instances that arise early in typhoid. For the latter no definite cause has as yet been found. Coming on, as it usually does, during convalescence, it manifests itself by swelling and edema of the extremity affected. There are *pain* in the thighs and calves, and tenderness (on pressure) over the course of the femoral vein, and often in the region of the calf of the leg as well. It causes *fever* of a moderate grade and irregular type, and then in the course of from two to three weeks the swollen member may be reduced to its normal dimensions. This complication is usually not of a serious nature. Occasionally, however, clotting extends into the pelvic veins, or even into the vena cava, when the condition becomes more serious, and sudden death has resulted from the detachment of emboli. The thrombus may undergo suppuration, to which systemic septic infection may be a secondary event.

Thrombosis, and less frequently embolism, in the arteries, combined with renal, splenic, and pulmonary infarcts, may be encountered in typhoid fever.

The large or small arteries may become obliterated, either by embolism or thrombosis, in extremely rare instances, but whether the thrombosis under these circumstances is brought about by a peculiar condition of the blood which favors clotting, or by a localized arteritis, or in consequence of the operation of these combined factors, is not definitely known. If, as is usual, the femoral artery be involved, the blood-supply to the foot and leg is cut off and *gangrene* of those parts must follow. The condition may be bilateral. It may be detected early, owing to the absence of a femoral pulse, before the signs of gangrene appear, but the condition is highly dangerous.

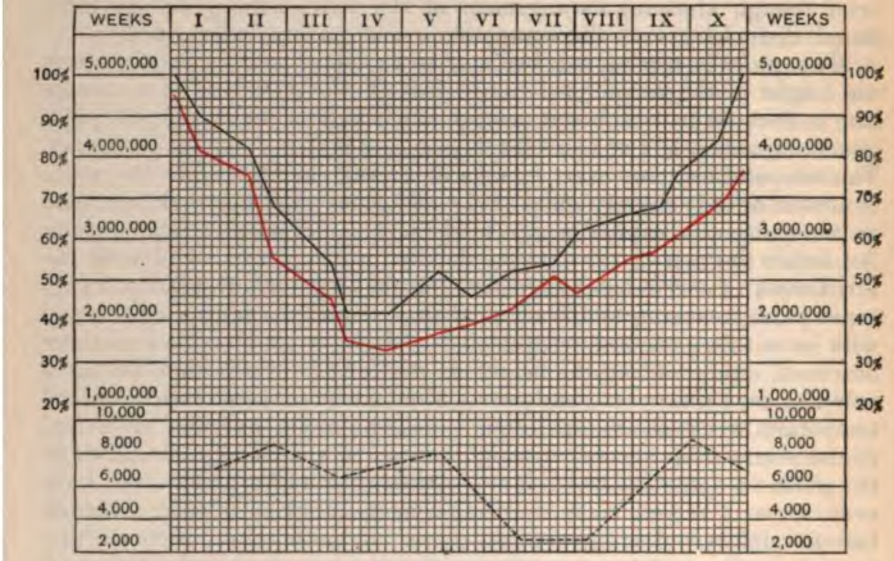

FIG. 4.—Chart illustrating the blood-changes in typhoid fever: upper curve, red corpuscles; middle curve, hemoglobin; lower curve, white corpuscles.

The *blood* presents certain changes, some of which are valuable for diagnostic purposes. In those rare cases in which copious diarrhea or profuse sweats are present the red corpuscles may be relatively increased in number during the febrile period, owing to loss of water. There is, however, in the majority of instances, little or no decrease in the number of red corpuscles till the end of the second week. They are markedly diminished, as a rule, during convalescence. Indeed, the oligocythemia may attain to an immoderate degree.

There is a greater relative decrease in the amount of hemoglobin than in the number of red corpuscles, and the restoration of the hemoglobin in the convalescent period takes place more slowly than that of the red corpuscles. The number of white corpuscles remains at or a little below the health standard until late convalescence, when it sinks to a moderate degree—furnishing a count of about 2000 per c.mm. This fact is an important aid in the differentiation of typhoid fever from acute *inflammations* and *infectious* (febrile) *affections* accompanied by exudation, in which leukocytosis is marked, and from all *suppurative processes* in which the polynuclear neutrophiles are moderately increased. Naegeli[1] found an early neutrophilic leukocytosis of moderate degree which rapidly decreases. In the second stage neutrophiles and lymphocytes are still further decreased, the former at last disappearing, while the latter begin to increase again, and so continue until defervescence. During the decline of the fever the neutrophiles reach their minimum, the lymphocytes are greatly increased, and the eosinophile cells gradually return to their normal number. After the disappearance of the fever a lymphocytosis may occur. The blood-characters in typhoid are shown in the accompanying chart (Fig. 4).

(*f*) **Nervous System.**—The persistent *headache* that is almost always present is among the most prominent symptoms during the first week, but it diminishes steadily during the early part of the second, as a rule. It affects the temporal, occipital, and cervical regions, and when the onset is comparatively sudden, pain in the back is also a more or less conspicuous feature during the first few days of the illness. In a small class of cases, however, the effects of the typhoid bacilli or their toxins are manifested solely in the nervous system from the very onset. In such there are violent headaches, retraction of the head, rigidity, photophobia, and muscular twitchings (rarely convulsions)—all of which symptoms indicate *meningitis*. The diagnosis of meningitis as a complication must be made with extreme caution, since, no matter how complete the clinical picture may be, the post-mortem examination usually reveals a total absence of meningeal inflammation. It must not be forgotten, however, that meningitis is one of the rarest of the complications of typhoid fever. *Vertigo* may accompany the headache, but it seldom outlasts the latter. Before delirium manifests itself wakefulness and restlessness at night are very annoying, and later the same symptoms may be observed associated with the delirium. In cases of moderate severity *mental dulness*, and even *actual hebetude*, are almost invariably present. Questions are apt to be answered inconsistently and in monosyllables.

Delirium is frequent in the severer cases. It is, however, not an uncommon event for those of moderate severity to be free from this symptom throughout the attack. It is, as a rule, most marked at night or at some time when the patient is left alone. His delusions may impel him to attempt to leave his bed, but more commonly there is *mild* or *noisy delirium*, with more or less restlessness. He may lie somnolent, soliloquizing in a loud whisper (muttering delirium), and this so-called *typhomania* may gradually give place to actual coma toward the close of the middle period of the disease. In not a few cases —mild or severe—*coma* is developed suddenly, and is often a mortal symptom. Still another unfavorable sign is a picking at the bed-clothes or a grasping at imaginary objects (carphologia).

The delirium may assume an hysteric type, the patient usually exhibiting the saddest emotions, and if he be an alcoholic he may be seized with *delirium tremens*. In a case of typhoid fever that I saw recently with Dr. S. W. Morton hysteric delirium developed during convalescence, but did not last more than twenty-four or thirty-six hours.

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[1] *Deutsche Archiv fur klin. Med.*, Band lxvii., Hefte 3 u. 4.

The *motor nerves* also present notable disturbances in association with the sopor and the forms of delirium previously described. Slight twitchings of the muscles of the face and extremities are quite common, and when they affect the tendons of the wrist and fingers the term *subsultus tendinum* is applied. The lips, tongue (especially when protruded), lower jaw, and even the extremities, are often in a state of constant tremor. During this motor irritability the reflexes are increased, but when profound coma comes on they are either largely diminished or totally abolished. The toxins of the typhoid bacillus, acting poisonously upon the nervous centers, are undoubtedly the cause of the nervous symptoms in typhoid.

Nervous complications and sequelæ may arise. Chief among these is paralysis, which is most probably due to neuritis. The lesion may involve one, two, or more nerves, and in this way we may have either a paralysis of one limb or, more rarely, a true paraplegia. *Aphasia* may be a sequel, particularly in children. *Hemiplegia*, due to hemorrhage or a localized encephalitis, may occur either as a complication or sequence of the disease. Following typhoid fever, the patient may exhibit evidences of *mental enfeeblement*, and even *insanity* where a predisposition to this condition has existed; and insanity is relatively more common after this disease than after any others belonging to the same class. I have seen four instances, all of which recovered, while Osler has seen five, four of which ended similarly. It is in most cases, as pointed out by Wood, a confusional insanity, due to exhaustion and impairment of the nutrition of the nerve-centers, while in a smaller contingent it takes the form of a true melancholia. After the conclusion of typhoid, as well as during its course, *neuralgia* affecting the occipital and other cranial nerves is not infrequent. Great hyperesthesia of the skin and muscles is common during convalescence, attacking the lower extremities by preference (Strümpell). The so-called "typhoid spine" (Gibney) has also been observed, and consists in an acute inflammation of one or more vertebræ following typhoid. The chief symptoms are pain in the back and hips of a lancinating character. The point of origin appears to be the small of the back; thence the pains extend paroxysmally up and along the spine and to the abdomen. They subside gradually, leaving the back weak and painful on attempts at turning in bed, etc. Plantar and other skin-reflexes increase, and the knee-jerks are preserved. G. E. de Schweinitz has described at length the *ocular complications and sequelæ* of typhoid fever. Affections of the conjunctiva and cornea and retinal hemorrhage are perhaps the most frequent, although optic neuritis and affections of the uveal tract also occur.

(*g*) **The Urinary System.**—*Urine.*—The urine is lessened in quantity and high-colored, with an increased specific gravity up to the arrival of the stage of decline. About this time, and rarely earlier, it grows light in color, larger in quantity than the normal, and the specific gravity is relatively diminished. Both urea and uric acid are increased during the earlier stages, and sometimes throughout the attack, while during convalescence both are diminished. On the other hand, the chlorids are diminished during the active stages of the disease and increased during its decline. A *febrile albuminuria* is quite common, and the sediment may show an excess of renal epithelium, a few blood-cells, and occasionally renal casts.

Acute nephritis may develop as a complication in the earlier or later course of the disease, and can be recognized to a certainty only by a thorough appreciation of the urinary phenomena. The urine is diminished in quantity, being often scanty, and there may be retention. It contains characteristic morphologic elements (albumin, casts, blood, and epithelium). The development of the *typhoid state* in this affection is rendered much more probable in the presence of this complication, and, moreover, uremic symptoms often put in an appearance at this juncture, and then the situation is really serious. Acute nephritis may arise at one or other of three different periods, and its significance varies with the time of onset: (*a*) at the beginning of the fever, when it often obscures the true nature of the malady. This is the *nephro-typhoid* of the German authors, and will be referred to hereafter (*vide infra*, Varieties); (*b*) in the early part of the fastigium or the second week of the disease. Coming on at this time—an event which I have observed in two instances—its relation to the typhoid bacillus or its toxin is not definable. It is probable, however, that it is to be ascribed to the local effect of the toxin upon the renal tissues. Both of my own instances proved fatal, and in both an autopsy was refused. Wagner[1] has had 5 cases of recovery in succession, but the high mortality mentioned by Amat—10 deaths in 12 cases—is the more common experience. (*c*) Acute nephritis may arise as a sequel of typhoid, when, with the usual symptoms of acute nephritis, there is almost invariably associated a decided edema. In this category of cases recovery is to be expected.

Diabetes mellitus is, in extremely rare instances, developed after typhoid. Hematuria has also been observed as an occasional symptom of the hemorrhagic diathesis.

The *diazo-reaction* of Ehrlich is an aid in diagnosis, but, unfortunately, may be present also in acute phthisis, meningitis, measles, pneumonia, and other fevers. To obtain it two solutions (*a* and *b*) are needed: We mix 1 part of solution (*a*), which consists of a 0.5 per cent. solution of sodium nitrite, with 50 parts of solution (*b*), which consists of 2 grams of sulfanilic acid, 150 c.c. of hydrochloric acid, and 1000 c.c. of distilled water. To this an equal volume of urine is added, and the contents of the test-tube are then thoroughly shaken. A layer of ammonium hydrate is now superimposed, and at the line of contact a ruby or pink ring develops. A more reliable change, however, is a rose-red (pink) hue of the foam. It is present in about 70 per cent. of the cases, and has considerable confirmatory diagnostic value. The reaction appears about the beginning of the second week, sometimes later, and lasts usually until defervescence is well advanced. A brownish ring is given by normal urine.

There is a post-typhoid, diphtheritic *pyelitis* in which the pelves and calices of the kidneys are primarily the seat of membranous exudation, and later of erosion and ulceration. The urine generally contains blood and pus.

Simple *vesical catarrh* is a rare complication, except as the result of catheterization for retention.

Orchitis and *epididymitis* as well as *ovaritis* are occasional sequels. Eshner[1] collected 42 cases of orchitis, of which 29 cases occurred during convalescence.

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[1] *Deutsche Archiv fur klin. Med.*, Bds. xxv. and xxxvii.

[1] *Philada. Med. Journ.*, May 21, 1898.

(*h*) **The Joints.**—Typhoid, septic and rheumatic arthritis may occasionally arise as a complication. The first is usually *mon-articular* (particularly in the hip); the last two commonly *polyarticular*. Keen has collected "in all 84 cases involving the joints."

(*i*) **The Bones.**—*Periostitis*, due to injury and muscular strain and often leading to *necrosis*, is a not very rare sequel of typhoid. The favorite seats are the tibia and ribs, though in a case of my own at the Philadelphia Hospital it affected the os calcis. *Osteomyelitis* may also occur. Keen has collected 216 cases in which the bones were attacked.

(*j*) **The Muscles.**—As in the case of the heart, so the voluntary muscles exhibit hyaline degeneration; also abscesses, in consequence of secondary infection or of infection with the typhoid bacillus itself. Typhoid abscesses likewise result from perforations of the gut.

Associated Acute Infectious Diseases.—**Malarial fever** may be combined with typhoid, though the relationship is not a vital one. In an analysis of 2122 cases of malaria typhoid fever was associated in 8.[1] Many instances of so-called typho-malarial fever would be shown to be pure typhoid by a careful blood-examination, as the presence of chills, sweats, and an intermittent temperature-curve are sometimes observed in this disease (*vide supra*).

Pseudo-membranous inflammation, as above intimated, may occur in the naso-pharynx, larynx, gall-bladder, and genitals. *Measles*, *scarlatina*, and *chicken-pox* have also been known to arise in the course of, or during convalescence from, typhoid fever.

Erysipelas is a rare secondary affection coming on either during the height of the affection or (more frequently) after its close. Typhus fever may be associated with typhoid, but is an exceedingly rare occurrence.

Clinical Varieties of Typhoid Fever.—These are numerous, and may grow out of peculiarities manifested during the course of the affection, as may be observed not only in different epidemics, but also in the same epidemic. The groups of cases described here have reference particularly to the degree of severity of the type, which varies between the wide limits of extreme mildness on the one hand and extreme severity on the other. The course of the disease may also be modified by the occurrence of one or more of its manifold complications.

(1) **The Mild or Rudimentary Form (Typhus Lævissimus).**—Of this variety many cases occur, and especially among children. The characteristic typhoid symptoms are scanty, and at times even entirely wanting. The spleen is almost always enlarged, the roseate spots are sometimes present, while the temperature is moderately elevated and often partakes of the same character as that of true typhoid. The fever, however, may pursue the remittent type. Complications presented by special organs are usually absent, but grave accidents (intestinal hemorrhage, perforation) are not impossible.

The diagnosis is always difficult, owing to the feeble development of the characteristic symptoms, and in the total absence of the latter is out of the question; but the recognition is assured if a causal connection between them and typical cases can be shown to exist, and if the Widal test gives a positive result.

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[1] "The Complications of Malaria," *Journal of the American Medical Association*, vol. xxiv. p. 919, by the author.

(2) **The abortive form** has a sudden onset, and is often marked by fits of shivering. The characteristic features of the disease (enlargement of the spleen, abdominal symptoms, rose spots, etc.) appear earlier than in the usual type, and soon become quite well marked. The fastigium is short, and the temperature, from the seventh to the twelfth day of the illness, declines by a prompt lysis, with profuse sweating. With the rather rapid fall of temperature there is a no less rapid improvement in every other leading symptom. Convalescence is speedy.

(3) **The Ambulatory Form (Latent or Walking Typhoid).**—The patient continues to walk about, either experiencing but slight disturbance or being unwilling to take to his bed. Such cases do not come under the care of the physician in many instances. Others, on account of debility, anorexia, diarrhea, and other vague symptoms, finally consult their physician, who may discover the presence of all the characteristic features of the disease. A third contingent, belonging to this form, continue to move about, or even to follow their usual vocations, till seized suddenly with profuse intestinal hemorrhage or general diffuse peritonitis following perforation.

(4) **The afebrile** is an exceedingly rare form of the affection—in this country at least. Liebermeister, however, has met with a number of cases at Bâsle, the symptoms being lassitude, depression, headache, neuro-muscular pains, anorexia, slow pulse, furred tongue, constipation or diarrhea, with enlargement of the spleen and roseate spots. These cases are often confined to bed, and there are occasional attempts at evening exacerbations of temperature (100.5° F.—38° C.).

(5) **Severe or Grave Forms.**—These may be dependent either wholly or in great part upon the degree of virulence of the typhoid poison. Under these circumstances there will be a profound intoxication of the system, as shown by high temperature, violent nervous symptoms, and great prostration. The grave types may arise in the course of cases of average severity from the development of serious complications. Again, to serious forms belong those cases that begin with the characteristic symptoms of a localized inflammation—*e. g.* the *cerebro-spinal form*, in which the nervous symptoms greatly predominate at the onset; the *nephro-typhoid* (before alluded to), in which the preliminary symptoms are those of acute Bright's disease; the *pneumo-typhoid* (*vide supra*), which begins with the manifestations of a more or less frank pneumonia.

Pleuro-typhoid.—The cases begin as an acute pleurisy, having its special characteristics, and these are followed, soon or late, by the diagnostic evidences of typhoid fever. Talamon[1] distinguishes these cases from simple pleurisy by the intensity and continuous course of the fever, by the general depression, headache, and vertigo, and by the sleeplessness. Should any doubt remain, it will be dissipated by the eighth or the ninth day by the presence or absence of rose-colored spots, by the enlargement of the spleen, and of the sero-reaction (*vide infra*). Eiselt[2] has described a special form under the name *spleno-typhoid*, in which the spleen is enormously enlarged without characteristic intestinal lesions. Perisplenitis with adhesions may be noted. The fever is often of remittent type.

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[1] *La Médecine moderne*, Paris, 1891.

[2] *La Semaine médicale*, August 27, 1891.

The *sudoral form* and *tonsillo-typhoid* (before described) also belong to this category. *Typhoid septicemia* may present the grave symptoms of an extreme intoxication, often merging into the typhoid state. Visceral and cutaneous hemorrhages may be superadded. Many circumstances connected with the individual influence decidedly the general course of the affection, and these may be expressed in part in the several forms following, which are based upon such factors as *age*, *habits*, etc.

(6) **Typhoid Fever in Children.**—The onset is rather more abrupt than in the adult, and certain prodromal symptoms are very generally absent (epistaxis, chilliness). On the other hand, bronchial and nervous symptoms are often quite pronounced. Again, during the fastigium some of the usual typhoid features may be missing—*e. g.* diarrhea and tympanites—while the eruption may either be entirely wanting or less copious than in older subjects. Intestinal hemorrhage is rare and perforation almost never occurs. The mortality is not over 1 per cent.

(7) **Typhoid Fever in the Aged.**—The course of the affection presents no regular type. The temperature is not as high as usual, but there is marked adynamia and serious danger from certain complications, such as pneumonia, nephritis, coma, and the like.

The diagnosis is difficult, owing to the prominence of the nervous and pulmonary symptoms on the one hand, and the frequent absence of the more characteristic symptoms of typhoid on the other (rash, enlargement of the spleen, and peculiar temperature-curve).

Diagnosis.—Unless all the chief characteristic features be present with a clear history, it is a golden rule not to make a positive diagnosis. Obviously, then, the physician at the first visit, often about the close of the first week, cannot, in many cases, diagnosticate typhoid with absolute certainty. If the case have been a typical one, the history of the gradual development of the disease, marked by such symptoms as languor, anorexia, headache, dulness, slight chills, increasing fever, and sometimes nose-bleed, will be obtained, and justify a strong suspicion of typhoid. When, in addition, diarrhea and the objective symptoms, splenic enlargement, tympanites, gurgling, with tenderness in the ileo-cecal region, are present, the diagnosis of typhoid is made highly probable. After the lapse of a few days—the beginning of the second week—the roseate spots and sero-reaction (the latter sometimes earlier, *vide infra*) usually appear, and then all doubt is removed. The typhoid bacilli can sometimes be cultivated from the rose-spots several days before the Widal reaction is obtainable (Richardson). In atypical cases a positive opinion must often be withheld till an advanced stage is reached. In such instances the occurrence of intestinal hemorrhage or a characteristic decline by lysis is helpful. To show a causal relation between an obscure case and one that is clearly typhoid leaves little to be desired. The diagnosis should include the particular stage of the disease.

Briefly, the most trustworthy diagnostic features are the gradual onset, peculiar temperature-curve (made up of the "step-ladder" stage of development, the continued type of the fastigium, and the decline by lysis), enlarged spleen, the rose-colored spots, and the sero-reaction.

Serum-diagnosis.—Investigations by Pfeiffer upon the specific bactericidal substances developed in the blood of animals immunized by injection of typhoid bacilli have furnished a reliable means of diagnosis of this disease from blood-serum. It remained, however, for Widal and others to show that if to a drop of blood-serum, or to a drop of water containing a solution of dried blood from a typhoid patient, a moderate number of typhoid bacilli were added, a specific reaction occurred.

Johnston of Montreal has simplified the technic: The blood is obtained upon a clean glass slide from a needle-prick of the ear or finger of the suspected case. It is allowed to dry, and is then carried to the laboratory. A loop of bouillon-culture of genuine typhoid bacilli is placed upon a clean cover-glass, and to this is added a large loopful of a watery solution of the dried blood-specimen. The cover-glass is inverted over the concavity of a hollow slide and sealed at the edges with melted vaselin. Under the microscope, with a high-power dry lens or with a one-twelfth oil-immersion lens, a rapid clumping of the bacilli in the hanging drop can be observed,[1] and their motions cease almost instantly.

It is of paramount importance that the technic be perfect. Again, Widal himself observes that this reaction is not limited to the bacillus typhosus—"That it can occur, but in different degrees, with related species." The balance of testimony goes to prove that the reaction is one of infection, and not of immunity.

Diagnostic Value.—A review of all preceding work in the Widal reaction reveals the broad fact that there is a general consensus of opinion as to its great clinical value. The large statistics of Kneass and Stengel, based on 2283 cases, and published in the *American Year-Book of Medicine and Surgery* for 1898, coupled with more recent available figures, show the presence of the reaction in 95.2 per cent., and no reaction in non-typhoid cases in 98 per cent. Widal, however, obtained universally positive reactions in 162 cases of typhoid fever. Cabot and Lowell[2] found the results to be negative in 204 cases of disease other than typhoid. Whilst somewhat contradictory results are occasionally recorded in the literature, accurate technique and the observance of coincident cessation of motion and clumping in the cultures place the diagnosis of typhoid fever beyond question.

Of 230 cases examined, 219 gave a positive result (Anders and McFarland).[3] In 128 of these cases this result was obtained prior to the appearance of the rose spots, or before the eighth day; in 36 cases the first reaction occurred during the second week; in 45, between the seventeenth and twenty-first days of the disease; in 8, not until the twenty-fifth day, and in 2 cases as late as the twenty-eighth day.

Interfering Conditions.—In the first place, a previous attack of typhoid fever may produce a reaction that may lead the careless observer into error. In 39 cases of pure typhoid tested at periods of from one to eighteen months after defervescence, 13 reacted positively (Cabot and Lowell). It may be possible for the scene to be dominated by some other morbid process (tuberculosis, etc.) when typhoid fever is present. Again, exceptional cases occur with a characteristic symptom-complex and course, yet give no reaction throughout. Brill has reported 17 cases of this sort; in such cases, however, the examinations must be repeated until after convalescence is completed, since the appearance of the reaction may be greatly retarded. A. C. Abbott[1] reports that, according to the records of Widal reactions carried out in 4154 cases in the city laboratories of Philadelphia, the error does not exceed 2.8 per cent.

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[1] *Medical News*, Nov. 14, 1896.

[2] *Boston Med. and Surg. Jour.*, Feb. 18, 1899.

[3] *Philada. Med. Journ.*, April 8, 1899.

The cases that begin with the well-defined local inflammatory lesions previously referred to (tonsillo-typhoid, pneumo-typhoid, pleuro-typhoid, nephro-typhoid) cannot be recognized at the outset. The same local inflammatory conditions may, independently of typhoid fever, be combined with a genuine typhoid state. In all instances of typhoid fever in which, at the time of onset, localization occurs the degree of fever and prostration are apt to be out of proportion to the local symptoms, and the former are apt to continue after the subsidence of the latter. A careful observation of the symptoms after the first week will usually detect undoubted symptoms of typhoid. I have found that the Widal test decides some of these cases.

The bacilli may be obtained in quantities from the stools in 50 per cent. of the cases.

Differential Diagnosis.—(1) **Typhus fever** is to be differentiated by its appearance as an epidemic, by its sudden onset, by the deeper stupor, the besotted expression of the features, the injected conjunctivæ, the contracted pupils, the appearance on the fourth day of maculæ which are transformed into petechiæ; by the shorter course, the abrupt termination by crisis, and the absence of the Widal reaction.

(2) **Acute miliary tuberculosis** has been, and still is, frequently mistaken for typhoid fever. The former is to be differentiated from the latter by the greater frequency of the pulse and respirations, the prominence of the cough, and in some instances by the bloody expectoration; by the pronounced cyanosis, the presence (sometimes) of choroidal tubercles, and the existence (constantly) of leukocytosis, which does not occur in typhoid. *Blood-examinations* have occasionally shown the presence of the tubercle bacillus. There is an absence of the peculiar temperature-curve, of the characteristic lenticular spots and the Widal reaction, and abdominal symptoms of typhoid.

(3) **Malarial fever** may assume the continued form of fever, and there are typhoids that affect both remittent and intermittent malarial fevers. The latter can be differentiated by the therapeutic test and the detection of Laveran's hematozoa in the blood.

Should *typho-malarial fever* be suspected, and should the typhoid symptoms be unequivocal, the finding of the malarial organism will differentiate the hybrid from pure typhoid.

(4) **Relapsing fever** is distinguished by its abrupt onset with rigor, high fever, pain in the epigastrium; by its brief duration, termination by crisis, and the occurrence of a relapse at the end of a week; by the absence of the characteristic eruption and sero-reaction; by the fever-curve; and by the nervous symptoms of typhoid. The finding of the spirilla, however, reliably discriminates relapsing fever.

(5) **Meningitis.**—In striking contrast with the specific typhoid symptoms meningitis exhibits marked hyperesthesia, intolerance of light and sound, exaggerated reflexes, and often muscular rigidity before the

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[1] *Philada. Med. Journ.*, February 25, 1899.

stage of effusion; also restlessness, peevishness (unlike the dulness observed in typhoid patients), vomiting, and constipation (*vide* Acute Miliary Tuberculosis). The temperature maintains a lower level on the average, and is more irregular in type than in typhoid; the pulse is more irregular, and the nervous symptoms assume greater prominence in the earlier stages, particularly headache and delirium. On the other hand, true typhoid symptoms are wanting in meningitis.

(6) **Tuberculous meningitis** gives a characteristic previous or family history, occurs in young subjects, and the tendon and cutaneous reflexes exhibit wide variations as to intensity, within brief periods and throughout the whole attack. An examination with the ophthalmoscope may reveal choroidal tubercles. There is a leukocytosis and the Widal reaction is missing.

(7) **Catarrhal enteritis** in children, with prominent abdominal symptoms, may simulate typhoid fever very closely. In the former the symptoms are all gastro-intestinal, save perhaps the occurrence of slight febrile disturbance and certain nervous phenomena, while typhoid fever manifests a wider range of symptoms (some of which are peculiarly its own—notably the greater prostration, marked fever, enlargement of the spleen, the sero-reaction, and the characteristic eruption). In young children the last-named symptom may be either wanting or atypical, in which case the existence of enlargement of the spleen coupled with other phenomena, particularly the Widal reaction, will suffice.

(8) **Salpingitis** on the right side may resemble typhoid. In the former there is usually a clear history either of antecedent vaginitis or of an abortion, and there exist special evidences of local peritonitis, with which may be associated the typhoid state, but not the classic features of typhoid fever. A digital examination *per vaginum*, however, is necessary to certitude of diagnosis in salpingitis.

The diagnosis between typhoid fever and typhoid pneumonia, and appendicitis will be considered hereafter.

Prognosis.—As in all other acute infectious diseases, so in typhoid, the prognosis depends upon three main considerations:

(1) **The severity of the type of the infection**, which is indicated in great measure, though not solely, by the degree of fever. A temperature of 106° F. (41.1° C.) is a serious symptom, and, if maintained at this point for a few days, an almost certainly mortal one. I have not seen a single instance in which the temperature has touched 106° F. (41.1° C.) for two or three successive days that has recovered. If the temperature mounts to and keeps at 105° F. (40.5° C.) for a longer period than three or four days, the prognosis should be made with due reserve. Temperatures above 106° F. (41.1° C.) I would regard as offering no hope of recovery. When the fastigium is prolonged, even though the fever be not exceptional, the prognosis is usually grave; while, on the other hand, marked nocturnal remissions are of favorable omen. A sudden, deep fall, however, implies danger (intestinal hemorrhage, collapse).

The researches of Isaac Ott have taught us not only that fever is due to an agent from within or without, which deranges the harmony of the thermotaxic, thermogenetic, and thermolytic apparatuses, increasing primarily tissue-metabolism, but also, that while high temperature is an indication of danger in specific fevers, it is not always the cause of it. He regards high temperature as being only a part of an infectious process, and points out that the thermotaxic centers of the cortex may be so disordered as to alter the harmony between the heat-production and heat-dissipation. Under these circumstances a specific fever of severe form may be associated with a slight elevation of temperature.

The power of resistance to the influence of a greatly elevated temperature is quite reliably indicated by the condition of the heart. So long as the pulse is regular and its rate does not exceed 110 or 120 beats per minute, the outlook is favorable. When, however, the pulse maintains an average rate of 130 or more—a condition with which there is usually associated some degree of cyanosis, pulmonary congestion, and edema—the outcome is to be regarded as doubtful. Collapse is apt to follow the occurrence of sudden complications (perforation, hemorrhage), but it may also arise independently of such a cause. It is ominous.

Serious types are also shown by the occurrence of certain nervous symptoms, that may assume unusual gravity. This is particularly true of delirium, stupor, and the symptoms of motor irritation.

(2) **Circumstances of the Patient.**—Certain individual peculiarities render the prognosis highly unfavorable. It is *bad* in very *fat* persons. In such cases there is a great and constant danger of sudden collapse, and this fact also holds to a less degree with reference to those persons who are subjects of certain chronic diseases (Bright's disease, heart-disease, gout, emphysema).

Age is an influential modifying factor. After puberty the gravity of the disease increases with increasing years. Indeed, it may be said that, as a rule, typhoid has an unfavorable prognosis in persons past forty years, and chiefly for the reason that at this time of life there are dangers from an added liability to pulmonary complications and failure of cardiac reserve. In children (*vide* Clinical Varieties) the tendency to hemorrhage and peritonitis is reduced to a minimum, while the disease shows little tendency to assume a grave type. Hence childhood is the most favorable period.

The **puerperal state** renders a typhoid patient liable to many accidents and peculiar complications, and it seems that independently of pregnancy the disease is more fatal among females than males. *Chronic alcoholism* is apt to be complicated with *delirium tremens*, often preceded by *pneumonia*, and to the latter disease the patient is very prone, perhaps to an equal extent with heart-degeneration and exhaustion.

Environment affects the prognosis, poor sanitary arrangements and poor attention greatly diminishing, and the opposite conditions greatly augmenting, the chances for recovery. Improved methods of treatment in recent years have also effected a decided lowering of the death-rate. Here it may be said that the average mortality of typhoid is from 8 to 10 per cent., as against 15 to 20 per cent. formerly. It must ever be remembered, however, that epidemics differ widely as to their mortality list—a fact which makes a precise statement regarding the question an impossibility.

(3) The third and last consideration is **the presence or absence of dangerous complications and accidents.** These have all been enumerated and their prognostic significance stated (*supra*). Merely to reiterate

some of those that lend fresh peril to the typhoid patient, arranging them with some regard for the order of their relative gravity, may prove helpful to the student. They are—perforation with diffuse peritonitis, intestinal hemorrhage, lobar pneumonia, lobular pneumonia, sudden collapse (due to cardiac weakness), excessive tympanites (often with marked diarrhea), and hypostatic congestion of the lungs.

Relapses of Typhoid Fever.

A relapse is a repetition of all the characteristics of typhoid after the latter has run its course. As a rule, the return occurs from one week to ten days after the beginning of convalescence, though it may be either earlier or later; and occasionally a relapse develops before the temperature has become normal. This is termed an *intercurrent relapse.* The cause of relapses is a reinvasion of the blood by the typhoid bacilli or their secretions; but whether this is attributable to a reinfection from without or from within (most probably the latter) cannot be definitely stated. The pathologic lesions differ in no essential way from those described as belonging to the primary attack, but the stages through which they pass are not quite so long.

In the *interval* between the primary attack and the relapse there may be present suspicious features, such as a slight enlargement of the spleen, a trivial evening rise of temperature, an unnatural apathy or dulness, and a more profound prostration than is usual. In the majority of instances, however, there are no premonitory symptoms. The *onset* is rather more sudden, and rigors are more common, than in primary typhoid. The temperature, however, rises in the characteristic "step-ladder" fashion, reaching the fastigium in two or three days, and the same relative abridgment of the fastigium and defervescence is observed. It follows that a relapse has a shorter duration than a primary attack, and, indeed, it rarely exceeds two or three weeks. The temperature may, however, touch a higher limit in the relapse than in the primary attack; but, with rare exceptions, when the primary typhoid is of average or even greater than average severity, the temperature in the relapse does not reach an equal height. The characteristic rash appears earlier —from the second to the fourth day—and is somewhat darker and coarser than that of the first attack. The spleen swells rapidly. The *intercurrent relapse* sets in while the temperature is declining; the fever again rises, and may become higher than in the primary attack.

Diagnosis.—Upon the points that are distinctive of a primary attack of typhoid fever rests the important diagnosis between a relapse and a recrudescence (*spurious relapse*). The latter is usually attributable either to errors in diet, to undue muscular exertion, or to great mental excitement; and, whilst it occurs during convalescence, it seldom lasts longer than one, two, or three days, and is not characterized by the diagnostic symptom-group of a relapse (peculiar temperature-curve, enlarged spleen, and specific eruption).

The **prognosis** of relapses depends very much upon the severity of the primary attack, those following severe attacks being relatively milder than those that follow the rudimentary, primary attacks.

The frequency of relapses differs widely in different epidemics.

Hence the fact that the percentage of relapses as estimated by different authors ranges from 3 to 15 per cent. need excite no surprise. The relapse may repeat itself once, twice, or even thrice, and two relapses occur in about 1 per cent. of the cases. In a case which I[1] reported three successive and typical relapses occurred. The pale line or ridge which was mentioned (*vide* Clinical History) as noticeable in the nails after typhoid occurs similarly after each relapse, and in the afore-mentioned case of my own four distinct whitish, transverse ridges were noted. Da Costa has recorded five relapses in two cases.

Recurrences.—The term recurrence should be applied only to those instances in which successive attacks are separated by longer or shorter intervals after complete recovery from a previous or the primary attack. Typhoid fever usually bestows complete and lasting immunity against subsequent attacks, but this is not an invariable rule. Eichhorst has studied 600 cases, and found that in 28 of the number (4.7 per cent.) a second attack occurred. I have seen two typical attacks of typhoid fever in two different persons, the intervals between the cases having been five and eight years respectively. Very rarely three separate attacks have occurred in the same person, and a second is usually milder than the first attack.

Treatment.—(*a*) **Prophylaxis.**—Modern hygienic resources happily enable us to minimize the number of occurrences of typhoid, and reports of the typhoid cases in a city may be taken as a safe indicator of its sanitary status. On the same principle it has been found that whatever tends to better the sanitary arrangements of a city diminishes to a corresponding extent the prevalence of the disease, particularly improvements affecting the water-supply and drainage.

The best means that can be employed during the attack, with a view to limiting the spread of typhoid, is *disinfection*, and the following brief description comprises its essential points as applied to this disease:

Disinfection in typhoid may conveniently be divided into (*a*) that of the excreta (stools, urine, vomitus, and sputum); (*b*) of the bed and its coverings; (*c*) of the patient and the sick-room. While all of these subdivisions are of the greatest importance in the treatment of a case, *the disinfection of the excreta* (*a*) is perhaps most frequently overlooked or most carelessly performed, and hence the importance of the statement that all stools and urine voided by the patient, as well as all vomitus and sputa, should invariably be treated in the following manner: The excreta should be received in a vessel that can be thoroughly disinfected inside and out with any one of several standard solutions, of which that of chlorinated lime is the most effective and satisfactory. Bichlorid of mercury (1 : 500) also may be used, but, as it requires a much longer time and forms an insoluble compound with the albumins in the feces and sputa, it is inferior to the solution of chlorid of lime, which is now very generally used in a strength of six ounces per gallon of water. A 5 per cent. solution of carbolic acid is also employed.

It is my custom to order that one pint of the chlorinated lime solution be placed in the bed-pan (or other appropriate receptacle) *before* the discharges are received therein, and from one to two pints *after*. The whole is thoroughly mixed by stirring and shaking, care being taken that all solid masses are broken up. The vessel is then allowed to stand for three hours before it is emptied into the water-closet. If the mercuric-chlorid solution be employed, at least six hours must be allowed for thorough action upon the excreta. The urine must be treated in the same conscientious manner as the feces.

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[1] *Med. and Surg. Reporter*, vol. xlvii. p. 66.

Gwyn[1] has given the results of most recent investigations into the question of typhoid bacilli in the urines of typhoid-fever patients: They are present in from 20 per cent. to 30 per cent. of the cases, and may be exceedingly numerous. The organisms may persist for months or years. For the disinfection of the urine in the bladder, urotropin is serviceable when administered by the mouth. As an irrigation, Gwyn recommends mercuric-chlorid solutions (1 : 100,000 to 1 : 50,000).

(*b*) It should be an invariable rule to change the bed- and body-linen daily, and as often as soiled. The mattress should be protected by a rubber cover, and this, together with the soiled linen and blankets, should be received in a sheet that has previously been dipped in a 5 per cent. solution of carbolic acid. The rubber sheets are to be washed with the carbolic-acid solution, but all other bed-clothes must be boiled for half an hour. When the patient leaves the sick-room the mattresses are to be fumigated and aired daily for a week, and the rubber covers and bedsteads washed with a solution of mercuric chlorid (1 : 1000).

(*c*) After every stool the patient should be cleansed with a compress of cloth or cotton wet with a solution of mercuric chlorid (1 : 2000) or of carbolic acid (1 : 40). The bed-pan and hopper are to be similarly treated, and the cloths used are to be immediately burned. Fitz recommends that the feeding utensils be cleansed in boiling water immediately after using. It is of advantage to devote adjoining rooms to the use of the patient, in order that the windows may be opened in the one not occupied by him, and the other ventilated through it.

Since it is well known that many epidemics are directly traceable to the drinking-supply of water and milk, it is necessary that all water and milk used by the patient and other members of the household be boiled for half an hour before being ingested; and if an epidemic be prevailing, the community at large should join in this precaution.

The municipal authorities possess in thorough filtration a power that might and should be used to advantage, as has been well shown by the improvement effected in certain water-supply and sewer systems—*e. g.* in Vienna, where by purification of the water-supply the death-rate in typhoid fever was reduced from 12.5 per 10,000 to 1.1 per 10,000.

Isolation of Patients.—I am of the firm belief that it is advisable to isolate typhoid cases as far as possible—*e. g.* in hospitals, to keep them in special wards; in private families, in special apartments, into which none but the attending physician and the nurse should enter. There is incontestable proof that typhoid fever is feebly contagious.[2]

Prophylactic Inoculations.—Encouraging results have also followed the *preventive inoculation* of healthy persons with typhoid virus, and Pfeiffer believes that this mode of prophylactic treatment promises to render great service in future epidemics. A. E. Wright, during the siege of Ladysmith, reports 10,529 men not inoculated. Of these, 1489

[1] *Philada. Med. Journ.*, January 12, 1901.

[2] *Philada. Hosp. Report*, 1891, vol. i. p. 149, by the writer.

developed typhoid fever, with 329 deaths. Of 1705 inoculated men, 35 developed typhoid, with 8 deaths. Pfeiffer has also had considerable success in inoculating human subjects with the virus.

(*b*) **Treatment of the Attack.**—(1) The **general conduct** of the case, including skilful nursing, is of paramount importance to the typhoid patient. He should be put to bed as soon as the indications point to this disease, and kept there continuously in the recumbent posture till the end of the attack. The sick-room should have a sunny exposure if possible; should be cool and well ventilated, though free from strong currents; and perfect cleanliness both of the room and of the utensils employed in the management of the case should be attempted. The bed should be provided with a woven-wire mattress, upon which should be placed one of hair. A rubber cloth is spread beneath the sheet, and the latter kept smooth in order to lessen the danger from bed-sores. A seriously ill patient should lie on an air-cushion or, better still, a water-bed, and to avoid bed-sores he should be instructed to turn gently to either side from time to time. His back, hips, and heels should be bathed frequently with a mixture of alum and salt in dilute alcohol. The use of the bed-pan and urinal is an absolute necessity. When a good nurse cannot be had, the attending physician must note *in writing* the directions regarding the disinfection of the excreta, bed-linen, and utensils, as well as regarding the exhibition of the food, medicine, etc. The mouth and throat should be kept clean, since by so doing we obviate unpleasant and even dangerous complications (apthous ulcer, thrush, parotitis, lobular pneumonia, etc.). In the latter the nurse or attendant should wash the mouth and tongue several times daily with a solution of boric acid (3 per cent.), and the throat may be sprayed at equal intervals with a similar solution. A frequent moistening of the tongue and mouth, and particularly the lips, with glycerin and water (equal parts) gives great comfort when they are dry and parched.

(2) An **appropriate liquid diet** is highly serviceable, and the best article of food is milk, which it is well to dilute with plain water (or lime-water), since aërated waters are objectionable in that they sometimes increase the meteorism. The daily quantity should not be less than three pints, and it is important that the stools be examined, since, if the milk be not thoroughly transformed, curds or (on microscopic examination) numerous fat-globules will be seen, in which case a smaller amount should be given. If curds or fat are still seen, the milk should be peptonized. Experience teaches that milk is often better taken and better borne when a little brandy, coffee, or tea is added to it. When milk cannot be taken or digested in sufficient amount, either whey, sour milk, or buttermilk may be tried; and if these be distasteful, we may replace them (wholly or in part) by meat-juices or broths of various sorts, together with one of the standard infant's foods made with milk or water. Albumin-water, prepared by straining egg-white through a cloth and adding an equal part of water, has given much satisfaction in my hands. It may be made pleasant to the taste by flavoring with vanilla or lemon, and with meat-juice and broths will often support a patient during the most trying period of the attack. There are typhoid subjects who cannot (on account of vomiting, etc.) take *per oram* sufficient nourishment to support life. In such cases we

may substitute or supplement the usual method of feeding by rectal alimentation, when from 3 to 4 ounces (96.0–128.0) of peptonized milk, ½ ounce (16.0) of meat-juice, and a little egg-white may be combined, and employed at intervals of four hours. In the case referred to previously, in which large hemorrhages occurred, vomiting was a distressing symptom, and the patient was nourished at times in part, and then altogether, by *nutrient enemata.* The latter mode of feeding in this disease may be the means of saving life. In early convalescence the patient may take well-cooked plain rice, entire eggs (diluted), or thin custards. Solid food should not be allowed till the temperature has been at the normal grade for one week at least. When defervescence is much prolonged and the patient becomes very weak or presents the fever of exhaustion, the administration of soft food (eggs, finely scraped meat) is often followed by notable improvement. The return to a full, solid diet should invariably be gradual.

Pure cold water is an excellent drink for fever patients, and it should be given to them regularly; it has a positive value as a diuretic in this and all other infectious diseases. Coffee, tea, and lemonade, sweetened with glycerin or saccharin, are admissible.

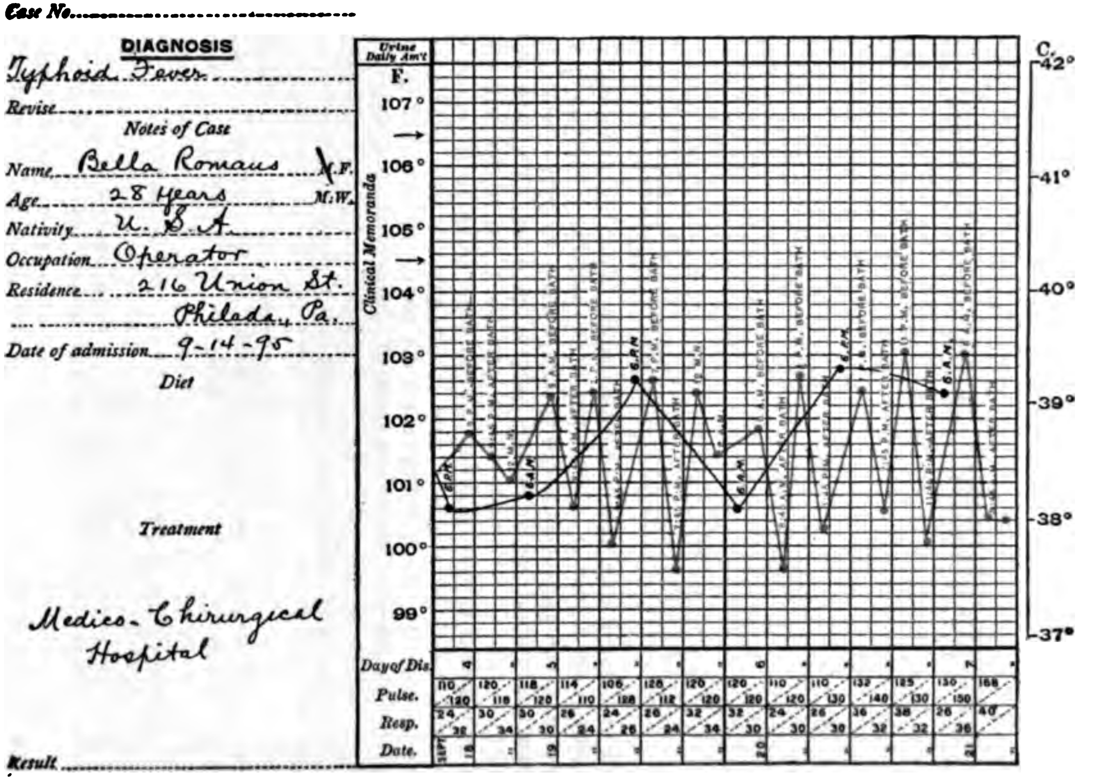
(3) **Stimulants** are useful in about 50 per cent. of the cases. When the heart becomes enfeebled, owing to exhaustion, alcohol should be used regardless of the temperature. In severe types whiskey is the best form; in milder ones some good wine, such as port, sherry, or madeira. It is well to begin with a moderate daily quantity, and then increase, if necessary, until the indication is fulfilled. If the patient so desires, we may use brandy instead of whiskey, and it is usually toward the close of the second or during the third week of the disease that the indications for the use of alcohol arise. It is not only the best spur for a flagging heart, but is of equal value in combating unfavorable nervous symptoms due to the typhoid septicemia; and the time for commencing its use may be indicated first by the latter symptoms (*e. g.* delirium, coma, tremor). The quantity to be administered must be regulated by its effects, which must be carefully studied in every instance, since it may act injuriously, and even aggravate the symptoms, though this is seldom the case. Threatened collapse may be met by full doses of alcohol (½ ounce (16.0) every hour), combined with strychnin (gr. $\frac{1}{16}$ (0.004) every three hours), exhibited subcutaneously till the depression has been counteracted. Effective doses of diffusible stimulants, as champagne, are useful during periods of sudden circulatory depression. Other cardiac stimulants are worthy of trial for their favorable supplementary action (digitalis, sulphuric ether).

(4) **Hydrotherapy.**—There is at the present day general agreement among medical authors that the best mode of treating typhoid fever is by means of the *cold bath,* which was originally introduced by Currie of London (more than a century ago), and reintroduced and successfully practiced by Brand of Stettin. There are obstacles in the way of carrying out hydrotherapy in private families, but since convenient and inexpensive portable tubs have been devised by Batt and Furbush of Philadelphia, Burr of Chicago, and others, most of the valid objections

to the method have been removed. At all events, the benefits offered to the patient by this method are so great and varied that it becomes the duty of every physician who treats typhoid fever to be prepared to employ it. The beneficial influences of the baths are as follows: (1) They absorb the body-heat directly, thus reducing the temperature and overcoming the ill effects of high fever, this action becoming more marked after a day or two of the treatment; (2) They improve the nervous symptoms, also render the mind clear; they diminish mental dulness, stupor, muscular tremors, and twitchings, and induce sleep; (3) They strengthen the heart, thus obviating the danger of sudden circulatory collapse and the consequences of increasing cardiac weakness (hypostatic congestion of the lungs, venous thrombosis, etc.); (4) They stimulate the respirations, whereby the inspirations are deepened and the tendency to pulmonary complications greatly lessened, especially severe bronchitis, lobular pneumonia, etc.; (5) The renal function is invigorated, and as a result the elimination of typhotoxins by the kidneys is increased (Roque and Weil); (6) On account of the cleanliness of the skin which they ensure, bed-sores rarely occur; (7) They may shorten the stay in the hospital or sick-room, but not the stay in bed, except, perhaps, in the lighter types.

Unquestionably, the good effects of the Brand method receive striking confirmation from statistical reports which have been prepared by Brand himself, Jürgensen, and others abroad, and by Baruch, Osler, Wilson, and others at home. According to the warmest European advocates of the method, the mortality is less than 0.5 per cent., and no deaths occur in cases that come under treatment before the fifth day. The results among American clinicians, however, have been less flattering, though strikingly uniform, and show an average mortality of 7.3 per cent. Of 102 cases of my own treated by cold and by graduated cold baths, 8 died—giving a death-rate of 7.8 per cent. During five years 408 cases have been treated by the bath-method in the Royal Victoria Hospital, Montreal, with a mortality of 4.4 per cent.

The *details*, which we will now consider, connected with the administration of this plan of treatment are of the utmost importance. The tub is to be brought to the bedside of the patient, and in hospital practice both bed and tub should be screened while the bath is in progress. After removing the night-dress and placing a large napkin around the loins, the patient should be lowered into the bath by a sheet held at each corner by an attendant (and, if seriously ill, with the least possible disturbance), and there carefully supported and held while in the bath. If sleeping, the patient must be awakened and the bath delayed for ten or fifteen minutes. Young subjects and adults in light cases of the disease may be handled properly by two persons, but I do not approve of allowing the patient to step from the bed into the bath, however light the case. While in the bath the skin-surface, particularly that of the back and limbs, is constantly rubbed by the attendants, in order to stimulate the peripheral circulation and as far as possible to avert chilliness and discomfort. The head of the patient rests upon a rubber air-cushion. At first he should be kept in the bath five to eight minutes; later, ten or fifteen minutes, according to the severity of the case. The head and face are bathed at once from a basin, and a



Case No.

DIAGNOSIS

Typhoid Fever

Revise

Notes of Case

Name Bella Romaus [✓ F.]

Age 28 years M. W.

Nativity U. S. A.

Occupation Operator

Residence 216 Union St.

Philada, Pa.

Date of admission 9-14-95

Diet

Treatment

Medico-Chirurgical Hospital

Result

Clinical Memoranda

Day of Dis.	4	"	5	"	"	"	"	6	"	"	"	"	7	"
Pulse.	110 / 120	120 / 118	118 / 120	114 / 110	106 / 128	128 / 112	120 / 120	120 / 120	110 / 120	110 / 130	132 / 140	125 / 130	130 / 150	168
Resp.	24 / 32	30 / 34	30 / 30	26 / 24	24 / 26	26 / 24	32 / 34	32 / 30	24 / 30	26 / 30	36 / 32	38 / 32	26 / 36	40
Date.	SEPT 18	"	19	"	"	"	"	20	"	"	"	"	21	"

FIG. 5.—Chart illustrating the influence of cold baths in the treatment of typhoid fever.

cold compress is applied to the forehead, and, if prominent nervous symptoms be present, often associated with high temperature, water at 70° F. (21.1° C.) or lower should be poured from an elevation of about six inches upon the head and nape of the neck several times during the bath. The ears must be stopped with cotton when douching is practiced. If while in the water the patient complains bitterly of the cold or is very restless, a stimulant may be administered—f℥j (32.0) of whiskey, diluted—and if this fails he must be lifted into bed and further stimulated. If he be very young, highly sensitive, or elderly, it is best to place him at the commencement in water of a temperature of 85° or 90° F. (29.4°–32.2° C.), and then gradually cool it down to 80° F. (26.6° C.). After he has become accustomed to the bath he may be immersed in water at the temperature of 80° F. (26.6° C.), to be reduced to 75° F. (23.8° C.) or even 70° F. (21.1° C.), below which it is unnecessary to go save in the rarest instances. This is the *gradually cooled bath* of Ziemssen. In the rigid Brand method, which is now generally adopted, and which I employ except in the above-mentioned instances, the patient is lifted at once into a bath at 70° F. (21.1° C.) and kept there for fifteen minutes. The patient is to be removed from the bath to the bed (previously protected by a blanket and mackintosh), wiped off gently, after which the sheet, blanket, etc. are withdrawn and he is covered with a blanket. If now reaction be retarded, some hot broth or about an ounce of whiskey should be administered and active friction applied to the back and extremities.

The effect of the bath is best shown by the rectal temperature, which is taken half an hour after the conclusion of the bath, and again a half hour later if the patient be not asleep. Usually the temperature will be found to be two or three degrees lower than before the plunge (see Fig. 5). In obstinate and severe cases the fall may be less than one degree, in which case it is advisable either to prolong the bath to twenty minutes or to reduce still further the temperature of the water. Protracted warm baths are highly recommended by Reisse and others when cold baths are badly borne or are unproductive of good results.

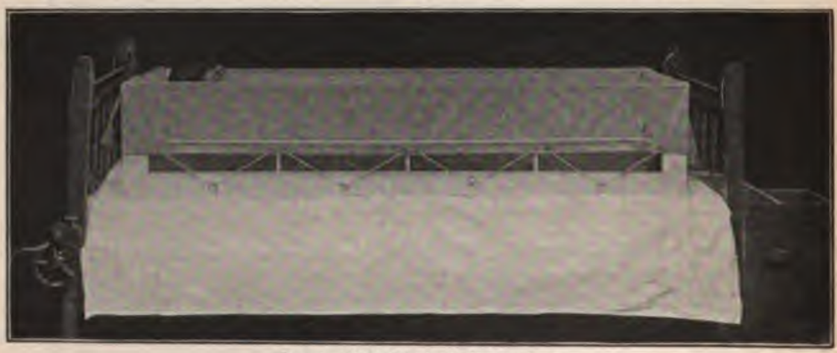


FIG. 6.—Portable bath-tub in use.

In light cases the cold bath should be repeated every six or eight hours; in severe ones, every three or four hours, but more frequently than once in three hours is not advisable, even in the worst cases.

Sufficient water to immerse the patient to the neck (about 30 gallons—114 liters) should be used. During the night the patient should be allowed to sleep for six or eight hours if he can do so.

As before stated, there are a number of convenient and satisfactory portable tubs in the market, but that devised by Dr. C. L. Furbush of Philadelphia possesses certain leading advantages (Figs. 6, 7). The frame is made of light wood, and when folded is 4 inches (10.156 cm.) in depth, 14 inches (35.546 cm.) in width, and 5 feet 10 inches (1.778 m.) in length, so that it can be placed in a closet or beneath a bed. Less than two minutes are required to prepare the bath, which the patient receives while lying in bed. When in use the ends are fastened by brass pins hung on small chains, and these hold the frame in a fixed position. The tub proper is made of double-faced sheeting, reinforced in the middle, so as to resist the greatest amount of pressure. The sides of the sheet have a casing through which is passed a wooden rod 4 feet 4 inches long (1.320 meters), and outside of this a margin of 1½ inches (3.808 cm.) is left for the brass eyelets, through which passes a rubber cord which is covered with woven cotton. This cord, which is attached to the sheet, is held to the frame by special brass fittings along the lower sides of the latter. By the use of the cord and wooden rods we have an even tension on both sides, combined with ample resistance to withstand the pressure of the water. An adjustable head-rest fits into the end of the frame. The wooden rod also enables the attendant to roll up the sheet quickly after the bath. Through the bottom of the sheet a 1-inch rubber tube is fitted with a stopper, and by means of this the tub can be emptied much sooner than by a siphon. The frame is covered with ivory-enamel paint, and can be cleansed easily, and the entire weight of the outfit is 25 pounds (11.33 kgms.).

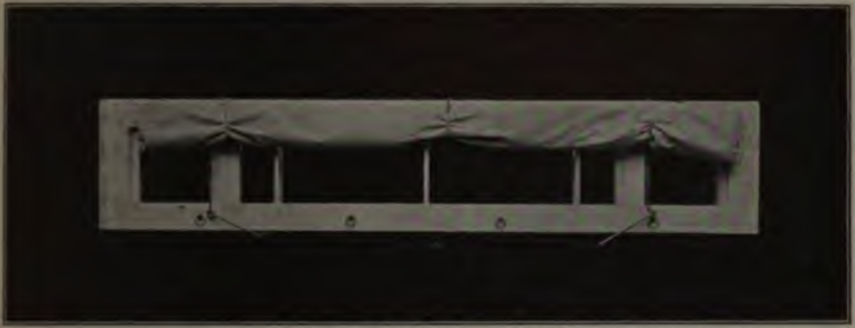


FIG. 7.—Portable bath-tub, folded.

Brand recommends that the baths be commenced when the temperature in the rectum reaches 102.2° F. (39° C.). The height of the temperature, *per se*, is not to be invariably regarded as an absolute indication for the employment of the cold bath, since the facts must be recollected that the essential effect is a stimulation of the nerve-centers which preside over the organic functions, respiration, circulation, etc. Moreover, cold baths exert a marked preventive effect in obviating serious symptoms and complications. I continue the baths until the evening temperature remains below 101° F. (38.3° C.).

The *contraindications* to the use of baths are—(1) *Intestinal hemorrhage*, which is in itself attended with danger and requires absolute quiet for a time (four days), when the baths may be resumed if there is no recurrence. (2) *Peritonitis*, the occurrence of which always excites suspicion of perforation. Here, again, rest and all that the term implies must be procured. (3) *Extreme Cardiac Weakness.*—The excitement in the necessary handling of the patient connected with the bath might prove fatal, as I have witnessed in one instance. This condition is sometimes met with in cases that come under observation at a late period, and have not been brought under proper treatment from the start, or in cases arising in aged and previously enfeebled subjects. (4) Cases that have progressed to an advanced stage (the third week of the disease) should not be immersed. Dangerous and even fatal collapse has been observed to follow cold baths under these circumstances.

Substitutes for the Cold Bath.—The prejudice which exists against the cold-bath treatment—at least in America—sometimes proves insurmountable. Again, there are many physicians who do not avail themselves of the means at command for carrying out hydrotherapy. In consequence of these facts substitutes for the cold and the gradually cooled baths are, unfortunately, quite commonly in vogue. Among them cold sponging of the body of the patient is often resorted to, though it secures for him few and trivial advantages as compared with those of the baths. If this method be employed, the water should be of the temperature of the air of the room or ward. The limbs should be sponged and dried in succession, and then the trunk. Whenever the temperature reaches 102.5° F. (39.1° C.) this measure is to be instituted, each sponging being continued until the desired effect has been produced (a reduction of the temperature of 1½° to 2° F. or 1° C.), unless the patient gives signs of uneasiness, when it must be cut short. It may be repeated as often as required. To the water used for the applications equal parts of vinegar or spirits should be added. The efficacy of the cool sponging is greatly enhanced by the simultaneous application of the ice-cap, either constantly or during alternate hours.

If this method fails, as it often does in severe types, the cold pack may form a satisfactory substitute; and I have found it of great use with children, in whom the reaction after a cold bath is often delayed or imperfect. The patient is placed upon a cot previously prepared by spreading over it a blanket, which is in turn covered with a sheet doubled and wrung out of water of the required temperature, 70° to 80° F. (21.1°–26.6° C.). The sheet and blanket are now wrapped about the patient evenly, and he is left in the pack for a period varying from a half to one hour. Free diaphoresis generally ensues, and this aids in maintaining the fall of temperature. The effect, in most instances, is to reduce the body-heat two degrees or more, and the treatment may be repeated at intervals of three or four hours if needful. The wet sheet alone may surround the patient, and be sprinkled at short intervals with a watering-pot containing water of a temperature of 70° F. (21.1° C.). In desperate cases in which cold baths are for adequate reasons out of the question ice-water enemata may be tried. If carefully administered, they accomplish a reduction of the temperature by two or more degrees. Leiter's coils may be applied to the head, chest, or abdomen.

Guaiacol has been used for its potent antipyretic effect by H. G. McCormick and others, from 10 to 30 minims (0.666–2.0) being applied to the skin surface. I have seen its use followed by rigors, hyperpyrexia, etc., but McCormick has adopted the rule of using sufficient only to lower the temperature to 100° F. (37.7° C.), and has thus avoided all ill effects.

(5) **Internal Antipyretics.**—Internal antipyretics are also effectual agents in combating immoderate temperatures, and certain of them, it must be confessed, have a powerful influence. Moreover, they soothe and moderate the nervous symptoms and act more or less potently as antiseptics. But even the most reliable of this group of medicaments (phenacetin, acetanilid, and antipyrin) are open to the serious objection that they depress cardiac power.

When the Brand method cannot be employed, or, as rarely happens, it is ineffective and there is present a high fever with decided nervous symptoms, internal antipyretics are allowable if properly administered. The safest among them is phenacetin, of which 5 grains (0.324) may be given at a dose (preferably about 3 P. M.), and repeated after four hours should the first dose fail of the desired effect. Acetanilid is more effective than phenacetin, but is not quite so free from injurious action as the latter. It may be prescribed in doses of 2 to 4 grains (0.129–0.259), to be followed by a second dose of equal size in four hours if necessary. The heart is always to be guarded by the use of stimulants.

(6) **Intestinal Antiseptics.**—Unquestionably these neither destroy the bacilli nor counteract the ill effects of their toxins, since both become active after they pass beyond the intestinal mucosa; but they are indicated in an affection in which extensive intestinal ulceration and moderate tympanites are usual manifestations. Some of the toxic substances occupying the intestines in this disease result from the acquired virulence of usually harmless organisms, and the amount of decomposable material is increased owing to defective hepatic secretions. The bowel antiseptic which I have employed quite extensively, and with uniformly good results, is salol, this drug being broken in the intestinal canal into carbolic and salicylic acids, and being capable of controlling meteorism as nothing else has done in my hands. The dose is 2 to 3 grains (0.1296–0.1944) every three hours, preferably administered in capsule. With it I usually combine quinin in doses of 1 to 2 grains (0.0648–0.1296) each. Henry speaks strongly in favor of thymol. Wilcox[1] urges that chlorin is capable of disinfecting the intestinal tract. Systematic lavage of the intestinal tract is advisable in excessive tympanites. In cases in which pronounced meteorism occurs the use of hydrochloric acid in small doses after each feeding is serviceable, since the secretion of this agent, which normally inhibits putrefactive changes, is lessened.[2]

Lactophenin (gr. vij–xv—0.4536–0.9720 per dose), in starch capsules, up to 1½ drams (6.0) daily, according to the indications, is highly recommended (Jaksch). Carbolic acid, iodin, and other antiseptic agents have their advocates.

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[1] *Medical News*, February 11, 1899.

[2] See *Therapeutic Gazette*, April 15, 1900, by the writer.

Turpentine fulfils in some cases a leading indication. When the tongue is dry and brown, the abdomen distended, the general prostration marked, and often muttering delirium present—symptoms of the typhoid state—the use of this agent, together with alcoholics, constitutes the best mode of treatment. Turpentine is best given in a capsule in the form of white turpentine—dose, 3 to 5 grains (0.1944–0.3240) every three hours.

(7) **Curative Inoculations with Cultures of Serum.**—The brilliant results obtained from the use of antitoxic serum in diphtheria and certain other affections have led to attempts at curative inoculations in typhoid fever. Though their specific virtue is yet to be demonstrated, it is deemed proper to state the results which have been obtained as concisely as possible. E. Fränkel and Manchot have treated 57 cases of typhoid fever with a sterilized liquid derived from a culture of the bacillus of Eberth in thymus bouillon and heated to 140° F. (60° C). Of this, ½ c.cm. was injected deeply into the gluteal region, with favorable results.

Hughes and Carter treated a number of cases with blood-serum derived from convalescent cases, but apart from a decided lowering of temperature the general course of the disease was not perceptibly modified. Wasserman and others have shown that the organs of animals, some inoculated experimentally, some not, contain protective substances. Pursuing this line of investigation, Jez[1] found that he was able to obtain from the organs of animals inoculated with typhoid bacilli substances capable of exercising protective and curative influences upon typhoid infection. The mode of producing the extract was as follows: Guinea-pigs were given, at short intervals, several intraperitoneal injections of bouillon-cultures of typhoid bacilli of progressively increasing virulence. When tolerance was established the animal was killed and its thymus gland, spleen, bone-marrow, brain, and spinal cord removed, finely divided, and rubbed up in a mortar with a solution consisting of sodium chlorid, alcohol, glycerin, and a small amount of carbolic acid. Subsequently a small amount of pepsin also was added advantageously. After standing on ice for twenty-four hours the mixture was carefully filtered, a clear, reddish fluid resulting, which did not cause agglutination and sedimentation of typhoid bacilli nor inhibit their growth, though exhibiting the faculty in marked degree of neutralizing typhoid toxin. This anti-typhoid extract was employed in the treatment of 18 cases of typhoid fever, being administered by the mouth in doses of from a teaspoonful to a tablespoonful every two hours, subcutaneous injections proving less serviceable. In the cases thus treated the characteristic temperature-curve was lost, the pyrexia becoming remittent and soon disappearing; the pulse declined in frequency and increased in strength, diarrhea ceased, the tongue cleared, the general condition improved, and convalescence speedily set in. Unpleasant secondary effects were not observed. The usual hygienic and dietetic and other precautionary general measures were not neglected.

(8) **Treatment of Individual Symptoms and Complications.**—*Headache.*—Early in typhoid the headache demands relief. Absolute rest and cold to the head frequently suffice. Depressant analgesics are to be avoided so far as may be, though it sometimes becomes necessary to

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[1] *Wiener med. Wochen.*, Feb. 18, 1899, p. 345.

resort to them. At such times those least objectionable are to be selected. I have found that a mixture containing sodium bromid (gr. x to xv —0.6480 to 0.9720) and the deodorized tincture of opium (♏iij to v—0.1998 to 0.3330) in each dose, given at intervals of three or four hours, exercises a striking palliative influence. In occasional instances the above mixture fails, and then phenacetin (gr. ij to iij—0.1296 to 0.1944) may be substituted for the opium in the same combination or separately in capsule.

Insomnia.—The cold baths or other measures calculated to relieve the headache often procure for the patient refreshing sleep. It is important not to allow him to go too long without sleep, since this tends to the development of a pronounced "typhoid state" and its concomitants. When the agents recommended for the headache fail, I employ morphin hypodermically in small doses (gr. $\frac{1}{16}$ to $\frac{1}{8}$—0.004 to 0.008) during the evening hours, with excellent results, and have yet to witness the unpleasant after-effects or the unfavorable influence upon the secretions that have been described by some authors. Codein, sulfonal, and, more recently trional and chloralamid, have proved useful.

Chloral is more certain in its action than the above agents, but I have abandoned its use for the reason that it apparently produced circulatory collapse in two instances.

Delirium.—Since the introduction of the Brand method delirium rarely calls for special medication. I have observed, in common with others, particularly during the advanced stages, that in cases in which the circulation was feeble and in which typhomania was a prominent feature, the administration of stimulants with a free hand completely dispelled the nervous phenomena. If alcohol fails, ether (♏x—0.666—at a dose) may be given hypodermically, and repeated in one or two hours if necessary. To combine with the arterial some nervous stimulant (musk, valerian) will be found serviceable, particularly in cases in which the delirium assumes an hysteric type. Of special value in meeting this symptom are the bromids, hyoscyamus, the persistent use of ice to the head, and the other agents suggested for the headache and insomnia.

Vomiting is rarely troublesome. Its chief cause is the irritation of the gastric mucosa, which may be caused by improper diet or medication. The best measure for the relief of this symptom, after the removal of the cause, is the use of ice, taken in small pieces and swallowed. If vomiting occur during the period of development, minute doses of calomel, combined with sodium bicarbonate, may be prescribed with good effect. If it occur during the fastigium, the amount of milk taken should be reduced by one half, peptonized, and then diluted, preferably with lime-water. If the patient experience a strong aversion to milk, it must be suspended temporarily and liquid beef-peptonoids or broths substituted. Dry champagne may be administered simultaneously. Excessive irritability of the stomach calls for perfect rest of the organ for a period of not less than twenty-four hours, the patient being meanwhile supported by rectal alimentation and subcutaneous medication.

Diarrhea more than any other single symptom claims special attention. Two to four movements daily do not constitute diarrhea and do not demand treatment, but if this number of stools be exceeded, the condition should receive consideration. It may be caused by overfeeding or by improper food—as shown by the stools, as a rule—in which case regulation of the diet is curative. It is often due to ulcerated and catarrhal lesions of the intestines, and particularly the large bowel, and in such cases requires medical interference. Unquestionably, the use of proper intestinal antiseptics and such as possess the property of insolubility to a high degree is most valuable. Astringents may be combined with the latter or given separately. The subjoined formulæ have yielded better results in my own hands than numerous others which have been tried:

℞. Bismuth. salicylat., ʒij (8.0);
Betanaphtol, ʒj (4.0).
M. et ft. capsulæ No. xxiv.
Sig. One to be taken every three hours.

Or,

℞. Salol., ʒj (4.0);
Bismuth. subgallat., ʒij (8.0).
M. et ft. capsulæ No. xxiv.
Sig. One every two or three hours.

Or,

℞. Plumbi acetat., gr. xxiv (1.555);
Ext. opii, gr. iss–ij (0.097–0.1296).
M. et ft. pil. No. xij.
Sig. One every three or four hours, as required.

The last formula may be administered in the form of a suppository, both ingredients being doubled in quantity.

Late in typhoid fever, when the ulcers are fully developed, opium is the remedy *par excellence*, since it tends to arrest the peristaltic action which keeps up the diarrhea and favors the spread of the inflammation to the peritoneum. I have recently observed brilliant results from the use of rectal injections of an astringent solution (tannic acid 1–2 per cent.), alternated with an antiseptic solution (salicylic acid 1–2 per cent.), each given once daily at intervals of twelve hours.

Constipation, which is often present, and particularly until the middle of the second week, is to be relieved by simple enemata of soapsuds every second day. Calomel may be used in the early stage of dynamic cases. Its employment in this manner may be followed by symptoms of a milder type than are ordinarily encountered. If constipation exists during the third week, accompanied by an oscillating temperature-curve, as rarely occurs, saline laxatives in small but repeated doses may cut short the attack.

Tympanites.—This is sometimes a most distressing symptom, and is often associated with marked diarrhea. The claim has been made that if turpentine be administered in suitable doses throughout, both tympanites and diarrhea are controlled. Turpentine is a good remedy, but only when certain indications exist (*vide supra*), and it is without the power to influence the general course of the affection. As a remedy for tympanites it is excellent and richly deserves a trial. When employed for this symptom alone I prefer to apply it in the form of stupes over the abdomen, although when, as is frequently the case, the gases occupy chiefly the large bowel, turpentine enemata should be given, and, these failing, a long rectal tube should be passed.

The meteorism is often increased by the milk taken, and a change of food to meat-juices and albumin-water may be tried.

Hemorrhages from the bowels demand complete rest. The bowel-movements, if the hemorrhage has been copious, must be allowed to pass into the draw-sheet. The ice-bag (suspended if possible) should be applied to the right iliac region, and ice freely given by the mouth. Opium, to control peristalsis, must be given, and, by preference, hypodermically. It may be combined with full doses of the acetate of lead. Cases in which slight oozing appears from time to time require turpentine. Ergotin may be used (hypodermically, to be repeated every hour) in severe bleedings. The amount of food should be greatly restricted for about twelve hours. For severe hemorrhages, *saline infusion*, either by the method of intravenous injection or by hypodermoclysis or enteroclysis, is to be strongly advised. The saline solution not only raises the blood-pressure in the vessels, but it also has a hemostatic action. The proper strength is 7 : 1000, and from 10 c.c. (3 fluidrams) to one-half liter may be employed if the collapse is marked, and repeated several times in the course of a day. Rectal injections may be somewhat larger.

Peritonitis.—When this is due to perforation the patient usually passes quickly beyond hope unless saved by timely surgical intervention. Operation offers some hope of cure, and with the progress of convalescence the chances of recovery from this accident improve. Deaver regards the acute development of pain and generalized abdominal rigidity and tenderness as an urgent indication for immediate celiotomy. Keen's statistics show that between twelve and twenty-four hours after perforation is the most favorable time for operation, this period giving 30 per cent. of recoveries. Rarely, appendicitis supervenes in typhoid fever, and presents nearly the same symptoms as when occurring independently. It usually demands prompt removal of the appendix. Peritonitis due to direct extension of the infectious inflammation of the bowel without perforation often admits of successful treatment. Saline purgatives, at the same time controlling pain by means of small doses of morphin, are the measures to be used.

Pneumonia.—Broncho-pneumonia is to be treated in the manner indicated in the section on this affection.

Lobar Pneumonia.—The treatment of that form of pneumonia which occurs in the advanced stage of typhoid will be considered hereafter (*vide* Secondary Pneumonia). That variety of pneumonia which rarely inaugurates typhoid requires the same treatment, until the true typhoid symptoms arise, as primary adynamic pneumonia (*vide* p. 156).

The *hypostatic congestion* of the bases of the lungs is to be met by heart-stimulants and by changing the position of the patient.

Bronchitis.—No special measures are necessary when the bronchitis is confined to the larger tubes, as in typical cases, while, if severe and diffuse, its management is identical with that of broncho-pneumonia.

Laryngitis.—For this condition, counter-irritation should be tried; and if this fails, a small blister may be applied below the angle of the jaw on either side. For edema of the larynx scarification and the inhalation of simple or medicated steam are useful measures. Then, should suffocation become imminent, tracheotomy should be performed without delay. Operation "gives a mortality of only 55.5 per cent." (Keen).

Bed-sores.—The preventive measures have already been considered, but the smallest bed-sore demands active treatment. It is to be kept clean by means of a weak solution of some antiseptic, and may then be dusted with a powder composed of equal parts of boric acid, calomel, and bismuth; if sluggish, with a powder made up of aristol and iodoform. I have found unguentum balsami peruviani (1 : 30) to be a valuable remedy in bed-sores. Should the edges of the ulcer become undermined, a drainage-tube is sometimes necessary.

Thrombosis of the femoral vein is best treated by elevating the part and keeping it at perfect rest. The following ointment may also be applied along the course of the vessel:

℞. Ung. ichthyol.,
Lanolin, *āā.* ʒij (8.0);
Ung. belladonnæ, q. s. ad ℥j (32.0).
Sig. Apply three times daily.

After the swelling has subsided an elastic stocking should be worn for a couple of months.

(9) **Management of Convalescence.**—Some of the points connected with this subject have already been discussed (*diet*, *time for getting up*, etc.). I may add that should a recrudescence occur the patient should be kept at rest in the recumbent posture and a return made to the liquid forms of food. Often a moderate laxative serves a good purpose, particularly if an indiscretion in diet have been committed. The ulcers may not be healed, though the temperature may have been normal for a week or ten days; hence the patient should not be allowed to stir about for a period of two weeks after the temperature has returned to the normal. At first his movements should be slow; he may soon, however, be allowed to exercise gently in the open air during seasons of favorable weather. Mental excitement is to be avoided, since it may produce a recrudescence of fever. Occasionally, during convalescence the diarrhea persists, being due to colonic ulceration, and is best treated by restricting the diet to milk and other light forms of albuminous food. The patient must be confined to bed. Medicinal treatment by the oxid of zinc internally and the use of astringent and antiseptic rectal injections, as before indicated, usually proves successful. Constipation may be a troublesome symptom in convalescence, and is best relieved by simple enemata. Most patients require tonics. We should begin with a vegetable salt of iron in combination with a simple bitter (such as the infusion of gentian), and later an inorganic salt of iron, with quinin and strychnin, may be resorted to. If there be a predisposition to tuberculosis, cod-liver oil and creasote should be given for a period of two or three months. Relapses are to be treated as primary attacks, and recurrences in the same manner.

MOUNTAIN FEVER.

(*Mountain Sickness.*)

The term "mountain fever" should be regarded as applicable only to that condition which develops shortly after ascent to a very high altitude. There is no definite *pathology* nor *etiology*, but the symptoms are attributable to the effects of a rarefied air upon the organic functions.

The **symptoms** are a much quickened pulse, urgent dyspnea, headache, vertigo, and at times nausea and vomiting. There is a subfebrile movement, the temperature touching 100° or even 101° F. (38.3° C.). Thirst is present and the appetite is lost. Malaise and a sense of exhaustion on attempting exertion are experienced. Hemoptysis has been noted, but rarely. The effect upon the human economy of high altitude varies with the extent of the differences in individual reserve nerve-force. Rest and acclimatization will almost invariably restore healthy function. Mountain anemia has been described; it is caused by the *anchylostoma.*

The "mountain fever" of the older writers is almost universally conceded at the present day to be typhoid fever modified by the effects of extreme altitude. The lesions of typhoid fever were present in two instances that were necropsied. Woodruff has reported 35 cases at Fort Custer in which the grouping of symptoms, including the Widal reaction, assured a diagnosis of typhoid fever. Gwyn recently recorded a case in which the clinical features of typhoid were present (except the Widal reaction), but cultures from the blood showed a "paracolon" variety of bacillus. Curtin has reported 4 cases all evincing the signs and symptoms of lobar pneumonia. These well-known affections, variously modified by high altitudes, should not be embraced in the nomenclature of "mountain fever" or "mountain sickness."

TYPHUS FEVER.

(*Ship-fever, Camp-fever, Jail-fever, etc.*)

Definition.—An acute contagious disease of unknown specific etiology. It is characterized frequently by an abrupt invasion, and is marked by rigor, high fever, early nervous symptoms of great prominence, a maculo-petechial eruption appearing between the third and fifth days, and a termination by crisis.

Historic Note.—This affection has been known from time immemorial. In 1759 the name *typhus*, which is at present universally employed, was given to it by Sauvages. In presanitary times it prevailed extensively in epidemic and endemic forms, particularly in Ireland and Russia, and also, though less frequently, in the seaport towns of our own country. It constituted one of the chief plagues of the olden times, and its devastations among the armies were more destructive of human life than even shot and shell.

In 1812 typhus fever first appeared in America in the New England States. Its ravages did not cease until every Eastern State had been visited by the plague, when it totally disappeared. In 1836 it reappeared in Philadelphia in virulent form and with deadly effect. It was at this period that Gerhard began his careful studies, which resulted in the separation of typhus from typhoid. During the last half century comparatively few instances of typhus have been met with in this country, though it still appears constantly in certain quarters, abroad (Great Britain, the eastern portion of Germany, Poland, Russia, and some parts of Southern Europe). All isolated cases and small groups of cases that have been observed in very recent times here have been properly attributed to importations from other countries, and chiefly from Ireland. Since the epidemic in 1836 the disease has not gained a foothold on our shores, although in the early part of 1893 it appeared in New York City, and 150 cases resulted.

Pathology.—After death the eruption continues to be visible, and often large ecchymoses are observable on the dependent parts of the body. Rigor mortis is often delayed.

Certain organs may present pathologic appearances, but they are not constant and are the result of the secondary infection which the typhus invites. The serous membranes—the pericardium in particular, and at times the gastro-intestinal mucosa—are the seat of ecchymoses. There is hyperplasia of the lymph-follicles, but no subsequent ulceration. Hemorrhagic extravasation may also occur into the muscles, the latter being dark and often showing hyaline and granular changes; the *heart-muscle* is especially apt to undergo a granular degeneration. The *spleen* is considerably enlarged, soft (even diffluent at times), and of a dark (frequently bluish) red color. The *liver* is somewhat swollen and may be softened, while the *kidneys* not rarely manifest the changes belonging to nephritis. In other instances they are merely congested. In the *lungs* are found a variety of lesions peculiar to different complicating conditions (bronchitis, lobular pneumonia, lobar pneumonia, pulmonary congestion with or without edema), and occasionally *pleurisy* (sero-fibrinous or purulent) may be present. *Nervous lesions* are conspicuous by their absence. An effusion, either serous or sero-hemorrhagic, into the subarachnoid space and the ventricles may be noted, and quite commonly there is *cerebral congestion.* In rare instances there may be a *meningitis.* The *blood-changes* are marked, the color being dark, the fluidity much increased, while the coagulability is greatly diminished; and the intima of the aorta is frequently blood-stained. The various viscera, however, present no distinctive lesions.

Etiology.—The **direct cause** or special micro-organism connected with the typhus contagion has not, as yet, been isolated, notwithstanding the fact that the morphologic and biologic studies of the blood obtained by Brannan and Cheesman from the finger-tips of six patients during the mild epidemic of typhus in 1893 showed the presence of a bacillus that proved pathogenic for rabbits, guinea-pigs, and white mice.[1] Lewaschew[2] has also detected in the blood of typhus patients a distinctive micro-organism. Further observations, however, with a view to showing the constant presence of these micro-organisms in typhus fever, are necessary to demonstrate that they are the specific cause of the disease.

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[1] *Annual of the Universal Medical Sciences*, 1893, p. 60, section H.

[2] *Ibid.*, p. 61, section H.

It is a known fact, nevertheless, that when typhus arises in a locality in which it was previously unknown it is dependent upon a transference of the typhus virus from without, and does not arise spontaneously; this cannot be too strongly emphasized. The different *modes of conveyance of this poison* are imperfectly known, although there are good reasons for believing that it may leave the body in the expired air, in the epithelial scales thrown off, and in other excretory or secretory products of the body. The poison is apt to be transmitted by *contagion* from the patient to others who approach him; and there is convincing proof that it may be transferred by means of *fomites* (wearing apparel, articles of furniture, etc.). What its precise *gateway* into the body is we do not definitely know, except that it is more likely to enter through the respiratory tract (by inhalation) than through the alimentary canal.

Predisposing Causes.—The influence of *insanitary surroundings* upon the spread of this affection is positive and vital. Among special conditions may be mentioned filth, poverty, famine, and overcrowding, and here it may be inferred that typhus is a disease of the lower classes. It prevails in jails and camps. Broadly speaking, any condition of the system in which the natural vitality and resistance to bacterial invasion are lowered increases susceptibility to the disease, and among additional influences which possess considerable etiologic influence are overwork, intemperance, depressing emotions, etc.

Age has no direct influence. Obviously, however, the young and middle-aged furnish a preponderant proportion of cases, owing to the fact that they are more liable to exposure to the virus than during other periods of life. *Sex* has no positive influence, and the *season* plays only a minor part. Epidemics may, however, occur rather more often in winter than in the other seasons, since the homes of the underfed pauper population are not so well ventilated, and hence are less cleanly in winter than during the rest of the year. It almost invariably prevails in an epidemic form.

Clinical History.—**Incubation.**—This lasts from nine to twelve days. There may be prodromal symptoms during the concluding days (one, two, or more of this period), such as anorexia, general malaise, etc., but in most instances *invasion* is sudden.

Pre-eruptive Stage.—The *early* symptoms are either a series of chills or one severe rigor, accompanied by vertigo, tinnitus, headache, muscular pains, profound prostration, and fever. The *temperature* quickly ascends to a high level, reaching 104° or 105° F. (40° or 40.5° C.) as early as the second or third day. The fever is continuous in type, and in severe cases a serious systemic condition may often be developed. The *pulse* is accelerated proportionately to the temperature and is of good volume. Bronchitis may be present, the appetite is lost, and the thirst is excessive, while a thick, yellowish-white coating covers the tongue. *Vomiting* occurs, and may be a prominent symptom. The *urine* is often scanty, its specific gravity is increased, and it may contain a trace of albumin. The cheeks are flushed and the eyes are injected.

Nervous symptoms appear early—in the worst cases at the very onset —and are quite pronounced. At first there may be either mild or active delirium, but soon there is stupor or even actual coma, and the face takes on a dull, stupid look. With few exceptions the spleen on palpation is found to be enlarged.

Eruptive Stage.—Between the third and fifth days of the invasion the *characteristic eruption* appears *without an accompanying decline in the temperature.* The *rash* comes out first upon the trunk (chest and abdomen), extending thence over the rest of the skin-surface of the body, but, strangely enough, often sparing the face. The crimson-red maculæ are changed in two or three days to a darker hue, becoming hemorrhagic (petechiæ), and when coalescence occurs we have the spotted effect that has caused the name of *spotted fever* to be given to it. This name is also given to cerebro-spinal meningitis, in which the eruption, though it resembles that in typhus fever, does not appear at any given time and is extremely inconstant. Not all of the maculæ are converted, but some may remain as rose-spots, and these disappear when pressed upon, while the petechiæ do not. It is chiefly in the milder grades of typhus that the rose-spots fail to become petechial (*vide infra*). The skin-surface between the spots is sometimes diffusely hyperemic, and the eruption is usually rather abundant, though in well-authenticated cases it has been scanty or even wholly missing. The skin may also present darker and lighter blotches, producing a mottled appearance. Unlike many other eruptive diseases in the stage of eruption, the symptoms of typhus fever assume an aggravated type in typical and severe cases. The temperature continues high, often reaching 106° F. (41.1° C.) or even higher, with slight nocturnal remissions. The pulse becomes quite rapid (120–140 or more), feeble, and possibly irregular (often dicrotic), and the respirations increase markedly in frequency. At this time severe bronchitis, leading to *broncho-pneumonia*, is apt to occur as a complication. The *tongue* is brown, fissured, tremulous, and occasionally black and rolled up, without power to protrude from the mouth. Sordes form on the teeth and lips. The *urine* is scanty, high-colored, and often albuminous.

The **nervous disturbance** is intense, and may take the form of typhomania, leading to complete coma or maniacal delirium. The patient often lies with eyes open, staring into space, yet unconscious and in the condition known as *coma-vigil.* The motor nerves show derangement (tremors, subsultus tendinum, etc.), and carphologia (picking at the bedclothes) is a common symptom. The decubitus is dorsal, as a rule; the flushed cheeks gradually become dusky, the face expressionless, and the pupils often contracted. The prostration reaches an extreme degree, and absolute exhaustion often terminates life.

As a rule, in favorable cases the end of the febrile period comes by *crisis* between the fourteenth and seventeenth days of the disease, and the temperature drops in the course of twenty-four or thirty-six hours to normal. Immediately preceding the crisis there is generally a great and sudden rise of the temperature (*perturbatio critica*), and the decline may be interrupted by slight irregularities or fresh exacerbations. The occurrence of the crisis is marked by rapid improvement in the symptoms in general. The stupor suddenly gives place to a clear mind (sometimes following a profound sleep), the eruption fades quickly, the facial phenomena disappear in inverse order of their appearance, and the general strength is rapidly recovered.

Leading Symptoms and Complications.—Course of the Fever.—Although the temperature, as stated above, rises rapidly on the first day of the illness, it should be added that the highest grade is usually reached as late as the fifth or sixth day. Maximum temperatures of 105°, 106°, or even 107° F. (40.5°–41.6° C.) are common. Hyperpyrexia usually heralds a fatal termination, the temperature mounting to 108°, 109° F. (42.7° C.), or higher, though in light cases the acme may not exceed 103° F. (39.4° C.). During the height of the affection the temperature pursues the continued type (slight morning remissions), with moderate oscillations, till the occurrence of the crisis which has been described. The fall of temperature may occasionally be more gradual than before indicated. About the beginning of the second week the typhus patient emits a disagreeable odor that is regarded as characteristic by some writers.

The **lungs** frequently present complications (*vide* Pathology), among which the most common are *bronchitis*, *broncho-pneumonia*, and *hypostatic congestion*. Broncho-pneumonia is especially dangerous, its development often preceding a fatal termination, and it may lead to pulmonary gangrene. If the gangrenous, consolidated areas connect with the pleura, empyema commonly results. Sero-fibrinous pleurisy also may occur as a secondary event, as may lobar pneumonia, and to recognize the latter the local physical signs must be fully appreciated, since the rational symptoms are feebly expressed.

The **heart** in typhus continues to grow progressively weaker until, in many cases, a fatal issue is reached. This is manifested by the change in the character of the first sound, which becomes more and more indistinct as the case progresses, A systolic *murmur* (probably of hemic origin) may be audible at the apex.

The **nervous phenomena** have been sufficiently detailed. *Meningitis* has been met with, but is very rare as a complication. Reference has been made to the occurrence of the ordinary febrile albuminuria in this disease, and it remains to be pointed out that *hemorrhagic nephritis* very rarely intervenes. During the febrile period the uric acid and urea increase in quantity, while the chlorids decrease.

The **digestive tract** rarely presents distressing symptoms and complications. *Hematemesis* is most common, and *cancrum oris* has been noted occasionally. Cases in which the mouth does not receive proper care are apt to develop *parotitis*, which often passes on to suppuration, and *septic processes*, causing abscesses in different parts of the body (joints, subcutaneous tissue), may arise as complicating events.

Among the **sequelæ**, *neuritis*, followed by *paralyses*, deserves first place, and *gangrene of the remote extremities* (toes, fingers) has also been observed.

The **general course and duration** of typhus are variable. There is a *mild type* whose course is run in from seven to ten days, and in such the crisis occurs soon after the appearance of the eruption, which may not proceed to the petechial stage. In this type the development of serious symptoms or grave complications is the exception. A *malignant type*, however, also occurs (*typhus siderans*), and this often proves fatal before the time for the appearance of the rash.

Some epidemics are characterized by the relative frequency of light forms, and others by the severer types of the disease.

Diagnosis.—On the known presence of an epidemic with special causative factors (unhygienic surroundings, exposure to the poison, etc.), and with the course and characteristic symptoms, the diagnosis of typhus fever can be made. Of special value is the eruption—its time of appearance (third to fifth day), mode of distribution, petechial character, and peculiar behavior under pressure. The recognition of lighter types, on the one hand, and malignant, on the other, is not possible from the symptoms alone, but it is so from the light afforded by a definite knowledge of the existence of an epidemic in the vicinity.

Differential Diagnosis.—*Typhoid fever* is distinguished from this affection by (*a*) its gradual onset, unaccompanied by severe rigor; (*b*) the relatively diminished violence and the later development of the nervous symptoms; (*c*) the less intense lumbo-muscular pains; (*d*) the less abundant eruption, which is non-petechial and appears on the seventh or eighth day; and (*e*) the gradual convalescence.

Cerebro-spinal meningitis may be distinguished by a more intense headache, by retraction of the head, hyperesthesia, intolerance of sounds, photophobia, palsies of the eye-muscles (strabismus), a tendency to convulsions, and by both the absence of the typhus eruption and the expressionless countenance. Quincke's lumbar puncture may be practised, and will settle the doubt in obscure cases.

Uremia is excluded by the absence of the previous history which it always gives (headache, vomiting, and diarrhea extending over a variable period of time), by the presence in typhus of high temperature and a petechial eruption, and by the absence of edema of the extremities and face. Characteristic urinary phenomena are associated in uremia, although rarely *acute hemorrhagic nephritis* occurs in typhus fever.

The eruption of *malignant measles* may bear a close resemblance to that of typhus; the rash in typhus, however, appears first upon the trunk, that of measles upon the face. Koplik's spots do not appear in typhus. Points connected with the epidemicity of measles, as the occurrence of mild and typical cases, must be taken into account.

Relapses are among the rarest of clinical events, and one attack, as a rule, bestows immunity for life.

Prognosis.—To arrive at a correct prognosis it is necessary to consider (1) the degree of severity of the particular type from which the patient is suffering, (2) the number and character of the complicating conditions present, or likely to occur if the case be of a severe grade, and (3) any peculiar circumstances connected with the individual, among which his food-supply and his sanitary surroundings are deserving of chief mention. In general terms, typhus fever is a grave disease, but its frequency of occurrence, and also its virulence, have been markedly reduced in consequence of better sanitation, so that the mortality-rate at the present day is between 10 and 20 per cent.

Treatment.—This embraces, in the main, the same principles that were evolved in the treatment of typhoid fever.

Prophylaxis demands *thorough disinfection* and *absolute isolation.* A special hospital for contagious diseases is always to be preferred to the best accommodations obtainable in private families. When, however, patients cannot be transferred to special hospital wards and must be treated in private houses, the sick-room must be kept clean, well-ventilated, and at a temperature ranging from 60° to 65° F. (15.5° to 18.3° C.). No one other than the doctor and nurse should be allowed to occupy or even enter the room. The thorough disinfection already described under Typhoid Fever must be enforced with equal care, and the importance of supplying fresh air to typhus patients has been abundantly shown by the great reduction in the mortality-rate among those treated in tents as compared with that in the hospital wards.

The **general management**, including the use of stimulants, in this disease does not differ from that advised in typhoid fever, except that a more prompt return to solid food can be made during convalescence than in typhoid. Fresh water should be given freely, and, in view of the blunted sensibilities of the patient, should be offered at regular intervals. *Hydrotherapy* constitutes the best means at our command for controlling (by virtue of its stimulating effect upon the cardiac and respiratory centers) the temperature and the nervous symptoms, while at the same time it obviates dangerous complications. In addition, the use of antiseptic agents and tonic measures is to be recommended. The fact that typhus is a self-limiting affection, and therefore curable if the crisis can be survived, gives those measures that are intended to combat exhaustion first rank in the treatment of this affection.

RELAPSING FEVER.

(*Febris Recurrens; Relapsing Typhus.*)

Definition.—An acute infectious disease caused by the spirillum of Obermeier, and characterized by febrile periods which usually last six days, and are separated by afebrile periods of the same duration.

Historic Note.—The first accurate account of this affection was published in 1739, though it is known to have prevailed in Europe and Ireland prior to that period. During the next century numerous epidemic outbreaks, more or less extensive, occurred, and in 1844 the disease made its first appearance in America at the Philadelphia Hospital, being brought by immigrants from Ireland. Subsequently small groups of cases occurred, and were reported by Flint and others, and in 1869 it prevailed considerably in Philadelphia (where it was studied especially by E. Rhoads and William Pepper) and in other large cities of the country. This was the last epidemic appearance of the disease in the United States, though in the years 1885 and 1886 Russia was visited by an outbreak of considerable magnitude.

Pathology.—The solid organs of the body present no characteristic anatomic changes, though when death occurs during the febrile period the various viscera (heart, liver, kidneys) are the seat of cloudy swelling, and sometimes of hemorrhagic infarct and extravasation. The *spleen* shows the most constant alterations, being enlarged, but in size it exhibits a great variability. Infarction is frequent, and the lymphoid element of the bone-marrow often shows hyperplasia. If jaundice has been present during life, it is visible after death.

Etiology.—**Bacteriology.**—In 1873, Obermeier discovered in the blood of patients suffering from relapsing fever a special organism, the *spirillum Obermeieri*, and subsequent investigations by others have fully confirmed his observations with reference to the causal relation of this micro-organism to relapsing fever. The specific agent, or *spirocheta*, is a delicate filamentous organism of spiral form and much elongated, its length equalling four to six times the diameter of a red blood-corpuscle (Fig. 8). Examined under the microscope during a pyretic period, it is seen to exhibit active motion among the blood-cells, this motion being spiral and following the long axis of the organism. It is aërobic, and may be demonstrated in *dry blood* by staining with anilin colors, but the spirillum has never been found in other fluids or secretions of the body. It is also apparent in the blood only during the paroxysms, and Dr. Van Dyke Carter's careful studies have shown that by inoculation of the blood containing spirillar organisms or their germs the disease may be conveyed to new or old subjects. Shortly before the crisis the spirilla disappear from the blood, and are, as a rule, absent during the whole of the succeeding apyrexial period, and inoculation now fails to produce the disease. After death they are found in all the organs, but they have not been cultivated successfully on artificial media, and little is known of their life-history.

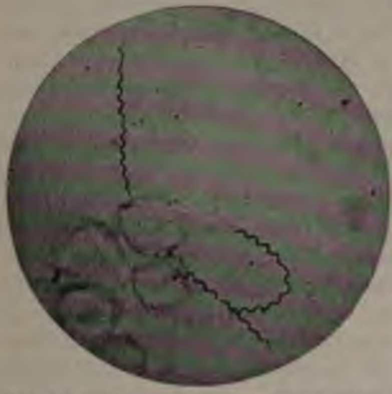
FIG. 8.—Bacillus of relapsing fever (from human blood); × 1000 (Günther).

Predisposing Causes.—*Age.*—The complaint is most common in young adults between fifteen and twenty-five years.

Sex.—A larger proportion of males than females is affected.

The disease is especially apt to prevail in times of famine, and amid antihygienic surroundings.

Mode of Infection.—Tictin's studies indicate that the medium of transmission may be through suctorial insects (as bedbugs).

Clinical History.—The **incubation** period ranges in its duration from four to ten days, though sometimes it is even briefer; and in this stage certain symptoms (malaise, fugitive pains, etc.) may appear.

The **invasion** is quite *abrupt*, often occurring on awakening in the morning, and commonly the attack is ushered in with a severe ***rigor***, though there may be only a repeated slight shivering. The chief accompanying symptoms are frontal headache, vertigo, severe pains in the loins and limbs, and marked physical prostration. The *temperature* rises soon, and often rapidly, reaching 105°–106° F. (41.1° C.), or higher still, on the first or second day. The *skin* is dry and pungent, and presents very soon either a "characteristic dirty-yellow color" or a distinctly bronzed appearance. The *cheeks* are flushed, the eyes sunken, and profuse perspirations often take place (sometimes alternating with chills), in consequence of which sudamina are frequently observed. Other forms of eruption have been described, but none that are either constant or characteristic. In certain epidemics *herpes labialis* has been very generally noticed. At first the *tongue* is moist and coated with a yellowish-white fur, and later it may become brown, dry, and fissured, with sordes on the teeth. *Ulcerative stomatitis* has been observed occasionally, and catarrhal pharyngitis and mild tonsillitis may be evidenced by pain on swallowing and other symptoms. Among the *earlier* symptoms are excessive thirst, anorexia, nausea, and vomiting. The vomitus may be yellowish-green, green, or even black in color, and consist of bile in varying proportions (rarely, also, blood) and gastric secretions. Constipation often precedes invasion, and is apt to continue throughout the attack.

The *pulse* rises rapidly with the temperature, though the normal ratio between the two is not maintained. At first the pulse is full and strong, and its beats number from 100 to 140 or more per minute; but in serious cases it becomes weak, irregular, or even intermittent, while at the same time the heart-sounds grow more and more feeble and indistinct. Hemic *murmurs* may be audible. The *nervous* derangements are not of a grave character, but the headache persists and is severe throughout, and the patient is restive and sleepless. Delirium is not common, and, though occasionally this symptom assumes a prominence toward the crisis, the intellect remains clear as a rule. The *urine* presents the ordinary febrile characteristics, and may contain albumin and casts. It also contains bile-pigment when jaundice is present. The *respirations* are accelerated, and immediately preceding the crisis urgent dyspnea may be developed.

The **physical signs** during the febrile paroxysms are few. The epigastric region and the nerve-trunks are tender to the touch, while the skin-surface and certain muscles are often hyperesthetic. *Palpation* detects a variable degree of enlargement of the spleen and liver, and the signs of bronchitis, of lobular pneumonia, and of hypostatic congestion of the lungs may be present. The symptoms above detailed persist with slight daily fluctuations of temperature till there occurs a turning-point.

The Crisis.—This occurs from the fifth to the seventh day, and rarely as late as the tenth. It is sometimes heralded by a critical rise of temperature, the mercury touching 108° F. (42.2° C.), but evidenced chiefly by a rapid fall of temperature (within twelve hours) to or below the normal, with profuse sweating. Coincidently, all other symptoms disappear with marvellous rapidity. The critical sweat may be replaced by diarrhea, intestinal hemorrhage, metrorrhagia, or epistaxis, and then follows a speedy afebrile convalescence, so that after the lapse of a day or two the patient expresses himself as being well.

During the *intervals* between the paroxysms the skin may exhibit a faintly jaundiced tint; there may be trivial evening exacerbations of temperature, particularly if complications be present and outlast the fever stage; and the spleen is evidently enlarged. There may be, though rarely, but a single paroxysm. As a rule, at the expiration of the second week there will be a recurrence of all the active symptoms of the primary attack, including the rigor or fits of chilliness and fever. Quite frequently a third pyrexial stage takes place, and rarely a fourth or even fifth.

The **duration** of the first relapse is briefer than the primary pyretic stage, and if there be subsequent relapses, each succeeding one is separated from its predecessor by the usual apyrexial period, but is briefer and lighter. Hence, should a fourth or a fifth febrile period occur, it is, as a rule, quite rudimentary. The relative duration and severity of the different febrile periods, their manner of recurrence, and the course of the fever are considerations that can best be appreciated by a glance at the accompanying temperature-chart (*vide* Fig. 9).

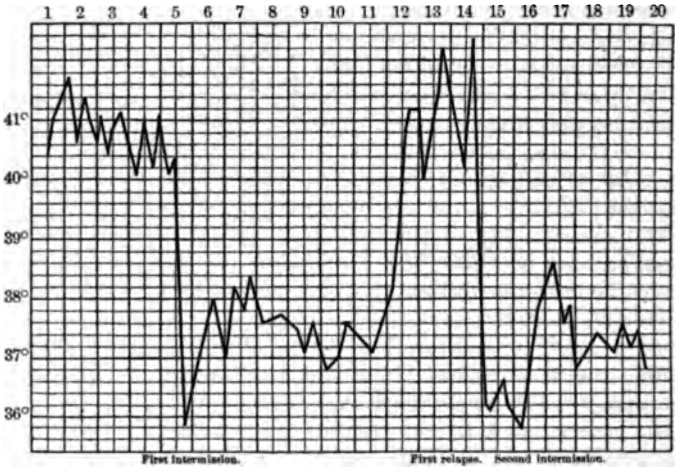

FIG. 9.—Temperature-curve of relapsing fever.

Complications.—These are not frequent. At the head of the list stands *lobar pneumonia*, and next comes *broncho-pneumonia*, which is always secondary. Other conditions, belonging to the latter class, are septico-pyemic processes, iritis, irido-choroiditis, suppurative parotitis, laryngitis, entero-colitis, and neuritis. In pregnant women abortion may take place. Epistaxis has been noted, and has even proved dangerous in some epidemics. Acute hemorrhagic nephritis is a very rare but serious complication when it does occur, and may be dependent upon the primary affection. As the result, most probably, of the very high temperature the heart may become exhausted, and the occurrence of sudden paralysis is not unknown.

Clinical Varieties.—The difference in the general course of cases in different epidemics, and even in the same one, is, for the most part, the direct result of the varying degrees of intensity of the infection. Thus very *light* or even rudimentary cases occur in which the whole course may be made up of one or two brief febrile periods, and their resemblance to ordinary intermittents may be close. The so-called "*bilious typhoid*," which is a form of relapsing fever, occupies the other extremity, being of malignant type. It is sometimes characterized by the usual symptoms of the disease, only greatly intensified; but more often, perhaps, the condition early merges into a typhoid state, to which are added certain grave features and complications (marked icterus, hematemesis and hemorrhages from other outlets of the body, uremia, sudden collapse, etc.). Septic and pyemic processes, including infarctions, are common accompaniments, and the outcome is frequently unfavorable.

Diagnosis.—The prevalence of an epidemic in which the cases present similar symptoms; the sudden onset; the course and intensity of the fever with its concomitants; the termination by crisis on or about the seventh day; and the peculiar manner of repetition of the fever-attacks after an afebrile period of equal duration,—are points that distinguish relapsing fever from other affections which simulate it more or less closely. Additional symptoms that are of special value for diagnosis are—enlargement of the spleen and liver, a negative character of the nervous and a prominence of the gastric phenomena, and jaundice. To be able to state that relapsing fever is positively present the *spirocheta Obermeieri* must be found in the blood, and this is particularly true in the earlier cases of an epidemic, before they have passed through their typical relapses. To demonstrate the presence of this parasite in the blood during the fever-stage is not a difficult task. A drop of blood obtained from the finger-tip is to be examined microscopically without previous dilution. On account of their size and motility the spirilla can be readily detected, and usually the attention of the examiner is first arrested by the peculiar joggling movements of the red blood-corpuscles. Then the real disturbing agents appear as slender spirals with a snake-like motion. Their identity may be confirmed by staining with anilin colors, and, in exceptional cases, by injecting them into the blood of the monkey, in whom they produce the disease.

Differential Diagnosis.—*Typhus fever* may be mistaken for relapsing fever, since both have the same predisposing causes, both prevail epidemically, both are characterized by an abrupt onset, with or without prodromes, and by a continued type of fever. Certain points of distinction, however, serve to separate them reliably. In relapsing fever the eyes are clear but hollowed, the cheeks are flushed, and there is a dirty-yellow tint of skin; in typhus the eyes are injected, the pupils contracted, the face wears a stupid, inanimate expression, and there is in addition the characteristic maculopetechial eruption. In relapsing fever, delirium and stupor are rare, the period of fever briefer, while the blood shows the presence of the spirillum. In typhus relapses are the exception. *Yellow fever* resembles relapsing fever in its general course, but in the former the stage of remission is both briefer and more incomplete. Yellow fever presents a stage of collapse with black vomit, and jaundice is more intense. The spirilla may be detected in the blood, and there is an extraordinary splenic enlargement in relapsing fever.

Pel and Ebstein have described a *febrile condition* which sometimes occurs in pseudoleukemia and simulates that of relapsing fever; but it may be distinguished by the absence of the spirilla from the blood, the general enlargement of the lymphatic glands, liver, and spleen, and the fact that the pyrexial periods do not tend to grow shorter.

Prognosis.—The prognosis of relapsing fever is good, but of "bilious typhoid" it is bad indeed. Apart from the type, we must consider, in this as in all other acute infectious disease, the number, character, and frequency of occurrence of the various **complications**. As stated, these are few, infrequent, and mostly benign. Among those signalizing danger are severe hemorrhages (epistaxis, metrorrhagia, hematemesis, etc.), premature labor, signs of uremia and syncope, marked jaundice and excessive vomiting, urgent diarrhea, etc. Perhaps the most frequent causes of death are pneumonia and acute hemorrhagic nephritis. Individual circumstances exert an influence upon the prognosis, and of those that render it more grave are the want of good nursing, privation, a previously enfeebled system, and old age (the disease being more fatal in elderly than in younger subjects).

The **duration** depends upon the number of paroxysms, since the latter are of definite length. In the majority of cases there is but one relapse, and in this event the disease lasts from eighteen to twenty days.

Treatment.—The general management, including the time and use of stimulants, must be based on the same principles as are employed in typhoid fever. The fever, as well as the nervous and other leading symptoms, is to be opposed by the cold or gradually cooled bath, employed as indicated in the article on the treatment of the latter disease. If, as may happen, there are adequate reasons why balneo-therapeutics cannot be used, then cold spongings, with the ice-cap or the cold pack, may be tried. Internal antipyretics may be reserved for use in cases in which the temperature is very high and the above-mentioned means are impracticable. Small doses of phenacetin (gr. ij to v—0.1296 to 0.3240) or acetanilid (gr. ij to iij—0.1296 to 0.1944) are to be administered, at the same time guarding the heart, and the signs of collapse must be promptly met by the free yet prudent use of stimulants (strychnin, alcoholics, ammonium, etc.). Vomiting often induces marked debility, and calls for the use of ice or iced champagne and small doses of cocain, morphin, or dilute hydrocyanic acid, preceded by a mercurial laxative. Counter-irritation over the epigastrium is also useful. For the intense muscular pain, restlessness, and sleeplessness nothing is so good as morphin given subcutaneously, and Dover's powder may be employed if. the pain be of moderate severity. During the intermissions the patient should be kept indoors for ten days or more, lest exposure or sudden exertion predispose him to a relapse. Solid food may now be gradually resumed, and tonics judiciously given. H. Löwenthal[1] treated 131 cases with antispirochetic serum (obtained from a horse that had been treated with blood containing the spirochetæ) with but 1 death. The treatment of *relapses* differs in no way from that of the first febrile period.

[1] *Deutsche med. Woch.*, October 27 and November 3, 1898.

MALARIAL FEVER.

(*Chills and Fever; Fever and Ague; Swamp Fever.*)

Definition.—An infectious, non-contagious disease caused by the hematozoa of Laveran. It is characterized by splenic enlargement, brief febrile attacks which recur periodically, melanemia, and a tendency in protracted cases to irregular fever and extreme anemia. The following sub-varieties will be discussed: (I.) *Intermittent fever;* (II.) *Pernicious intermittent;* (III.) *Remittent fever;* (IV.) *Malarial cachexia;* (V.) *Masked intermittents;* and (VI.) *Malarial hematuria.*

Historic Note.—There are few diseases with which the profession has been acquainted longer than with malaria, and chief among the earliest known hotbeds of this disease were the city of Rome, the Pontine marshes about the latter, and the swamps along the lower Danube. It is pretty generally believed that the prevalence of the disease long has been, and still is, diminishing. This view is fully corroborated by my own observations upon the cases from four leading hospitals of Philadelphia, and is doubtless due to the drying of marshy districts of a malarious character, thereby rendering the districts unsuited to the development of the mosquito.

New England, once a region in which the disease was very prevalent, now affords few cases. Again, in the southern portion of the United States the severer forms of malaria prevailed extensively in the past, but a marked tendency to progressive reduction in the number of cases has also been observed here. In foreign lands (England and Continental Europe) the constantly decreasing prevalence and virulence of this disease have been noted by numerous careful observers. The relation between malaria and the mosquito is suggested in the writings of such ancient authors as Columella and Varro. The peasants say "in such a place there is much fever, because it is full of mosquitoes." Shepherds returning from the European mountains to their cabins smoke them, to drive out the mosquitoes, and often the sheep occupy the cabin for a time, when the famished mosquitoes inflict their bites upon these animals, after which they show little tendency to bite man (an ancient prophylaxis). Mbù is the term used in Eastern Africa for both malaria and the mosquito.

In 1848 Noth, of America, maintained that both yellow fever and malaria were transported by the mosquito, and King, in 1883, showed that malaria was transmitted in this manner. In 1891 Laveran recognized the mosquito as an intermediary host of this parasite. Similar views were held by Pfeiffer and Koch in 1892, Bignami in 1894, and Manson in 1896.

Pathology.—The chief and most constant morbid lesions are attributable to the direct effect of the malarial parasites upon the blood. The symptomatic anemia (often quite pronounced) results from the destruction of red corpuscles by the parasites. There is a marked tendency to an accumulation of pigment in the blood and in certain of the internal organs, particularly the spleen and liver. To account for this is the fact mentioned in the description of the amebæ (*infra*) that the hemoglobin of the blood is converted into melanin (pigment) by the organisms. The malarial parasite also engenders a toxin which may be in part responsible for the morbid lesions of the disease.

The spleen is engorged with blood, and at first is swollen (chiefly during the febrile paroxysm), but it soon becomes permanently enlarged ("ague-cake"). A rare accident in intermittent fever is rupture of the spleen. Hemorrhagic infarcts are occasionally presented by this organ.

The liver is also engorged, but not to the same extent as the spleen.

The heart-chambers may be found to be acutely dilated. Neuritis has been observed by Gowers, Bamstark, Ewald, and V. P. Gibney. W. G. Spiller[1] reported a case that showed partial sclerosis of the motor tracts, and recent hemorrhages within the left internal capsule.

Etiology. — **Bacteriology.** — At present our knowledge of the malarial parasite may be discussed under five heads: (1) Discovery of the Plasmodium malariæ in the blood of persons suffering from the disease. (2) Its developmental cycle (sporulation), as shown by Golgi, in 1885. (3) The discovery, by McCallum, of its method of sexual fertilization. (4) Its development in an intermediate host (the mosquito), as first pointed out by Surgeon-Major Ross. (5) The observations of the Italian school, showing its method of re-entering the tissues of man.

(1) It remained for Laveran to discover the malarial parasite in 1880, but it was not until 1883, when Marchiafava, Celli, and Golgi published their confirmatory investigations, that the profession accepted malaria as a disease of parasitic origin.

(2) Laveran and Golgi observed that certain parasites, especially those found in estivo-autumnal fever, developed into peculiar crescentic bodies (gametocytes). Other more rounded, ring-like bodies were seen to display abnormal agitation in from ten to fifteen minutes after being withdrawn from the body, followed by the discharge from the protoplasm of several filamentous bodies, or flagella (microgametocytes). The latter were seen to separate from the parent-cell, after which they were found swimming independently in the blood.

(3) The significance of both the crescentic and flagellated bodies was first described by MacCallum,[2] of America. While studying the life-history of the "halteridium," it was noted that a limited number of ellipsoid bodies were produced, corresponding to the crescentic bodies seen in human malaria. Certain of these bodies were hyaline, others granular, and it was the former variety only that developed flagella. A flagellum, on swimming away from the mother-cell, was seen to display peculiar agitation on approaching one of the granular bodies (crescentic). Other similar hyaline bodies also directed their movements toward this granular ellipsoid, which became agitated by their approach. One of the flagella was seen to enter the granular body and effect a symbiosis with it (*sexual fertilization*). Fertilization was followed by a short rest, after which the granular body was seen to assume a worm-like form, and then swim slowly away, its pointed end directed foremost and trailing behind it pigment-particles, which were formerly situated within its

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[1] *Amer. Jour. Med. Sci.*, December, 1900.

[2] *London Lancet*, Nov. 13, 1897.

protoplasm. Later MacCallum was able to confirm these investigations in a case of malaria in man (estivo-autumnal).

(4) Manson correctly supposed that the mosquito sucked blood from persons sick of malaria; but his subsequent deductions and the suppositions on which they rested have been shown by MacCallum to be erroneous.

Surgeon-Major Ross,[1] of Liverpool, convinced by Manson's theory, began his investigations in India, in 1895, by endeavoring to determine what became of the parasite after entering the mosquito's stomach. During August and September, 1897, two members of the species (genus Anopheles), bred from the larvæ, were fed on the blood of patients containing crescents, and he found certain peculiar spheroidal cells on the wall of the stomach, which convinced him that "these cells constituted the long-sought mosquito stage of the parasite" (*zygote*).

In 1898 Ross studied the "zygotes of protozoma" of birds. He found that they attached themselves to the outer coats of the mosquito's stomach. The zygote grows rapidly, without movement or change in form, protruding into the insect's body-cavity. Later its capsule becomes easily perceptible and the cell-substance is seen to divide into from ten to twelve "meres." In from one to three weeks, depending on the external temperature, the zygote matures, when each mere contains a number of delicate, "thread-like" blasts.

The next important step in the development of the parasite is the rupture of its capsule, setting free these "thread-like" blasts within the insect's body; they are then carried by the blood's currents to all its tissues, particularly into the insect's salivary gland, where they are found in large numbers. The common duct of the salivary gland of the mosquito (genus Anophelės) passes along the middle stylet of the proboscis, opening at its extremity, thereby necessitating that a portion of the secretion of this gland be poured into the wound caused by the insect's bite. In the human body the blasts return to the amebulæ with which the life-history of this parasite began.

(5) In 1898 Grassi found three chief species of the mosquito in malarious localities: the Anopheles claviger was constantly present, while two varieties of Culex were also observed.

In Nov., 1898, Bastianelli, Bignami,[2] and Grassi conducted a series of experiments, by feeding mosquitoes the blood from persons suffering from estivo-autumnal fever, confirming the findings described by Ross, in Aug., 1897. These investigations showed further the *mode of infection;* that healthy mosquitoes become infected by sucking blood from malarial patients, and that in from eight to twenty-one days such insects may infect healthy men with the disease by their bites. One mosquito may infect many persons, and may possess this power for an indefinite period, "since not all of the germinal threads escape from the venomo-salivary gland."[3] Both Ross and the Italian workers have found that neither the common house mosquito (genus Culex) nor the Anopheles nigripines take part as an intermediary host for this parasite. The fact that mosquitoes (Anopheles claviger) are known to occupy non-malarious districts proves the innocence of the uninfected insects. A single case of malaria transported to such territory, and subjected to the bites of these same mosquitoes, often results in an epidemic.

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[1] *Brit. Med. Jour.*, Dec. 18, 1897.

[2] "Malaria and Mosquitoes," *Lancet*, Jan. 13, 1900.

[3] *Jour. Amer. Med. Assoc.*, Feb. 3, 1900, A. Woldert.

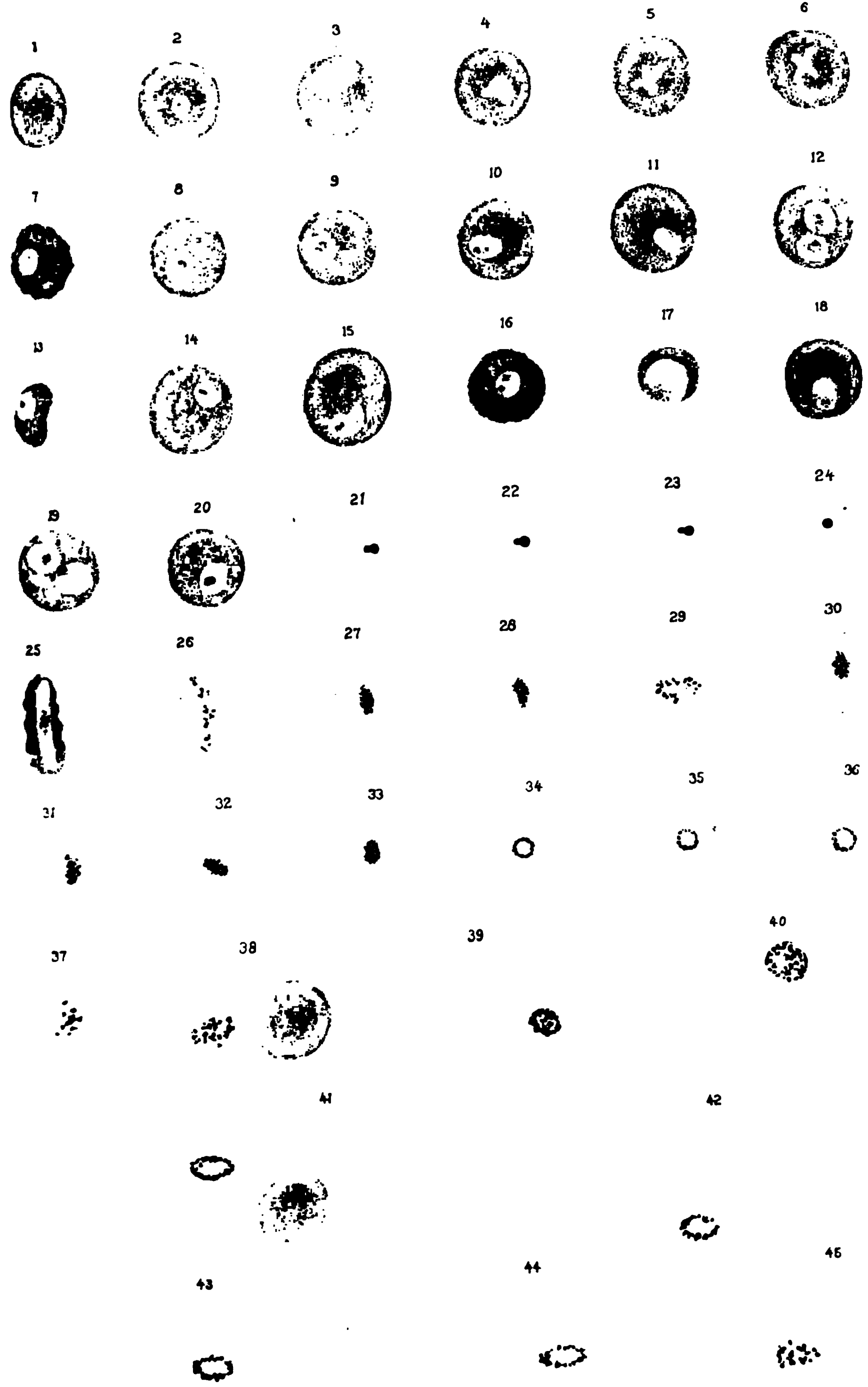
The *malarial parasite* of Laveran belongs to a subclass of the protozoa known as hematozoa. Of the latter, three varieties, corresponding with the three leading clinical forms of the affection, have been distinguished, and the evolution of two of these parasites at least takes place within the red blood-corpuscles. They enter the red cells in the form of small, non-pigmented plasmodia, exhibiting ameboid motion, and then feed upon their host, transforming, at the same time, the hemoglobin of the latter into dark pigment-granules as they develop. When the intraglobular plasmodia have consumed the red blood-corpuscles the granules of pigment accumulate in the center of the parasite, while on its periphery the processes of subdivision and sporulation are taking place, forming fresh generations of hematozoa. These young parasites assume the form of minute, more or less spheric, hyaline bodies, which again enter the red blood-corpuscles and start on a new cycle of development. It is probable, as Golgi suggests, that the third variety is not intimately connected with the circulating medium, but that its evolution principally takes place in the internal organs (spleen, bone-marrow, etc.). The special varieties of the malarial parasite will be described separately.

(1) *The Ameba causing Tertian Intermittent Fever.*—This begins its cycle of evolution in the red blood-corpuscle as a small hyaline ameba. Its development is attended with the appearance in its interior of fine, brown, motile granules in the form of pigment, and when matured it about equals the size of a normal red corpuscle. It now assumes a spheric form, the pigment collecting centrally, and sporulation into fifteen to twenty or more segments follows. The tertian parasites are exceedingly numerous in the blood, and pass through the various stages of their life-cycle almost simultaneously, the sporulation of an entire generation occurring within the space of a few hours (Golgi). The occurrence of the malarial paroxysm follows the process of sporulation, which is attended, most probably, with the development of a *toxin*, and the symptoms of the disease may be attributable chiefly to the effects of the latter. The red corpuscle that includes the parasite becomes enlarged and decolorized as the latter develops. The parasite of tertian intermittent runs its cycle in about forty-eight hours. Hence infection by a single generation would result in sporulation every second day, followed by the malarial paroxysm. Quite commonly, infection by two groups of parasites occurs on successive days, and, since each has a definite period of evolution, a daily malarial paroxysm is the result (quotidian intermittent). Multiple infection with this parasite may occur, but with great rarity.

(2) *The Ameba causing Quartan Fever.*—This cannot be distinguished from the tertian parasite at the beginning of its brief career, but later differences are clearly perceptible. Its ameboid movements are more deliberate, and its pigment-granules are coarser, darker, and also less motile than those of the tertian organisms. Unlike the latter, it does not attain the size of the red corpuscles, and during sporulation the segments (five to ten in number) encircle in an orderly way the central

PLATE II.

The Parasite of Aestivo Autumnal Fever.



pigment-mass or clump, "rosettes" of great beauty thus being formed. The red blood-corpuscle that harbors the quartan parasite contracts upon its destroyer, appears shrivelled, and its color changes at the same time from the normal to a deep greenish or bronzed tint. It sporulates about seventy-two hours after it enters the red corpuscle; hence, if only one group of parasites be present, febrile attacks occur every fourth day, forming the simple quartan intermittent. On the other hand, double quartan infection results in paroxysms on two successive days, followed by an intermission lasting one day, while triple infection, or the presence of three groups, causes daily paroxysms—the quotidian intermittent. Infection by more than three groups of the quartan parasite may occur, but is very rare.

(3) *The Ameba causing Estivo-autumnal Fevers.*—The cycle of this variety is evolved, chiefly, in certain of the internal viscera, and the microscopic examination of the blood in the various stages of the disease does not always give a positive result, as in the tertian and quartan types. The organism invades the red blood-corpuscle, but to what extent is not definitely known. It is a quite small hyaline body, its size at maturity scarcely equalling one-half the dimensions of the red corpuscle, and it accumulates very few fine pigment-granules. The parasite may be found in the later stages in the blood from certain internal viscera, such as the spleen, bone-marrow, etc. After the condition has lasted a time characteristic oval and crescentic bodies, which are more or less refractive, may be observed in the fresh blood. These so-called "sickle-form bodies" show central rods and clumps of coarse pigment, and are especially connected with this category of malarial fevers. Ring-form bodies, and, at times, the signet-ring forms, are observed. Pigmented bodies are also present. The red corpuscle, at whose expense the parasite develops, assumes a brassy-green hue, becoming shrivelled and often crenated.

It would appear from the studies of Marchiafava and Bignami, and Surgeon Craig,[1] that two varieties of parasite are concerned—quotidian and tertian forms of autumnal fevers.

The parasites of tertian estivo-autumnal fever are larger than the quartan parasite, and during the hyaline stage the signet-ring form, sluggish ameboid movement, clear-cut refractive outline, and the occurrence of one organism in a blood-cell which is not wrinkled are observed; during the pigmented stage, the ameboid movement and fine granular motile pigment. Segmentation takes place outside the corpuscle. Crescents are large, slender, deeply pigmented, and show a clear outline.

The quotidian parasite is smaller, at times actively ameboid, and more than one parasite may occupy a single red cell, which is usually wrinkled. Their pigment is motionless, and usually in the form of short rods. Crescents are small, plump, and often present a double outline. Segmentation occurs within the red corpuscle.

Flagella (*Microgametes*).—Certain of the ellipsoid bodies are seen to display fine cilia after the blood is drawn from the body, and these flagella may be found floating in the blood, when they display decided movement. Their function, in connection with that of the crescent's, has been previously described.

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[1] *New York Med. Jour.*, Dec. 23, 1899.

Predisposing Causes.—(1) **Soil.**—Fresh-water marshes favor the development of malaria, and are most fruitful in influencing its growth when located near the coast and tainted with salt water. Again, marshy districts affording luxuriant vegetation are notorious as malarial foci. Keeping in remembrance the foregoing facts, we can readily see why malaria is unusually prevalent in certain countries (chiefly tropical), and why it is chiefly confined to the low-lying estuaries and the deltas of rivers. The same facts explain satisfactorily why certain districts which were very liable to the affection should have become, as the result of denudation of the virgin soil and its subsequent drainage and cultivation, entirely free from the complaint. Epidemics following the upturning or the removal of the surface of the virgin soil are probably due to importation of the disease, and are common on the frontier of the South and West. H. J. Waring has shown that when the water-level is highest malaria is most prevalent.

(2) **Climate.**—Malaria is more prevalent in tropical and subtropical than in temperate climates, and more common in the latter than in the polar zones. Hence it occurs more frequently in the southern than in the northern States of our own country.

(3) **Rapidly growing trees** dry the soil by absorbing enormous quantities of water. In the Roman Campagna extensive experiments have been made with the eucalyptus tree, and the results have been remarkable, districts protected in this manner becoming almost entirely free from malaria in a few years, the environment being unsuited to the mosquito.

(4) **Seasons.**—In temperate latitudes most cases are developed in the autumn, the maximal period corresponding with the month of September. This dictum is based upon 4841 cases of malaria gathered by the writer from the records of the leading Philadelphia hospitals.[1] Cases that develop before the "Anopheles claviger" makes its appearance (in June) are possibly relapses. In the tropics the case seems to be different, and two maximum periods—spring and autumn—obtain. Statistics from the hospitals of Rome, collected from 1864 to 1898, show the maximum number of cases to occur in August, September, October, November, and July, respectively, while in June the minimum number is seen.

(5) **Gravitation.**—Persons occupying the upper stories of a house or living on slight elevations are affected with relative infrequency, for the reason that mosquitoes are always found near the earth's surface, where the air currents are feeble. This fact also explains the nocturnal infection.

(6) **Race** exerts little influence in other lands than our own, but in the United States negroes are slightly less susceptible than are the whites.

(7) **Sex** is without effect when men and women are equally exposed. Cases are, however, vastly more frequent among males because of their increased liability to exposure, and particularly while following certain occupations (agriculture, marsh-draining, etc.). The 5044 cases col-

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[1] *Univ. Med. Mag.*, May, 1897.

lected by myself gave the numerical proportion of 6 to 1 in favor of males.

Immunity.—There are individuals immune from malaria and experimental malaria. An individual may present this property after a mild fever has been cured by quinin. Maurel has shown that all races are equally susceptible, but that by living in a malarious district whites may in time show marked immunity. By the use of methylene-blue and euchinin an immunity may be established against the inoculation of from 1 to 2 grams of estivo-autumnal blood.

Incubation.—According to Bignami and Bastianelli, the period of incubation for experimental malaria is: Quartan, 15 days; spring tertian, 12 days; estivo-autumnal tertian, 5 days. The administration of potas. brom., potas. iod., arsenic, carbolic acid, antipyrin, and phenocoll may result in a longer period. Angelo Celli has seen spring tertian show incubation of 22 days, and the estivo-autumnal tertian 17 days.

Epidemiology.—Estivo-autumnal fevers are rare in their recurrence, while mild tertian and quartan prevail with each new spring, and the first cases of tertian are noted to occur in the same house in which the last recurrence of these fevers appeared. After the first cases there is a lapse of from seventeen to eighteen days, after which the epidemic spreads. The life and habits of the Anopheles have a direct bearing upon epidemics—" either the first cases of these fevers in July are recurrences of a previous infection, or the very first cases of these fevers in July are primary" (Celli). " Both hypotheses are possible. In both we have to deal with a contagion circulating, so to speak, between the temporary host (man) and the definitive host (mosquito), a contagion which, by means of the blood of the relapsing cases of the preceding year, is transmitted by the agency of mosquitoes, and starts the epidemic of the following year." There are many interesting questions not yet explained.

(I.) **Intermittent Fever.**—**Symptoms.**—The clinical history presents itself under two heads: (*a*) the paroxysms, and (*b*) the manner in which the paroxysms recur.

(*a*) *The Paroxysms.*—There may be premonitions lasting from one to several days, and most significant, yet not distinctive, are headache, pain in the nape of the neck, yawning, a yellowish complexion, and a slight splenic enlargement. In a large proportion of the cases, however, the onset is abrupt. Typical paroxysms present three stages—*chill*, *fever*, and *sweating*. The chill is intense, causing shivering, and often chattering of the teeth. Malaise is marked, the skin is cool and pale, face slightly cyanotic, and limbs painful. This stage usually occurs in the morning hours, though the time of onset is by no means constant; its duration, also, varies greatly, generally lasting from one to two hours. The internal temperature rises rapidly; the pulse is small, rapid, and of high tension.

The *hot stage* succeeds the chill, and, in striking contrast with the first stage, the face wears a decided flush and the skin is burning hot to the touch. The temperature continues to rise, but not so rapidly as in the first stage. Its maximum level, usually from 104° to 106° F. (40° to 41° C.), is soon reached, and may either be maintained uniformly for several hours, or the curve may show two small summits if the temperature be recorded frequently (Fig. 10). The pulse is full and bounding, except in the rare instances in which acute dilatation of the heart ensues, when it is quite feeble and sometimes irregular. The length of the second stage exceeds that of the first, being from three to six hours. The temperature generally begins to decline before the close of the febrile stage.

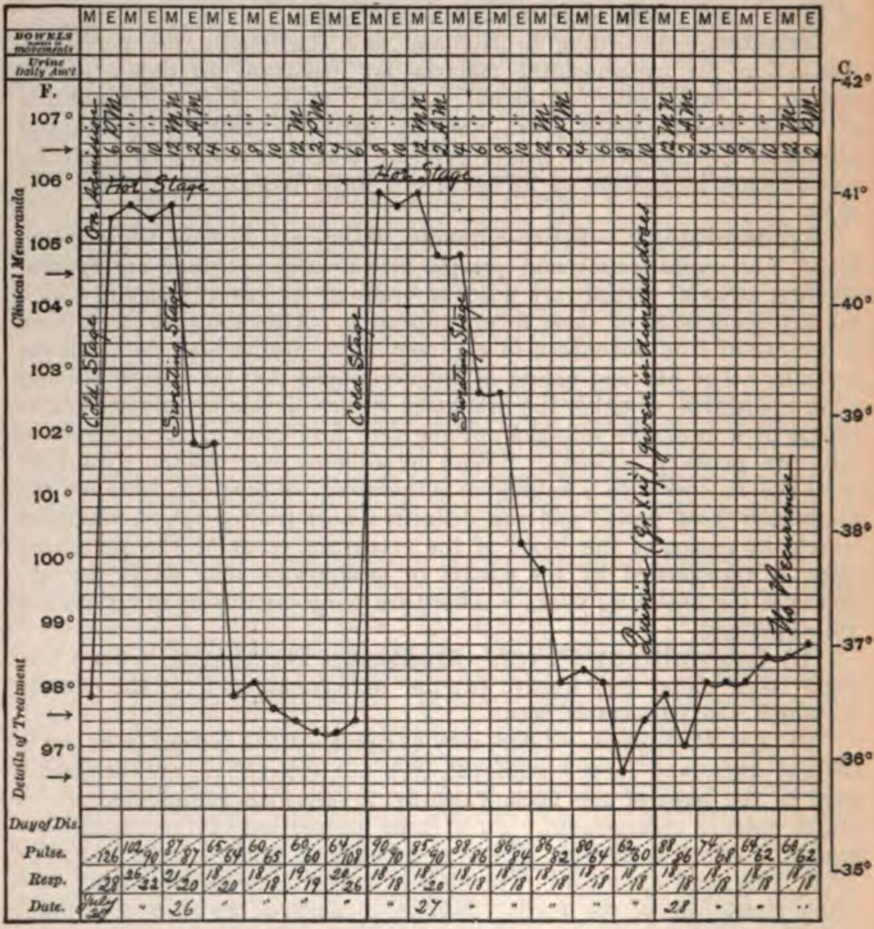

FIG. 10.—Temperature-curve in a case of double tertian fever. C. F. C., aged forty-one years.

When *sweating*, which soon becomes profuse, sets in, the symptoms of the hot stage are promptly relieved. The temperature falls by crisis, touching the normal level in a few hours; the decline, however, is less rapid than the rise at the beginning of the paroxysm. The fall may be unbroken by any fresh elevations of temperature, though more often the latter occur, and less frequently defervescence occurs by steps, the temperature dropping one or more degrees, and remaining at the new level for a short period. It again drops about an equal distance, and so on until the normal is reached. Usually, following the paroxysm, the temperature becomes subnormal (about 97° F.; 36° C.). The length of the typical malarial paroxysm ranges, in all save exceptional instances, from eight to twelve hours.

(*b*) *The Manner in which the Paroxysms Recur.*—The special characteristic of this form of intermittent is the regularity with which the paroxysms recur in cases that are not under treatment. The intermission, or time between two successive paroxysms, is most frequently twenty-four hours (quotidian intermittent fever); almost as often it is forty-eight hours (tertian intermittent); and less frequently it is seventy-two hours (quartan intermittent). If there be two paroxysms on one day—a rare occurrence—the term "double quotidian" is used to designate the case. Of the above types, as stated in the life-history of the parasite, two only—the *tertian* and the *quartan*—have been clearly distinguished. The quotidian ague (the most frequent clinical variety) is generally due to double infection by the tertian parasite, and very rarely is it to be attributed to the presence in the blood of three groups of the quartan parasite, resulting in daily sporulation. It sometimes happens that the paroxysms recur a couple of hours later each successive day, when it is called a "*retarding*" intermittent fever, or they may recur a little earlier, when the term "*anticipating*" is employed.

Other More or Less Characteristic Symptoms.—Apart from the paroxysms and the regularity with which they recur, *splenic enlargement* is almost always present, and hence is of considerable clinical import. After the first paroxysm or two the swelling is usually marked and demonstrable, especially by palpation. The organ can be shown to increase in size with each succeeding paroxysm. Tenderness is elicited on pressure, and commonly outlasts the course of the affection for a considerable length of time. Moderate enlargement of the liver may be present, but this is neither so significant nor so constant as enlargement of the spleen.

Connected with the *skin* are two symptoms of considerable diagnostic value: (1) a *yellowish-brown discoloration*, the so-called "malarial complexion," due to the deposition of pigment; and (2) *herpes*. The latter occurs usually on the prolabia or on the nose, though rarely elsewhere. Other skin-eruptions, as urticaria and purpura, have been described by authors, but they have no real clinical worth.

As stated under Pathology, *acute dilatation of the heart* may develop, attended with the usual physical signs of this condition, but it rarely lasts longer than the brief febrile paroxysm. Murmurs of functional origin may also be heard in the heart during the attack, and the *lungs* upon auscultation sometimes present the signs of a dry bronchitis.

The *urine* may contain a small amount of albumin, and rarely there is acute nephritis—a rather common sequel in the negro. There is a temporary increase in the amount of urea eliminated, and this may be observed from two to six or eight hours before the chill, so that an approaching paroxysm can be foretold if a quantitative analysis of the urine be made at the proper time (Jaccoud).

Gastro-intestinal symptoms may be present, but are not prominent, if we except a diarrhea which is sometimes considerable. *Catarrhal* *jaundice* and paroxysmal vomiting may be observed, but these are limited to the graver forms of intermittent.

There is no *leukocytosis*, but there is a rapid diminution in the number of both red and white corpuscles, proportionate "to the severity and the number of the attacks" (W. W. Johnston).

Clinical Varieties.—Besides the typical attacks, *mild* or *rudimentary* forms are met with, these either being due to slight infection or appearing as the remnant of cases of usual severity after active treatment. The separate stages of the febrile attacks are not well marked, and one or more may be missing; thus the chill may be absent (dumb ague), and less frequently the sweating stage may fail to appear.

In *children* there is no rigor noticeable. They grow pale, the visible mucous membranes often being slightly livid during the chill, and the paroxysms may be initiated by a convulsion or by other nervous phenomena. Acute nephritis is a rather frequent sequel in white children.

(II.) **Pernicious Malarial Intermittent.**—This truly serious form occurs chiefly in highly malarial districts, and rarely also in the widespread regions in which the simple variety prevails. Hence in the United States it is encountered most frequently in the Southern and Southwestern States. In this form of malaria the parasites of estivo-autumnal fever are constantly associated. The paroxysms do not recur with strict regularity, and the primary paroxysms are rarely pernicious in character; but second or subsequent attacks may, in addition to the usual symptoms, present the gravest phenomena.

Pathology.—This type of malaria may arise (1) as a fresh infection, and (2) as a reinfection.

(1) *Infection.*—The blood is more or less hydremic, and the blood-disks are in all stages of disintegration. The *spleen* is considerably swollen, soft, and its parenchyma is turbid and lake-colored, all its tissue elements being more than naturally pigmented, though this may not be macroscopically appreciable. Upon microscopic examination, however, pigment-granules and red corpuscles containing parasites and phagocytes are observed, particularly in the pulp adjacent to the arterioles. The *liver* is enlarged, soft, and turbid, and pigmentation occurs, but it is also microscopic. In the minute vessels phagocytes and parasites containing pigment are perceptible within the red corpuscles, and numerous small necrotic areas have been observed. The *kidneys* show microscopic pigmentation, most marked in the vicinity of its blood-supply. Minute areas of cell-death are sometimes seen. The *brain* may be abnormally colored, assuming in severe cases a chocolate tint, and in mild types a lighter hue. The brain-tissue is often anemic, and more rarely edematous. Occasionally there is congestion. The minute vessels and capillaries are literally blocked with phagocytes and blood-disks more or less disintegrated (containing parasites), and perivascular infiltration and minute hemorrhages may rarely occur, producing a focal lesion.

(2) *Reinfection.*—The blood is often extremely hydremic. The *spleen* may or may not be much enlarged, and is usually quite firm, with a well-marked pigmentation that is obvious to the naked eye. The *liver* is, as a rule, increased in size to a moderate extent only, and is some-

what indurated, while macroscopically it is seen to be deeply pigmented. The changes presented by the *kidneys* differ in no essential manner from those of the liver. The microscopic appearances of the liver, spleen, and kidneys, apart from the fact that the amount of pigment present is relatively greater, are entirely analogous to those met with when a fresh infection occurs.

Clinical Varieties.—Three varieties merit description:

(*a*) *Congestive Chills* (*Algid Form*).—These are accompanied by raging gastro-intestinal symptoms (vomiting, purging, etc.), inducing systemic collapse, which simulates to a nicety the algid stage of cholera. The temperature of the interior of the body is much elevated. True dysenteric symptoms may arise, and in a certain proportion of the cases jaundice, followed by grave nervous symptoms, may be a secondary development. This condition is to be discriminated from yellow fever, with which it has frequently been confounded. The parasites in this affection center in a special manner in the gastro-intestinal mucosa, in the vessels of which they may be seen in unusual numbers, sometimes forming distinct thrombi. In the United States this is the most common among the pernicious forms.

(*b*) *Hematuric Pernicious Malaria.*—In this form the chill is severe and prolonged, and during the hot stage the urine is bloody and scanty, containing considerable albumin, with bloody, epithelial and granular casts. Hemorrhages from other outlets of the body (mouth, rectum, vagina, nares, etc.) may also occur, together with larger and smaller cutaneous ecchymoses, and the yellowish-brown malarial complexion is intensified. The mind may remain clear throughout, although the patient is restless and anxious. Urinary suppression may ensue, and uremic toxemia be superadded; the greatest dangers being cardiac failure, uremia, and delirium (or coma independently of the latter). Death is rarely the direct consequence of excessive loss of blood.

(*c*) *Comatose Form.*—The chill may be absent. Grave cerebral symptoms, as acute delirium or sudden coma, seize the patient violently. The hot stage is attended with high fever, and if the patient survives the paroxysm, the violent nervous symptoms either disappear suddenly with the appearance of the sweating stage, or may outlast the latter by several hours. Primary paroxysms rarely prove fatal, but recurrences bring imminent danger. This dangerous variety is due to an inordinate localization of the malarial parasites in the brain, where they form complete thrombi, and induce, as a consequence, pathologic lesions in the adjacent structures.

(III.) **Remittent or Continued Malarial Fevers (Estivo-autumnal Fever).**—On account of the intensity of the gastro-intestinal symptoms this variety is also termed *bilious remittent fever*. Its severity exceeds that of intermittent malarial fever. It prevails for the great part in warm and truly tropical climates, though it is also seen in its milder forms in temperate climates. The estivo-autumnal parasites previously described are the specific cause of the disease.

Pathology.—Melanosis of the spleen, liver, and brain is generally observed; on the other hand, in rare instances in which the specific parasite had even been demonstrated during life, the internal organs were found to be non-pigmented on autopsy. The degree of the pig-

jaundice and paroxysmal vomiting may be observed, but these are limited to the graver forms of intermittent.

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served in 4.7 per cent. of the cases (Thayer); while either a slight or marked hepatogenous jaundice may appear. *Herpes labialis* is quite common. The nocturnal remission again ensues, and in the *mild types* or in those brought promptly under suitable treatment the febrile paroxysms grow briefer, resulting in an intermittent form of fever. The course of light cases is run, usually, within two weeks.

In *severe types* or in neglected cases the separate febrile paroxysms grow longer until the remissions become slight and simulate continued fevers. These are the cases that are distinguished by the same symptoms as those that mark typhoid fever, save the eruption which is peculiar to the latter. The discovery of Laveran is of the highest practical value in this category of cases. The course of the attack, if not properly treated, generally prolongs itself to three, four, or more weeks, and under these circumstances the salient features of pernicious intermittent may suddenly appear and the disease may terminate life. On the other hand, *mild forms*, in which the fever is of the continued type, also occur, and these yield promptly to the specific—quinin.

(IV.) **Malarial Cachexia.**—This is an exceedingly *chronic* condition, and is usually a remnant of one of the acute forms, particularly of the ordinary intermittents. When the latter are not properly treated, they are apt to drag on, and finally assume the characteristic features of chronic malarial cachexia. The condition may, however, develop in truly malarial localities without the intervention of primary acute malaria. It originates, however, only in truly malarial districts.

The **symptoms** are varied both in character and in intensity. There is *fever* at intervals, but chills do not occur, and the temperature-curve is typical neither of remittent nor intermittent fever, but may approximate either the one or the other. Again, the fever is sometimes wholly irregular, though its range is not high, and it seldom excels 103° F. (39.4° C.). The *skin* often presents the dirty yellowish-brown complexion to a marked degree. The spleen is enormously enlarged and indurated, and hypertrophy with hardening of the liver may also be pronounced. The *blood* is profoundly anemic, the count in one of my own cases showing but 1,300,000 red corpuscles per cubic millimeter.

Many of the local and general symptoms that remain to be given (including, in part, the fever) are chiefly dependent upon the well-marked *anemia*. Among general features may be mentioned debility, frequent sweatings, and dropsy. *Nervous symptoms* may also be noticeable, and chief among these are tremors, neuralgia, palsies, vertigo, wakefulness, and nervous palpitation of the heart. Among the rarest concomitants of this condition is paraplegia. Slight cough and dyspnea evidence the presence of mild *bronchitis;* and anorexia, nausea, diarrhea, and other symptoms mark the presence of *chronic gastro-intestinal catarrh.* The joints and voluntary muscles may be painful. *Hemorrhages* from the various mucous surfaces and into the retina are common; and I have seen one case in which spongy, easily-bleeding gums, with cutaneous ecchymoses and numerous petechiæ, pointed to the existence of associated scorbutus. *Tuberculosis* finally developed and carried off the patient. Not only the latter affection, but also *chronic dysentery*, *chronic Bright's disease*, and *amyloid disease*, may develop and prove serious complications. These cases do well, generally, if the patient can be removed permanently from the malarial district and if proper treatment be persistently pursued. In long-standing cases the spleen does not return to its natural dimensions. In all other instances, however, complete recovery may be expected, though it may require months or even years to bring it about.

(V.) **Masked Intermittent.**—This presents itself in much the same forms as chronic malarial cachexia, but with the important difference that there is no fever. This type comprises a long list of conditions, at the head of which stands *neuralgia*, most frequently involving the supraorbital branch of the trigeminus. Often a striking periodicity is observed, the painful paroxysms usually beginning in the morning and terminating in the late afternoon hours, the patient's sufferings increasing steadily in intensity until just before the close of the attack, when they suddenly abate. Among other nerves implicated with relative frequency are the occipital, the intercostals, and the sciatic. Except the blood-appearances be characteristic or unless the attacks yield promptly to quinin, a certain diagnosis of malarial neuralgia should not be ventured. Masked intermittents may assume the forms of paresthesia, anesthesia, convulsions, or paralysis; non-febrile intermittent malaria may also appear under the guise of edema, hemorrhages from the various mucous outlets of the body or into the skin, intestinal flux (diarrhea, dysentery), dyspepsia, etc. But, since these affections may all obey the law of periodicity, caution should be used in pronouncing in favor of malarial infection. Indeed, unless they yield readily to the therapeutic specific, a positive statement had better be withheld.

(VI.) **Malarial Hematuria and Hemoglobinuria.**—I have previously described a hemorrhagic form of pernicious intermittent in many cases of which hematuria is a prominent symptom. Among other general features are jaundice, prostration, nervous symptoms, and nephritis (Plehn). The blood shows pigmented parasites (forming rosettes) and sometimes crescents and pigmented leukocytes.

Boisson[1] in 3 cases of hemoglobinuric fever, occurring in soldiers attacked with malaria in Madagascar, found an enormous reduction in the red corpuscles, reaching 670,000 in 1 case, while 7 out of 10 red cells contained parasites. I have observed several instances of malarial hematuria in the Kensington district of Philadelphia, and find that they are met with wherever the moderate forms of malaria prevail. Hematuria in its severest form is seen with the approach of cold weather (Jones). It is rare in the negro.

The **symptoms** consist of a mild cold stage, a subfebrile temperature to which is added hematuria, or more often hemoglobinuria. The paroxysms may recur daily, bi-daily, or at longer intervals, and in severe forms the hemoglobinuria may be continuous, with aggravations at definite intervals. Suppression of urine may appear early, and may be accompanied by nausea, vomiting, and paresis of the bowel, and a semicomatose state, the patient presenting the general picture of uremia. The *diagnosis* demands the demonstration of the malarial parasites in the blood, and of the hemoglobin in the urine. Tyson recommends Teichmann's (hemin crystals) test to show the presence of hemoglobin. The earthy phosphates are precipitated, filtered out, and a small portion placed on a glass slide and carefully warmed until completely dry. A minute granule of common salt is carried on the point of a knife to the dried mass and thoroughly mixed with it. Any excess of salt is then removed, the mixture is covered with a thin glass cover, a hair interposed, and a drop or two of glacial acetic acid allowed to pass under. The slide is then carefully warmed until bubbles begin to make their appearance. After cooling, hemin crystals can be seen by the aid of the microscope, and, though often very small and incompletely crystallized, are easily recognizable by an amplification of 300 diameters. Chemically they are hydrochlorate of hematin.

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[1] *Rev. de Méd.* May 10, 1896.

According to Frank A. Jones, obesity occurs among persons coming from a climate free from malaria to the Mississippi's delta. They do not have chills, and have none of the chronic manifestations of malaria. "The obesity subsides rapidly by changing from a malarious to a non-malarious climate."

Complications.—The author's analysis of 1780 cases of malaria showed complications in about 10 per cent. The more common among these were not particularly grave in nature, as may be seen by a glance at the subjoined list, in which they are placed in the order of frequency of occurrence: Enteritis (16), nephritis (14), rheumatism (10), typhoid fever (8), lobar pneumonia (5), jaundice (5), and dysentery (4). The opinion of the profession is, and long has been, divided upon the question, "Has pneumonia any special connection with malaria?" but, according to the results of my own collective investigations, *lobar pneumonia* cannot be regarded as being frequently in association with the latter disease. That it is so rarely, however, cannot be denied, since the diagnosis in two of the cases was confirmed by autopsy.

Typhoid fever is a complication of malaria, according to these researches, but the relationship between these two leading affections cannot be close.

Diagnosis.—(1) **Of Intermittents.**—This is quite difficult, unless the brief febrile paroxysms, with their characteristic stages and other more or less diagnostic features (enlarged spleen, malarial complexion, and herpes), together with the rigid periodicity of the paroxysms, be present. The diagnosis is assisted by a knowledge of the fact that the patient resides in a malarial district. In cases in which a microscopic examination of the blood cannot be made early a positive diagnosis is rarely possible until after the patient has been observed long enough to ascertain the manner in which the paroxysms recur, in addition to noting the symptoms presented. The only unquestionable, as well as the most satisfactory, method of diagnosis is provided by a microscopic examination of the fresh blood.

Differential Diagnosis.—Non-malarial affections, exhibiting an intermittent form of fever, are often mistaken for malarial intermittents. Of these, (*a*) *pyemia* is very apt to be thus confounded. It will be observed, however, that the chills recur at more irregular intervals, and the more profound prostration and other general features during the intervals between the febrile exacerbations serve to distinguish it from malarial fevers. The etiologic factors and place of residence are also to be considered. In doubtful instances the blood should be examined microscopically, and, if this be impossible, the therapeutic test will, as a rule, remove the doubt. *Leukocytosis* is common in pyemia and absent in malaria.

(*b*) *Acute tuberculosis* and, more rarely, *incipient chronic tuberculosis* may present a febrile movement in no way differing from quotidian intermittent, except that in the former the pyrexia develops in the afternoon, instead of the forenoon, as in the latter. A clear history, the associated local and general symptoms, along with the results of a careful physical examination, usually render tuberculosis probable and distinguish it from malarial intermittents. Leukocytosis is common in tuberculosis and is absent in malaria, while in tuberculosis the chills recur despite the use of quinin, and this is not the case in malaria.

(*c*) *Ulcerative endocarditis* may exhibit an intermittent pyrexia, but in this affection the history is different, and the associated clinical features are more numerous and, as a rule, decidedly more grave. A blood-examination reveals leukocytosis—a distinguishing feature. Again, quinin is without effect. The irregular forms of intermittents are difficult in the extreme to diagnosticate. If, in suspected cases of "erratic" malaria, quinin is resisted, we cannot feel certain of our diagnosis unless we obtain the microscopic evidence of the presence of the malarial parasite in the blood.

(2) The diagnosis of **remittent fever** would be easily made if it did not sometimes bear a strong resemblance to typhoid fever. Its certain recognition demands the detection in the blood of the estivo-autumnal parasite. In *typhoid fever* the history points to a more gradual onset, the remissions are less marked, and there is not the epigastric oppression witnessed in remittent fever. Again, typhoid has its characteristic eruption and gives the sero-reaction.

Method of Examining the Blood for the Malarial Parasite.—The finger or lobe of the ear is carefully cleansed, and then slightly cut with a sharp lancet. The first drop of blood is wiped away and the second collected on the center of a clean cover-glass, which is immediately placed upon a clean slide and the blood allowed to spread in a thin film, and examined immediately through an oil-immersion objective. It is all-important that the blood be perfectly spread between the surfaces of the slide and cover-glass, in order that the corpuscles do not rest one against the other. In the fresh specimen one is able to detect the parasite during all its developmental stages seen in man. If the blood of estivo-autumnal fever be exposed to the air a short time and then mounted in this manner, it is likely to display flagella. If desirable to preserve the specimen or if impossible to make the microscopic examination at once, smears should be prepared by laying another cover upon the first, allowing the blood to spread in a thin layer, and then sliding them apart quickly and drying in the air. The specimen may then be fixed in a mixture of equal parts of alcohol and ether, by heat or one of the other usual methods. The most satisfactory stain is methylene blue. A few drops of a watery solution should be placed upon the cover-glass, allowed to remain about a minute, and washed off with clean water. The specimen can be examined in water or dried and mounted in Canada balsam. The organisms appear as small blue bodies, often containing pigment. Eosin may be used as a counter-stain. For the crescent and oval forms, which are sometimes difficult to find, it may be advantageous to allow a drop of blood to dry upon the cover-glass without spreading, fix as before, and then wash with dilute acetic acid; wash thoroughly with water and stain as before. The hemoglobin of the red cells is dissolved, and only the white cells and the parasites remain visible.

Prognosis.—All cases of uncomplicated intermittent fever under prompt and proper treatment will probably recover, though fatalities sometimes occur. It is to be borne in mind that in certain malarious regions and in certain seasons pernicious types are prevalent. *Primary pernicious attacks* are moderately dangerous, while recurrences are highly so. The mortality-rate in this variety of malaria is between 20 and 25 per cent.

In *remittent fever* a fatal issue may be due to asthenia, particularly when the type is severe and when, following the typhoid state, wrong notions as to treatment prevail. Suppression of urine, followed by uremic symptoms, hemorrhages, and intense jaundice, are untoward complications.

Treatment.—**Prophylaxis.**—The investigations cited above show that an individual ill of malaria is a source of danger in a community, and should be promptly treated. Localities that furnish breeding-places for the mosquito (*e. g.*, marshes) should be thoroughly drained. The homes, and more particularly the sleeping-apartments, of persons residing in paludal regions, should be protected against invasion by mosquitoes.

1. For **intermittent malarial fever** there is an almost infallible remedy in quinin. "When shall its use be commenced?" is a pertinent question. It would certainly seem highly desirable to check the course of the disease as soon as possible, and especially since transmission of the simple intermittents into the pernicious forms may occur if the disease be not arrested. At the present day specific treatment is often delayed in order to give full opportunity for making a blood-examination with a view to completing the diagnosis. There is no decided advantage in commencing the use of quinin during the first paroxysm, when the blood may be examined; but on finding the case to be one of malaria, quinin should be administered after the paroxysm, so as to prevent a recurrence. For like reason, if the history at the physician's first visit, combined with the symptoms presented, make the diagnosis of intermittent malaria reasonably certain, and there is no opportunity to examine the blood microscopically, the principal antiperiodic remedy should be commenced at the close of the paroxysm.

The quinin cures malaria by acting directly upon the intracorpuscular hematozoa.

During the *paroxysm* we should aim to make the patient comfortable. He is to remain in bed, is to be well covered, and external heat applied during the cold stage; and he is to be lightly covered, given cooling drinks and cold spongings during the hot stage.

During the apyrexial intervals the patient may leave his bed, provided that he feel strong enough, and, as before intimated, the specific remedy is given during the afebrile period. Certain authors recommend that the entire daily quantity be given at one dose from four to six hours before the succeeding paroxysm is expected, the object being to surcharge the blood at the time when the hematozoa sporulate. Others give the remedy in divided portions, administering the last dose from four to six hours before the next paroxysm is due. My own experience leads me to favor the divided doses rather than the single large ones. The total amount per day required to cut short the intermittents is from 16 to 20 grains (1.036 to 1.296) in most temperate climates. When this fails more may be given—24 to 30 grains (1.555–1.944). My own practice has been to administer immediately after the close of the sweating stage gr. iv or v (0.259 or 0.324), repeating the same dose a few hours later, and the remaining 8 or 10 grains (0.518 or 0.648) (or one-half the daily dose) six hours before the time for the next paroxysm. I have thus escaped the slight toxic symptoms (tinnitus, deafness, nausea, etc.) which are apt to follow single large doses. The remedy is best given in capsules, followed by a few drops of dilute hydrochloric acid, with a view to dissolving the quinin in the stomach. After the attacks cease to recur quinin should be continued in amounts of 6 to 8 grains (0.388 to 0.518) daily for several days. If quinin cannot be taken *per os*, it may be tried by enema or by suppositories in appropriately large doses. In very young subjects I have for a long time administered quinin by suppository.

The physiologic effects of the drug can be quickly obtained by administering it hypodermically. Hence, if there be no time for absorption from the stomach (four hours being the shortest period it is safe to allow), the drug should be thus employed. For this purpose the more soluble salts (hydrobromate, etc.) of quinin are to be preferred to the ordinary and more insoluble sulphate, which requires the addition of a mineral acid.

Many preparations of cinchona other than the salts of quinin may be tried, and among these cinchonin administered in the same manner as the latter is the best substitute. Some contend that the sulphate of quinidin has antiperiodic power, almost equal to quinin. In prolonged cases the salts of quinin and other preparations of cinchona sometimes lose their specific influence, and arsenic is then to be employed, either alone or in combination with the former agents. The dose of the arsenic, beginning with ♏iv (0.266) t. i. d. of Fowler's solution, must be increased until its physiologic effects are produced. Arsenious acid often does even better service than Fowler's solution. So soon as the disease is controlled the dose of arsenic is to be reduced. Administered as above indicated, this remedy is most efficacious in malarial cachexia and masked forms of intermittents; it may be combined with iron and quinin. In cases of malaria that are resistant to quinin (often the quotidian forms), methylene-blue has been found extremely active and serviceable. Cardamates believes that it is indicated only when quinin is contraindicated, as in hemoglobinuria or in pregnancy when abortion is feared. While in charge of the out-patient service of the Episcopal Hospital, Philadelphia, I employed in chronic malarial cachexia, with very satisfactory results, the sulphate of cinchonidin in daily doses of gr. xxx–xl (1.944–2.592). In this class of cases Warburg's tincture (℥ss (16.0) three times a day) has been warmly recommended.

2. **The Treatment of Pernicious Intermittents.**—(*a*) *Prophylaxis.*—By treating all ordinary intermittents actively after the first paroxysms the

occurrence of pernicious forms can be obviated. Not to pursue this course in seasons and in localities in which these serious types are known to prevail, is next to criminal.

(*b*) *The first pernicious attack* must be treated immediately, and there is not a moment to be lost. Hence in all varieties of pernicious intermittents quinin should be administered hypodermically until the patient is fully cinchonized—a condition that must then be maintained for several days. In all varieties stimulants are to be used freely if the heart's action becomes feeble, and the patient is to be well nourished throughout. There are other details, though of relatively minor importance, and they vary with the individual forms. Thus in "congestive chills" external warmth is useful, and morphin combined with atropin should be given hypodermically, this combination tending to allay gastro-intestinal symptoms as well as to warm the extremities, and meeting really important indications. Rectal feeding must be resorted to should the stomach refuse to retain nourishment. In the *comatose form* the nervous symptoms are most successfully combated by prompt and energetic antiperiodic treatment, together with vigorous stimulation and feeding, since they are due to the intensity of the infectious process.

(*c*) During the apyrexial period every effort must be made to prevent a recurrence of the paroxysm, and to this end the patient must be kept fully cinchonized until the time for the next paroxysm is over.

3. **Treatment of Remittent Fever.**—The mode of treatment in this form differs somewhat from that appropriate for intermittents. At the onset a mild mercurial is advantageous (calomel gr. ¼ (0.0162) every hour for three doses), followed by a saline laxative (Rochelle salts, ʒij; 8.0). During the febrile exacerbations cool spongings of the body, coupled with the use of the ice-cap, are serviceable. The gastric symptoms demand chipped ice by the mouth or small doses of cocain, and a mustard plaster externally. Immediately after the first remission sets in quinin must be exhibited, and large doses are now indicated (gr. xv (0.972), to be repeated at 8 or 9 A. M.). A third and even a fourth dose of the same size may be required. The exacerbations of fever generally yield to this remedy, but if, as rarely happens, they do not, then small doses of pilocarpin (gr. ⅛ to ⅙; 0.008 to 0.010) may be administered hypodermically during the height of the fever. This causes free sweating in many instances, and in consequence renders the remission more marked and more prolonged; thus, in short, rendering the course of the affection speedily favorable. The heart must be guarded.

A case that has been allowed to run on for one, two, or more weeks is often greatly benefited by the use of Warburg's tincture, as before recommended, for several days, when quinin may be re-employed. The patient, especially if the case be protracted, must be vigorously fed, and *per rectum* if it cannot be accomplished by the mouth. In typical cases, which are promptly controlled by quinin, stimulants are rarely needed, or at least not until the convalescent stage is arrived at. In severe and neglected cases the indications for their employment may be presented early, and they should then be given, the physician conforming to the same rules as in typhoid and other acute infectious diseases. The renal congestion and anuria are to be met by internal diaphoretics (pilocarpin, etc.) and by saline laxatives. Most efficacious, perhaps, is a combined hot-water and steam bath. The patient is placed in hot water, and then a blanket is put around the neck, its free ends being allowed to extend over the edges of the tub.

4. **Treatment of Malarial Hematuria.**—As intimated above, in hemorrhagic pernicious malaria quinin is absolutely indicated, if there be no uremic features. The use of quinin in moderate doses (gr. xvj—1.036—daily) successfully relieves the hemoglobinuria occurring in connection with mild forms of malaria, and its subsequent use in smaller doses (gr. iij (0.194) to gr. vj (0.388) daily) will prevent a recurrence. It is claimed by some writers that quinin may produce hematuria (Plehn, Richardson, and others), and also that this remedy is of no value in combating this symptom. The specific remedy should not be abandoned, however, and large quantities, such as might act as an irritant to the renal tissues, are not necessary to effect a cure, except in pernicious forms. In the latter, methylene-blue deserves a trial.

DYSENTERY.

Definition.—An infectious inflammatory disease of the large intestine, characterized anatomically by ulceration of the intestinal mucosa, and clinically by frequent mucous and bloody discharges, tenesmus, fever and prostration becoming profound, a tendency to abscess-formation in the portal system, to paralysis, and, finally, to pronounced anemia. It is a truly epidemic disease, yet it also occurs constantly in endemic form, and particularly does this occur in temperate climates.

There are three distinct varieties: (*a*) Catarrhal, (*b*) Amebic, and (*c*) Diphtheritic. I shall also describe briefly chronic dysentery, which is the sequel of the acute forms.

Historic Note.—Few diseases have been longer known than dysentery, of which we have a description by Hippocrates. Galen localized the chief seat of the affection in the colon, and in 1626, Sennertus defined its sporadic and epidemic character and some of its leading clinical features. To Morgagni belongs the credit of having made the first postmortem anatomic study of the disease. Further and more accurate pathologic contributions were made in the earlier part of the present century by Cruveilhier and Rokitansky, and, more recently still, the whole subject of the morbid anatomy of this disease has been carefully investigated by Virchow, whose results have settled most of the questions connected with the subject. In the United States dysentery has prevailed epidemically upward of a century, the time of greatest prevalence in different districts having been about the middle part of the present century (1847–55). Woodward has given us the only complete record of the various outbreaks in this country, and an account of the ravages of dysentery in both armies during the War of the Rebellion is given in his *Report*, which records 259,071 cases of acute and 28,451 of chronic dysentery. The disease is far less frequent than formerly, owing to the advance made in recent times in sanitary science.

Etiology.—A few general considerations, having reference to the causation of the different forms in common, may be adduced here, and, as each variety presents different anatomic lesions in leading particulars, their pathology will be considered separately.

Among disposing factors, *season* heads the list, dysentery being most common in the summer and autumn; great and sudden changes of temperature are more potent than equal changes in humidity. *Climate* has a marked effect, and high temperature must be regarded as a powerful agency, since the disease is much more prevalent in warm than in cold climates, though it is met with in epidemic form as far north as Norway. *Malarial districts* suffer more than non-malarial. *Unhygienic conditions*, as shown by the local epidemic outbreaks in armies, jails, barracks, institutions, etc., predispose to the affection.

Among factors connected with the individual are (*a*) *Catarrhal* conditions of the intestinal tract, particularly if the latter be caused by unripe fruit or other unwholesome forms of food; (*b*) *Age.* Although no age enjoys immunity against dysentery, most cases are met with in adults under thirty-five years. *Sex* and *race* are probably without appreciable influence.

CATARRHAL DYSENTERY.

Pathology.—There are two forms: (*a*) In this the solitary follicles are affected chiefly, and are the seat of hyperplasia, followed by necrosis, with the formation of small ulcers. This is common in children. (*b*) Here a purulent inflammation of the entire mucosa, with more or less erosion of the surface and superficial ulceration, exists. In both forms the lesions are mainly confined to the large intestine, though the ileum is sometimes implicated to a lesser extent.

Special Etiology.—The specific bacillus of catarrhal dysentery is not known, but it will probably be shown to be the bacillus coli communis, which may become pathogenic when the state of the mucosa of the bowel is altered by sudden changes of temperature, etc. (Arnaud, Maurel). Curtis in a recent outbreak found the bacillus pyocyaneus in every specimen of water used and in the intestinal discharges.

Clinical History.—There may be *prodromes*, lasting one or two days, which take the form of a mild gastro-intestinal disorder (anorexia, slight pains in the abdomen, followed by diarrhea).

The *characteristic symptoms* are mild colicky pains in the abdomen, followed by discharges from the bowel, which at first number from three to six daily. Soon they become frequent and are accompanied by straining and tenesmus, and now their number ranges from ten to no less than one hundred or more per day. Indeed, the desire to go to stool may be almost constant, and the rectum is the seat of intense burning sensations during and after each evacuation of the bowel. The character of the discharges varies with the different periods of the affection. During the first thirty-six or forty-eight hours they are feculent (sometimes scybalous masses), rather copious, and intermingled with some mucus and blood. For the next four or five days the stools are scanty, measuring from 2 drams (8.0) to ½ ounce (16.0), and are made up of a sero-mucous fluid or of a muco-purulent material with blood. The chief constituents of the stools are mucus, blood, and pus, any one of which may preponderate, thus giving rise to mucous (most frequently), purulent, or bloody stools.

Microscopic examination of the usually glairy stools shows red blood-corpuscles, numerous leukocytes, generally large, oval or round epithelioid cells containing fat-globules, vacuoles, and bacteria (especially those of putrefaction). Occasionally the *Cercomonas intestinalis* is seen (Osler).

A few shreds (portions of necrosed mucous membrane) may appear from time to time in the discharges, and particularly in severe forms of the affection. These usually increase in number at the close of the first week, and a little later the discharges become less frequent and the amount of mucus and blood diminishes. The stools are now of a greasy brown or dark-green appearance, fecal matter reappearing in them, and soon they are again fully formed.

Other Symptoms Referable to the Alimentary Tract.—The tongue has a greasy coating—moist at first, dry later—and at last may become red and glazed. Anorexia is present, with excessive thirst, and vomiting may rarely occur. A distressing though uncommon symptom is hiccough. There will usually be tenderness over the line of the colon, but there is an absence of tympanites, and the abdomen is apt to be flat and somewhat tense.

The *general symptoms* are well marked in the severer types. The patient is much debilitated, sometimes even collapsed, as shown by the small, frequent pulse, cool skin-surface, the rapid wasting, and weak, hoarse voice. The temperature is not much elevated, though it may touch 103° or 104° F. (40° C.) at the outset, and the curve is an irregularly remittent one.

Diagnosis.—This can easily be made upon the intestinal features and from the character of the stools—frequent, small, slimy (or bloody) discharges, accompanied by distressing tenesmus.

Differential Diagnosis.—Symptoms simulating dysentery may appear in the course of certain rectal affections, such as *strangulated hemorrhoids*, *syphilis*, and *epithelioma*. In these conditions, however, there is a different history and the symptoms of proctitis are usually less acute, while a physical examination of the rectum will settle the diagnosis in doubtful cases.

Prognosis.—The *duration* of mild cases is from eight to ten days, and in severe types from three to four weeks. The *prognosis* varies in different epidemics or according to the type of the affection; but commonly this is not aggravated and recovery is generally to be expected. Occasionally, however, the disease is threatening to life. Besides the systemic prostration and collapse above referred to, serious nervous symptoms (great restlessness, delirium followed by coma) may develop and cause a fatal termination. When death occurs it is usually due to exhaustion, and is seen particularly in persons previously enfeebled or in the very young and the aged. Complications influencing the prognosis are exceptional (peritonitis and liver-abscess).

AMEBIC DYSENTERY (TROPICAL DYSENTERY).

Bacteriology.—This form of dysentery is caused by the *amœba coli* or the *amœba dysenteriæ* (Councilman and Lafleur). The amœba dysenteriæ is a unicellular, motile organism, in size 3 to 7 times the diameter of a red blood-corpuscle (15 to 30 micromillimeters). Its protoplasm consists of two zones—an outer colorless (ectosarc) and an inner granular zone (endosarc), with a visible nucleus and one or more vacuoles. This micro-organism was first described by Lambl (1859), but it remained for Lösch, and especially Kartulis, to show its close association with dysentery. The ameba (*amœba coli mitis*) is occasionally found in healthy individuals, and also in other bowel-affections than dysentery (mucous enteritis, simple diarrhea, proctitis due to engorgement), and two species are recognized—a virulent and a benign form (Quincke and Roos). The ameba is found not only in the discharges, but also in the pus from the secondary liver-abscesses. Flexner[1] affirms that bacterial association probably has much influence on the pathogenic powers of the amebæ.

Hehir has found the *bacillus dysentericus* associated with the *amœba coli*, and considers it to be pathogenic. The principal causative *role* in the production of this form of dysentery has been ascribed to the pyogenic cocci by Tancarol, and more recently by Ascher, Silvestri, and by Bertrand and Baucher.

The mode of transference of the ameba is not definitely known, though the principal source of the dysenteric germs is most probably the drinking-water. The poison is feebly communicable by contact.

Amebic dysentery is not confined to the tropics, but is met with also, though less frequently, throughout Europe and North America.

Pathology.—The lesions are almost always situated in the large intestine, although rarely the ileum is also invaded. The first visible change is a hyperemia of the mucosa, most marked in the descending colon and rectum; but the changes which produce the characteristic dysenteric ulcer begin with infiltration and swelling of the submucosa, followed by necrosis, which involves the overlying mucosa with its epithelium (Kruse and Pasquale). How the amebæ reach the submucosa has not yet been observed. The visible infiltration occurs usually in circumscribed areas which are oval or hemispheric in shape, and project above the level of the surrounding mucosa. The submucosa presents a grayish-yellow appearance, and is soon thrown off in the form of a slough.

The ulcers take various shapes—chiefly irregular, and less frequently round or oval. Their edges are ragged and undermined, and the floor, which is more or less covered with pultaceous material, is rough or crater-like, and formed by the musculature or the outer serous coat of the intestine. From the manner in which the ulcers are formed it is obvious that cellular infiltration (followed by necrosis) may occupy the submucosa for a greater or less distance beyond the borders of the ulcers. In this way fistulous channels may be produced beneath the mucosa and connect two or more ulcers. Usually this ulcerative process affects only certain portions of the large gut, especially the flexures—hepatic and sigmoid—and the rectum; but it may be general, and I have seen an instance of this kind. Similar cases are not uncommon in which the ulcers are so numerous as to include almost the entire mucosa of the large intestine.

Healing is attended with the development of fibrous tissue along the edges and in the base of the ulcer, and secondary contraction of this new connective tissue is often productive of colonic stricture, which is usually either partial or irregular. The cases that come to autopsy often show diphtheritic inflammation as a secondary or terminal condition.

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[1] *Jour. Amer. Med. Assoc.*, Jan. 5, 1901.

The *microscope* reveals proliferation of the fixed connective-tissue cells, and the presence of amebæ in the walls and the base of the ulcers, in the lymph-spaces, and rarely in the blood-vessels. Pus can only occasionally be detected.

The **liver** may be the seat of prominent lesions. These are (*a*) *abscesses*, which may be single or multiple, the latter being small, and the former often large. The single or solitary abscess is usually situated near either the upper convex or the lower concave surface, while the abscess-cavity is formed in a manner similar to the intestinal ulcers. The area affected is at first infiltrated; it then becomes necrotic, and finally more or less liquefied. Upon the full development of the first stage the part invaded is a grayish-yellow pultaceous mass, but in the second or necrotic stage the abscess contains a yellowish or greenish-yellow, spongy material with beginning liquefaction. The contents of the mature abscess consist of a greenish- or reddish-yellow purulent material and of remnants of liver-tissue. The walls of the recent abscess are irregular and ragged, those of an old abscess being dense and fibrous, and a section of the abscess-wall shows an inner necrotic zone, a middle zone (in which there is great proliferation of the connective-tissue cells, compression and atrophy of the liver-cells), and an outer zone of intense hyperemia (Osler). The contents of the abscess show either few or many amebæ, and only rarely pus. When pus is present it is due to a secondary infection by the pyogenic germs. In what way the amebæ gain access to the liver is not definitely known, but it is probable that in multiple abscesses they are propagated along the blood-current, either from the ulcers or from a single primary focus. Cultures are generally sterile.

(*b*) The parenchyma of the liver may be the seat of numerous circumscribed necrotic spots, which are supposed to be due to the action of the chemical secretions of the amebæ.

The **lungs** sometimes show changes similar to those in the liver; they are the result of direct extension of the hepatic abscess through the diaphragm into the lower lobe of the right lung.

Clinical History.—The mode of *onset* is *gradual* except in a small proportion of the cases, in which it is sudden with well-marked symptoms. When, as generally happens, it is insidious, the initial symptom is often a trivial diarrhea. The affection is then characterized principally by *intermissions* and an exacerbating *diarrhea*, the liquid stools containing necrotic tissue of a grayish-brown and sometimes yellowish-gray color. The latter are often bloody and mucoid, particularly at the outset, and in fully developed cases are fluid. The *number* of discharges per day is exceedingly variable in different epidemics, and even in individual cases, though in most instances they range from six to eight or ten daily.

Microscopic examination of the feces during the exacerbations discloses amebæ that are almost invariably endowed with motion, though usually not when the stools have become formed. Tenesmus is not a prominent feature in most cases, and may be entirely absent. Colicky abdominal pains are rare, and nausea and vomiting are equally so.

General Symptoms.—Fever is usually present, but it is slight and exhibits marked variations. In certain instances, however, the temperature is below the normal curve throughout the entire or greater part of the course. From the time of onset there is gradual though progressive loss of flesh and strength, and anemia usually becomes well marked.

Complications.—The most common is *hepatic abscess*, and secondary to the latter may arise abscess of the right lung. Authors are not agreed as to the frequency of occurrence of liver-abscess[1] in amebic dysentery, but it is certainly comparatively rare in this country, not exceeding, perhaps, 3 per cent. of the cases. *Peritonitis* may result from perforation of a dysenteric ulcer, causing death. The point of perforation may, however, be in the rectum, when *periproctitis* is the result; or it may be in the cecum, when *perityphlitis* is the sequel. In tropical or subtropical countries the disease is often complicated with malarial affections, and in malarial regions intermittent and remittent fevers are among the commoner complications. The presence of an intermittent fever is not, however, sufficient to warrant the assumption that malaria complicates dysentery; and in order to show the latter combination we must be able to demonstrate the presence of the *plasmodium malariæ.* In pyemia and in suppurative processes generally—conditions sometimes met with in dysentery—the temperature-curve is often distinctly intermittent. *Typhoid fever* is a rare complication. The latter disease cannot be said to coexist with dysentery unless all of the characteristic symptoms are present. Certain cases of dysentery are characterized by the development of the *typhoid state*, and pyemia and septico-pyemia may appear late. Among special manifestations of the latter are pylephlebitis, pericarditis, endocarditis, pleuritis, and rheumatoid pains in the joints.

Diagnosis.—The slow course, marked by intermissions and exacerbations of the bloody fluid stools, the mild general symptoms, apart from emaciation and debility, are salient features, but an assured recognition of the affection demands a microscopic examination of the stools. Cases have been recorded by Councilman and Lafleur in which the diagnosis rested upon amebæ being found in the sputa, the latter being complicated with pulmonary and hepatic abscesses which discharged through a bronchus, while the intestinal symptoms were negative.

Prognosis.—The prognosis is graver than in the catarrhal variety, and the mortality-rate in certain epidemics has been frightful, particularly among soldiers in the field (amounting to 70 or even 80 per cent.). In sporadic cases the danger to life is less, the mortality-rate in temperate climates being not over 5 or 6 per cent. The complications which render the prognosis unfavorable are various (peritonitis, hepatic and pulmonary abscess, pyemia secondary to the latter, broncho-pneumonia, malaria); death may be due to hemorrhage or peritonitis, but in a preponderating proportion of the cases to asthenia. A dangerous degree of debility is indicated by great nervous depression; a cool, clammy skin; a sunken, pinched facies; a dry tongue; a feeble, rapid pulse; and by restlessness, alternating with marked apathy or low muttering delirium.

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[1] For the diagnosis of this condition the reader is referred to the section on Hepatic Abscess in the article on Diseases of the Liver.

Course and Duration.—The average duration ranges from eight to ten weeks in uncomplicated cases; the disease can, however, be cut short by appropriate treatment. It manifests an innate tendency to pursue a chronic course, interrupted by frequent exacerbations or true relapses, and convalescence occupies a long period of time in consequence of the profound anemia and debility that supervene.

PRIMARY DIPHTHERITIC DYSENTERY.

Definition.—An intestinal inflammation (usually colonic), accompanied by a croupous, or true, diphtheritic exudation. It is the epidemic dysentery of Japan, but prevails wherever large aggregations of persons occur, as in armies, asylums for the insane, ships, and the like.

Pathology.—In mild grades a grayish-yellow, croupous exudate appears upon the inflamed mucosa, with a necrosis of the epithelial layer that is often limited to the top surface of the folds of the colon. In other instances the diphtheritic infiltration involves all the layers of the bowel, which now becomes greatly enlarged, its mucous membrane being converted into a yellowish-brown, thick, elastic mass, sometimes extending along the entire length of the large intestine. The changes may be confined to the circumscribed areas (flexures of the colon and rectum), and thick sloughs may be cast off, leaving behind ulcers of corresponding size and depth.

Bacteriology.—The distinctive pathogenic agent is the *bacillus dysentericus*, discovered by Shiga[1] during his investigations into Japanese dysentery. Flexner, in his studies of the dysentery of the Philippine Islands, found the same organism. This bacillus and the bacillus typhosus are almost identical, although slight and decisive criteria of difference are observable by the bacteriologist. The Shiga bacillus "shows less marked motility when first isolated and a tendency rapidly to lose motility in the artificial cultivations; it displays a more uniform generation of indol; after brief preliminary acid-production in milk there follows a gradually increasing alkalinization; it is inactive to blood-serum from typhoid cases, but reacts with serum from dysenteric cases to which bacillus typhosus does not respond" (Flexner).

Clinical History.—The affection usually has an *acute onset*, and one characterized by an appearance simultaneously of severe local and general symptoms. There may be an initial *chill*, and there is *fever*, which rises rapidly, together with a marked and early appearing prostration and delirium. Severe abdominal *pains* are complained of, and the discharges may be numerous, containing shreds and large sloughs, or even tubular pieces, of false membrane. When these elements are present in the stools the latter are of a dark-brown color, emitting a fetid odor, and generally containing more or less blood and mucus. *Tenesmus* may be intense.

The **physical signs** are often prominent. The belly in most instances is greatly distended, and on pressure very tender—signs due to the fact that the lesions are situated chiefly in the large bowel, and not, as a rule, to peritonitis.

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[1] *Centralbl. f. Bakt. u. Parasitenk.*, 1898, xxiv., Nos. 22–24.

The **diagnosis** rests upon the intestinal symptoms and the character of the dejections, associated with a grave general condition suddenly developed. All doubt is removed by the appearance of the cases in an epidemic form.

Complications.—These are both numerous and varied, and include metastatic abscess of the liver; perforation of the gut followed by peritonitis either localized or generalized (according to its seat); also pleurisy, endocarditis, and pericarditis.

The **prognosis** is almost wholly unfavorable. Occasionally recovery follows, though more frequently the disease takes on a chronic course.

SECONDARY DIPHTHERITIC DYSENTERY.

Here the lesions are similar in kind to those of the primary form, but in the majority of instances of a less intense grade. Rarely they may be both extensive and severe. This variety is met with as a terminal condition in not a few acute and chronic diseases; among the former it is with great relative frequency seen to develop in pneumonia (Bristowe), and less frequently, though in not rare instances, in typhoid fever, according to my own observation. Among chronic affections, upon which this condition may become engrafted, are nephritis, organic disease of the heart, and pulmonary tuberculosis.

Clinical History.—No characteristic symptoms attend upon its invasion. There may be slight diarrhea—two to four liquid stools daily—but it is not often accompanied by tormina and tenesmus, and the discharges rarely contain any noticeable amount of blood or mucus. Very rarely shreds of pseudomembrane are passed with the stools. Secondary diphtheritic dysentery often induces fatal asthenia.

Sequelæ of Dysentery.—In all forms a relapse is likely to occur, each attack increasing the liability of the patient to subsequent ones. Moreover, in persons who have recovered from acute dysentery we often observe a disordered digestion and irritability of the bowels. Rarely, chronic nephritis follows dysentery. The most interesting sequel, however, is paralysis, which occurs mainly in the form of paraplegia (S. Weir Mitchell). Stricture of the bowel is a rare sequel.

Treatment.—**Prophylaxis.**—This embraces isolation and a thorough disinfection of the discharges, which contain the specific germ of the disease, as soon as passed. The drinking-water during the epidemic prevalence of dysentery should be thoroughly boiled, and healthy persons should avoid the use of improper food, while an unhygienic environment (overcrowding, etc.) is to be corrected as far as possible. All sufferers from dysentery must be kept in bed, and should occupy a well-aired apartment.

The **diet** should consist of milk whey and light animal broths during the period of active intestinal symptoms. The blandest articles only are either acceptable to the stomach or allowable in the diphtheritic variety, as egg-white, zoolak, and good beef-extract in small portions.

In the amebic form of the disease it is well to allow easily digestible solids, as raw oysters, eggs, well-boiled rice, fowl, fish, and the like, in small quantities. During convalescence a return to the usual dietary is gradually to be made.

Alcoholic Stimulants.—With the development of asthenia and cardiac failure stimulants must be employed, as in other acute infectious diseases. Diphtheritic dysentery calls from the very outset for free stimulation. The diffusible stimulants (*e. g.*, champagne) are often invaluable. Strychnin may be required also in cases of extreme debility.

Medicinal Treatment.—If scybalous masses be passing still, the treatment should be commenced by administering a dose of castor oil or a saline purge, and this may be repeated if necessary. It is well to convert dysentery into diarrhea, thus cleansing the bowel thoroughly. Measures tending to deplete the mucosa of the intestine and at the same time inhibit undue peristalsis are most effective, as magnesium sulphate. In the later stages purgatives are attended with baneful effects.

Ipecacuanha has long been, and still is, regarded as possessing a specific influence in cases of dysentery. Its administration is usually preceded by a dose of opium (laudanum or morphin) which is given when the stomach has been empty for a few hours. Most authors recommend that large doses—gr. xx to ʒj (1.29 to 4.0)—should be administered; but it is probable that a small dose—gr. ⅙ to ¼ (0.010 to 0.016) every half hour—is quite as effective; and in children the smaller doses are to be preferred and will be found to be quite efficacious. Other remedies should also be employed, and among these opium is particularly beneficial in combination with ipecacuanha or in the form of Dover's powder, which contains both agencies. Three chief symptomatic indications are met by the opium—pain, restlessness, and undue peristalsis —and to obtain the best effects from the opiate it should be administered in the form of morphin hypodermically. In cases in which tenesmus is an unusually distressing feature an opium suppository (gr. ij—0.1296) or laudanum (♏xxx—2.0, by enema) exercises a beneficial effect. Bismuth in full doses is useful (ʒss–j—2.0–4.0 every two hours), and in cases of sporadic dysentery I have frequently found the combined use of Dover powder, bismuth subnitrate, and salol of signal service.

In diphtheritic dysentery antiseptic substances by the mouth for the purpose of disinfecting the intestinal canal and favoring the healing of the ulcerated surfaces after the removal of the necrotic pseudomembrane, such as benzonaphtol (gr. xl–lx—2.592–3.788—in the twenty-four hours in divided doses), salol, opium, and silver nitrate are the remedies of choice. The naphtol preparations being insoluble should be given in capsule and the silver nitrate in pill form one hour after food. Iodoform in a pill or capsule in doses of ½ to 3 grains (.032 to .194 gm.) has been much lauded. Bose and Vedel employed in 4 cases intravenous injections of sodium chlorid, 7 : 1000 being the maximum strength. The injections should be made early, and repeated, so that they will develop sustained general reaction and a modification of the general condition which can lead to recovery. Care should be taken as to the quantity used and the rapidity with which it is injected (½ to 3 ounces each minute should not be exceeded).

Antiseptic irrigation of the bowel would be, if properly carried out, a curative measure, since by this means we may destroy the bacilli and solutions of numerous antiseptic substances and astringents have been recommended for this purpose. Unfortunately, the bowel is frequently so irritable as to seriously interfere with this mode of medication. If, on this account, large injections cannot be given, small ones should be substituted and the quantity gradually increased. Preliminary to their use we may also employ cocain in the form of a suppository, or a small quantity of a solution of cocain (4 per cent.), or a laudanum enema (♏xxx—2.0, in starch-water), after which a large injection may be tolerated if administered slowly and the flow be interrupted at brief intervals. Among the best agents are silver nitrate (gr. ss–j—0.032–0.064—ad ℥j—32.0), tannic acid (1 to 2 per cent.), salicylic acid (1 to 2 per cent.), and mercuric chlorid (1 : 6000). I have for a number of years been in the habit of employing these astringents and antiseptic solutions alternately, administering each once daily. The tannic-acid and the salicylic-acid solutions are best borne during the more active stages of the disease. The temperature of the water should, at first, range from 100° to 110° F. (37.7°–43.3° C.), and subsequently this may be slightly reduced. The patient during the administration of the enemata should assume the dorsal position or that upon the left side, but in either case with the hips well elevated, so as to aid the flow by gravitation. In amebic dysentery warm injections of quinin (strength 1 : 1000–1 : 5000) have been used with good effects by some authors, but with directly contrary effects by others.

Local means in the form of hot fomentations, light poultices, and turpentine stupes often afford much comfort. The various complications must be met by appropriate treatment, as under other circumstances.

Chronic Dysentery.

This form of the disease almost always succeeds an acute attack. Very rarely is it chronic from the start, and particularly if it be the amebic variety.

Pathology.—In most instances the large intestine is still the seat of ulceration. Some of the ulcers show no signs of healing; in others this process is going on; while in still others it is completed and puckered cicatrices are presented. The ulcers are deeply pigmented, as is the unulcerated mucosa, which often presents a slate-gray or blackish color. The submucous and muscular coats are hypertrophied, as a rule, with occasional narrowing of the lumen of the bowel, and cystic degeneration of the intestinal glands is sometimes observed. It is to be noted that in a certain, though small, percentage of the cases ulceration does not occur, the mucosa everywhere presenting an uneven, puckered aspect, due to deposits of fibrous tissue.

Symptoms and Diagnosis.—Many of the most characteristic features of the acute form are either but feebly expressed or altogether wanting. This is particularly true of the tormina and tenesmus. Certain elements found in the stools of the acute type (blood, shreds of pseudomembrane, and tissue) are also rarely present. True *dysenteric symptoms*, however, may arise during acute exacerbations, with or without pain or tenesmus; then from three or four to a dozen or more fluid *dejections* are passed daily. The latter are often frothy (when starchy articles of food are taken), being composed chiefly of fecal matter and undigested particles of food, with considerable mucus; and in severe forms blood and pus may be constantly present in the discharges. In many cases the stools are semifluid (pultaceous), and rarely they contain scybala; or the rather frequent liquid or semifluid discharges may alternate with constipation. In such instances the lesions are apt to be situated in the lowest portion of the large intestine. The *character* of the discharges is much influenced by the sort of food taken; thus when a mixed dietary is partaken of, they are thin, more frequent, and contain more undigested masses of food. *Gaseous distention* of the intestines is often an annoying symptom.

The **physical signs** are negative, save for slight tenderness along the line of the colon.

Associated symptoms referable to other organs are not without value in the diagnosis. The gastric digestion is poor, the appetite generally impaired (though variable), and the tongue is clean, red, and glazed, presenting the appearance of raw beef. There are progressive emaciation and asthenia, which eventually reach an extreme degree. The skin-surface becomes dry, harsh, and cool, the facies grim, the pulse exceedingly feeble, the mental faculties greatly weakened in the advanced stage; and, as in the acute form so in the chronic, death is usually due to asthenia—with this difference, that in the latter the end is reached more slowly. Rarely peritonitis in consequence of perforation of the bowel is the immediate cause of death.

Differential Diagnosis.—The disease is discriminated from *chronic diarrhea*, often with great difficulty. In chronic dysentery there is the history of an antecedent acute attack, with the appearance from time to time of exacerbating periods when mucus, pus, and often blood are contained in the discharges. The latter are, at the same time, more frequent and apt to be accompanied by more or less abdominal pain and tenesmus, and the presence of these features would serve to eliminate chronic diarrhea. From *tuberculous ulceration* of the intestines it is distinguished by the absence of any history of tuberculosis, family or personal, and of tuberculous new growths in other portions of the body, particularly the lungs.

The **complications** are the same as in acute dysentery, if we except the greater liability, due to the great and protracted weakness of the patient, to certain serious intervening diseases (chronic nephritis, tuberculosis, pneumonia). Ulceration of the cornea has frequently been noted.

The **duration** is long, the disease lasting for many months or even several years.

Treatment.—This should be directed mainly to the local condition, and should consist in methodic irrigation of the bowel with a view to promoting the healing of the ulcers. Formerly it was sought to accomplish the latter indication by the use of certain remedies internally, as silver nitrate, balsam of copaiba, bismuth subnitrate, etc., but the only preparation which I have found useful is the zinc oxid (gr. v–x—0.324–

0.648) three times daily. The latter preparation is decidedly palliative, sometimes even curative.

Intestinal irrigation is to be tried, and various disinfectants and astringent remedies should be alternated as advocated in the acute form. Among individual remedies the silver nitrate (gr. ss–ij—0.032–0.129—ad ℥j—32.0) every second day is doubtless the best. On intervening days antiseptic remedies may be used in solution, such as mercuric chlorid (1 : 6000) or salicylic acid (1 to 2 per cent.); and of other useful agents I may mention tannic acid, alum, acetate of lead, creolin, and quinin sulphate.

Prior to the use of any of the above-mentioned enemata the bowels should be well flushed with a large injection of tepid water, so as to remove the fecal and other irritating materials. The same details are to be observed in carrying out this mode of treatment as in the acute forms of dysentery. Gallay[1] has related the curative effects of large enemata of a solution of crystallized silver nitrate in distilled water, a scruple to a quart (1.296 per liter), to which 20 or 30 drops of laudanum have been added. Amelioration follows the third or fourth washing, but a course of sixty is recommended to secure permanent relief. I agree with the late Austin Flint that the lower part of the rectum should be examined with the speculum, and appropriate topical applications made if ulcers in this situation be discovered.

The *dietetic* treatment in chronic dysentery is of the utmost importance, and the lightest forms of albuminous foods are to be adhered to strictly, to the exclusion of vegetable substances. Milk is excellent when it can be taken. It is well to examine the stools, and if on microscopic examination curds or numerous fat-globules appear, the amount of milk should be reduced or skim-milk substituted. Other forms of food that are allowable and useful are egg-white, meat-broths or beef-juice, whey, and the like. The patient should wear flannels next the skin, so as to protect against the vicissitudes of weather, and, while open-air exercise is useful, it should be moderate. During inclement weather the patient should remain in-doors. I have known change of climate, with proper regulation of the mode of living, to be productive of rather brilliant results. Tonics and alcoholic stimulants are sometimes required to assist the appetite, digestion, and systemic strength, and among the most efficacious tonic remedies are iron, strychnin, mineral acids, and arsenic, which may be used in succession.

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[1] "Radical Cure for Chronic Dysentery of Recurrent Type," *British Med. Journal*, No. 1779, p. 276.

CHOLERA (EPIDEMIC).

(*Asiatic Cholera; Cholera Algida, etc.*)

Definition.—Cholera is an acute, infectious, epidemic disease. Its specific cause is the spirillum of Koch, and its most characteristic symptoms are copious watery dejections, painful cramps, collapse, and suppression of the excretions. In some localities it is endemic.

Historic Note.—During the Middle Ages cholera made deplorable ravages, chiefly along the belts of the Ganges, and has probably been endemic in India for centuries. Only during the present century, however, has the disease been widely known in Europe and America, and when it has appeared it has always been in the epidemic form. The march of epidemics has been from east to west, and always along the lines of commerce and travel by land or sea, sometimes spreading over the entire globe. While interesting, it would not be profitable to the student to detail here the progress of the various epidemics of cholera in Europe and America. It will suffice to state the years in which the chief of these occurred: in 1831–32, in 1835–36, in 1847–49, being brought by immigrant ships from Europe; in 1852 in Europe (touching our shores in 1854 and prevailing extensively); in 1859 (Europe), in 1866–67 (mild outbreaks in America), in 1869–73 (America in 1873), in 1884 (in Europe), and in 1892–93 (abroad). It is seen that there have been no epidemic visitations in America since 1873, though a few small groups of cases have on several occasions been brought to our shores.

Pathology.—The body is generally much emaciated, the features sharp and drawn, and the skin of the dependent parts presents a mottled appearance. A post-mortem rise of temperature often occurs. The tissues are dry, owing to the draining of the liquids of the body, and hence putrefaction is delayed. The kidneys, liver, and heart, as well as other organs in a less degree, show excessive cloudy swelling and often considerable fatty degeneration of the parenchymatous tissues. Rigor mortis comes on directly after death, is persistent, and the muscles often contract so as to cause the body to assume various uncommon positions.

The Visceral Lesions.—The chief of these are confined to the intestinal canal, and depend greatly upon the period of the disease at which death occurs. In the early stage the serosa of the small bowel is congested, presenting a roseate hue. The muscularis is relaxed. The mucosa is the seat of catarrh, being deeply injected, swollen, at times edematous, and often coated in the early stage with more or less tough mucus. Shortly the coils of intestine are filled with an almost transparent or slightly turbid liquid ("rice water"), and, occasionally a small amount of clotted blood is seen in the bowel. The solitary follicles and Peyer's patches are at first swollen, and may later, in rare instances, become ulcerated. Denudation of the epithelial lining—most probably a post-mortem change—is the rule, and large or small ecchymotic spots are visible in the intestinal mucosa. If the patient has died late in the disease (stage of reaction), patches of false membrane (diphtheritic), sometimes dark-brown in color and fetid, may be found anywhere along the intestinal canal, though chiefly in the large bowel; and this secondary croupous-diphtheritic process may attack other mucous surfaces (bile-ducts, vagina, etc.). The bacilli are observed in the mucous membrane of the intestine and in the dejections.

The stomach shows changes similar in character to those found in the intestines. At first the mucosa is congested; then, as the result of transudation, it becomes filled with "rice-water" material. Soon the hyperemic mucosa becomes swollen and ecchymoses appear. At last the organ is empty and collapsed.

The esophagus also exhibits about the same changes, though with an absence of the characteristic transudation.

The spleen, contrary to its condition in other infectious diseases, is small as a rule, though if death occur late it may show some degree of enlargement with softening.

The liver presents marked passive hyperemia and cloudy swelling, with minute spots of beginning fatty change. Desquamation of the epithelium of the cystic mucosa may occur and lead to a blocking of the bile-ducts.

The kidneys show important lesions, being enlarged from passive congestion, especially the cortex, and the capsule being somewhat adherent. They exhibit cloudy swelling and decided coagulation-necrosis. Desquamation of the epithelium in the uriniferous tubules is extensive. Microscopically, the histologic changes are those of acute nephritis in the cases in which death takes place in the advanced stage.

The bladder-changes differ in no way from those of other mucous membranes. Its mucosa is congested, ecchymotic, and sometimes the seat of diphtheritic deposit. The ureters and the pelves of the kidneys may also present identical appearances.

The Circulatory System.—The pericardium is dry, the parietal layer being covered with an adhesive secretion, while the visceral layer is the seat of more or less ecchymosis. The *heart* is dry and anemic-looking. The left ventricle is contracted, while the right is often distended with blood and soft clots, the latter sometimes extending to the pulmonary artery and the superior and inferior venæ cavæ. Outside of the heart the veins, including the cerebral sinuses, contain most of the blood. The latter is thicker than normal, and its color darker, resembling "the juice of huckleberries;" its specific gravity, albumin, and corpuscles are all increased, while its saline constituents and coagulability are decreased.

Respiratory Organs.—The larynx, trachea, and bronchi are hyperemic, and at first covered with tenacious mucus; later they may present ecchymoses and diphtheritic processes.

When death occurs before the stage of reaction the lungs are bloodless and collapsed, and the mouth of the pulmonary artery may be distended with blood. If life is prolonged until the third stage, the lungs may show congestion and edema (particularly at the bases) or pulmonary infarction. The post-mortem of a case in this stage, and especially during convalescence, may exhibit the lesions of broncho- or lobar pneumonia.

The brain and its membranes may be the seat of hyperemia, except when death takes place at a late period, and then the brain-substance may be more or less bloodless and edematous.

Etiology.—The causes are (*a*) *specific* and (*b*) *predisposing*.

(*a*) The **specific** cause is the comma bacillus of Koch, which is found in the intestinal canal of persons ill of cholera. Recent investigations into the bacteriology of the affection show that almost uniformly the cholera spirillum is associated with certain bacteria, most commonly the bacillus coli communis. It has also been shown pretty clearly that true cholera is a *nitrite*-poisoning, the result of the growth of the specific spirillum. The comma spirillum is not found in any other disease. Its form is that of a slightly curved rod, and its length about half that of the tubercle bacillus, but it is thicker and sometimes has the form of the letter S (Fig. 11). It is to be classed as a spirocheta, and has been grown successfully on media of various sorts and equally successfully inoculated upon inferior animals.

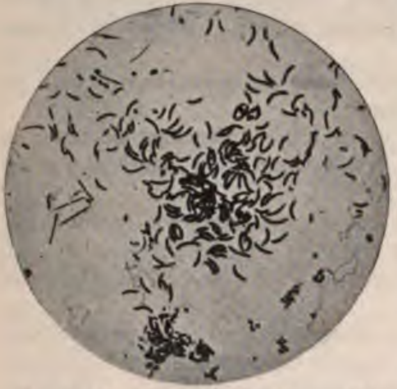

FIG. 11.—Comma bacilli (from the mouth); × 1000 (Günther).

The organism is found in a variety of positions—in the intestine, the dejecta (even quite early), and in great profusion in the pathognomonic rice-water stools. Kemp in his review has shown that the comma bacillus is often absent from the evacuations, and that in these cases the bacterium coli is usually present and sometimes streptococci. He believes, however, that the apparent absence is due to faulty technique. To find it in the vomitus, however, is rare. On the other hand, it may be seen in the stools of well persons during epidemics, displaying virulent properties.

Cholera spirilla have been repeatedly found in the outer world, and almost invariably in water. C. Fränkel during the European epidemic of 1892 studied them in flowing water, and in other epidemic outbreaks they have been found in the water used for drinking-purposes.

(*b*) **Predisposing Causes.**—(1) **Locality.**—Near to the sea-coast cholera is more common than in the inland districts or towns, and the frequency of occurrence lessens with increasing altitude, this fact possibly being due to a gradual decrease in soil humidity and porosity.

(2) **Atmospheric Temperature.**—The spirillum of cholera can only flourish in a warm temperature or in a warm climate; hence the disease is *endemic* in certain tropical and subtropical climates only; and hence we see in temperate latitudes the *epidemic prevalence* of the disease only, and that during the warm season.

(3) **Seasons.**—From what has been stated it may be seen that cholera can have no permanent home except in very warm climates in which all the other essential conditions prevail. For equally obvious reasons it is more common in the warm than in the cold months, most epidemics, both in Europe and America, having occurred toward the close of summer and in the early autumn.

(4) **Age,** as a rule, has no decided effect. It should be stated, however, that old people are very prone to the affection. **Sex** is without perceptible influence.

(5) **Debilitating Causes.**—Whenever the private conditions correspond to rigid scientific requirements during epidemic outbreaks cholera becomes less prevalent and also less virulent. On the other hand, the deplorable state of municipal sanitation, individual disregard of proper hygienic rules, nervous depression, intemperance, overcrowding, etc. all predispose markedly to the disease.

(6) Mere attacks of **intestinal disorder** due to improper diet, cold, etc. are potent, and are the sole agencies by means of which the disease is disseminated.

Modes of Infection.—The spirilla leave the body with the stools, but the most frequent bearer of cholera-poison is the drinking-water. Naturally, the individual susceptibility varies greatly (many persons being even insusceptible), and yet the degree of contamination of the drinking-water and the virulence of epidemics are almost strictly proportionate. As an illustration, Vienna had enjoyed exemption from cholera for nineteen years—a fact attributed to the excellent quality of the drinking-water and to hygienic improvements. In the same city the mortality-rate in the more recent epidemics has been small (7 per 1000) for a like reason. On the other hand, in 1872 there occurred in a single commune (Hamburg), which had a polluted water-supply (the Elbe) and no filtration plant, 17,862 cases, with the enormous death-rate of 42.3 per cent. Biernacki demonstrated the presence of spirilla in the spring-water of a house in which 13 cases of cholera occurred.

The *choleraic poison* may be conveyed with the water used for washing, cooking, and other purposes to other fluids imbibed by man (beer, milk, tea), and also to food-stuffs taken by him (lettuce, cresses, and other raw vegetables, fruits, meats, bread, butter). The organisms live and maintain their virulence on these articles of food from four to seven days at least. The infection may reach the esophagus with the water used for washing the mouth or teeth, or that used for washing the utensils, dishes, food-receptacles, etc. Again, the hands, commonly those of laundresses and nurses, may become soiled in the careless handling of bed-linen or garments worn by cholera patients or the stools, and convey the poison to the mouth or lips, to be carried into the stomach along with the drink or food. Flies may transfer the infectious element to food-articles (Simmonds and others).

Cholera is not contagious from mere contact with those ill of the disease. The disease is not acquired by inhalation (Shakespeare), and, since desiccation rapidly kills the spirillum, there is little probability that the latter is wafted by the wind-currents or is air-borne. Nor is there any clinical evidence to show that the poison may enter the system through the skin-surface. Probably the germs are *swallowed*, and the acid gastric juice may then destroy them if the size of the dose of the poison is not too large, or a sufficient number may pass into the intestinal canal and there manifest pathogenic powers. It is to be borne in mind that after the spirillum reaches the intestine, whether or not an attack of cholera is the result depends both upon the size of the poisonous dose and upon the personal degree of immunity.

Opposed to the drinking-water theory of this disease is that of Pettenkofer, which contends that the spirilla found in the serous evacuations of cholera patients must enter an appropriate soil and there undergo further development before becoming truly pathogenic. While soils possessing a certain degree of moisture and perviousness and contaminated with organic matter favor the growth and multiplication of the specific organism, these telluric conditions are not essential, as is shown by the virulence of the intestinal discharges when swallowed in ample quantity. Pettenkofer and Rubino[1] claim that the fully developed poison rises from the subsoil into the lower atmospheric strata as a miasm, especially at the time of the subsidence of the ground-water level in summer.

Immunity is not conferred by a previous attack of cholera. Pfeiffer and Marx have proved the existence in the blood-serum of human beings of bactericidal bodies (not a true antitoxin) that cause rapid destruction of the cholera bacilli. To these anti-bodies is ascribed the "Pfeiffer serum reaction," by means of which the vibrios are differentiated from other micro-organisms. Pfeiffer and Marx have also shown that the virus of cholera can be effectively preserved by a 0.5 per cent. solution of carbolic acid, and that it in no way impairs its immunizing properties.

Clinical History.—The **incubation period** varies from a few hours to five days. During this prodromal period the patient is either quite well or (during the latter portion) exhibits certain *local* symptoms. These are occasionally nausea, a feeling of distress in the abdomen, increased peristalsis which may be visible or palpable, slight pain and tenderness, and either a mild or a decided diarrhea. The discharges are feculent, colored, and semifluid, or more rarely quite fluid, and may be quite copious. These symptoms may all be present, though oftener a few, and rarely a single one, is noted; moreover, they are not distinctive unless seen during an epidemic and unless the patient have been exposed to the poison. *Prostration* may be rarely marked and there may be slight muscular cramps. The so-called *premonitory diarrhea* may terminate in recovery at the end of from one to three days; but if not, it is followed by an attack of true cholera. This has three stages.

(1) **Stage of Serous Diarrhea.**—The *dejections* are generally painless, very frequent, odorless, copious, and fluid or watery, and usually present the characteristic "rice-water" appearance. Rarely they are distinctly colored with bile, and in severe cases with blood, and rarely also are they frothy. Suspended in them are numerous small, whitish, mucous flakes; their reaction is neutral or alkaline, and they contain a small percentage of solid constituents made up largely of albumin and sodium chlorid. The microscope brings to view epithelium, mucus, triple phosphates, and numberless micro-organisms, of which latter the only ones characteristic are the comma bacilli (spirilla) of Koch. In *cholera sicca* these serous evacuations are absent. Death comes quickly, and post-mortem examination shows the intestine to be filled with rice-water material, which is probably retained because of speedy paralysis of the musculature.

Gastric symptoms appear early. Vomiting soon becomes frequent, and at first the vomitus may be bilious; later it is characteristically serous and excessive in amount. Thirst is almost intolerable, anorexia is complete, and the tongue often has a thick coating which early becomes dry. Gastro-intestinal pain is not severe, but a feeling of pressure or burning in the abdomen is experienced, and occasionally there are griping pains with tenesmus. The *physical signs* are few. The belly is usually flat and flaccid, though it may be scaphoid and hard, and in some cases palpation detects fluctuation.

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[1] *Sajous's Annual*, vol. ii., p. 214, 1899.

Painful cramps in the *muscles* form an early characteristic symptom. They affect the voluntary muscles of the legs, calves, and feet, more rarely the arms and hands also. Their duration is momentary, but they recur at intervals, and are due to the local action of the toxins.

Owing to the withdrawal of fluid from the lymphatics and blood-vessels the tissues become dry and shrivelled and the blood much thicker. This condition of the blood obviously increases the labor of the heart, which beats rapidly, and there may be at first a distressing palpitation; but soon the heart grows more and more feeble and venous stasis ensues. The *pulse* is at first rapid, soft, and small; it may then be lost at the wrist. The cardiac impulse and heart-sounds may disappear with increasing asthenia.

The *facies* and *general appearance* also indicate loss of fluid. The cutaneous surfaces of the face and extremities grow cool: there is rapid general emaciation, which may become most pronounced, and the skin is wrinkled. The complexion assumes a livid or blue-gray tint, while the lips become quite dark. The extremities are cyanotic (the finger-tips in particular), the orbits are deeply sunken, the cheeks hollow, the features intensely pinched, the voice husky and feeble, and there is utter prostration. The *surface-temperature* drops below the normal, even to 96° or 95° F. (35.5°–35° C.), while, *per contra*, the internal or rectal temperature rises to 102° F. (38.8° C.) or over. The *mind* may remain clear until the close, but oftener the patient is apathetic, and in grave cases this condition may deepen into stupor or even actual coma. The *reflexes* are greatly diminished; restlessness and jactitation may rarely appear.

The *urine* becomes very scanty and is highly concentrated, the standing specimen depositing a heavy sediment. On analysis albumin and casts (chiefly granular) are found. In the serious forms the kidneys fail to eliminate the urea, and there is finally complete anuria.

(2) **Stage of Algidity or Collapse.**—The symptoms which characterize this grave condition are the same as those noted under the latter part of the first stage, only intensified. Asthenia is extreme; the pulse is missing and the heart beats faintly; the voice is lost; respirations are perceptibly shallow; lividity is intense; the surface ice-cold; and there is usually stupor or even coma. The excessive serous discharges have given place to mere dribblings from the now relaxed anus. During this stage, which may last a few or many hours, the faint glimmerings of the vital spark are often extinguished.

(3) **Stage of Reaction.**—This sets in promptly, and when reaction follows the first stage directly the case may pursue a favorable course, with return to accustomed health by the end of a week or ten days. The first urine passed is usually albuminous and contains tube-casts and sometimes blood-cells. Relapses into the stage of collapse may occur and be repeated; in many instances, however, this stage is both protracted and dangerous. It is aptly termed *cholera typhoid*, since a genuine typhoid state of the system with more or less fever develops. The skin may present so-called choleraic eruptions (macular, roseolar, erythema, purpuar).

Recovery may now take place, or a great diversity of local secondary inflammations may supervene (*vide* Complications).

Acute nephritis, which may or may not be an essential part of the process, may arise in this stage and lead either slowly or directly to uremic poisoning, as shown by the projection upon the scene of grave nervous phenomena—headache, vomiting, delirium or coma, and convulsions. A fatal result may be looked for.

Complications.—In this place are to be enumerated the conditions due to secondary infection, including (commonly) septic and pyemic processes. Diphtheritic inflammations affecting mucous surfaces, but especially the throat, colon, and the external genitals, are among the more common. Bronchitis, pneumonia, and pleurisy may arise, and erysipelas and parotitis are not rare. During convalescence digestive disorders may show themselves, and indiscretions in diet may precipitate a relapse.

Clinical Types.—(*a*) **"Premonitory Diarrhea."**—This type has been outlined with sufficient fulness in the foregoing discussion.

(*b*) **"Cholerine,"** in which the symptoms are mild, resembling those of cholera nostras. Many of the symptoms characteristic of true cholera are also present, particularly the cramps and prostration, cold extremities, and scanty albuminous urine. The stools, however, are not, as a rule, typical of the disease, but are feculent in character, as in ordinary cholera morbus. The duration is from seven to ten days, subject to relapses.

(*c*) The more typical forms—both moderate and severe—have been described under the Clinical History (*supra*).

(*d*) **The Foudroyant or Asphyxic Form.**—This may kill instantly; more frequently the patient lives for a few hours, with or without vomiting and purging. *Cholera sicca* should be classed with this type. The virulence of the cholera-poison explains the intensity of the symptoms.

Differential Diagnosis.—This is difficult in the absence of an epidemic unless bacteriologic and microscopic tests be made, and yet these alone differentiate a sporadic case. The disease most commonly mistaken for cholera (especially cholerine) is *cholera morbus*, and the following points pertaining to the latter disease will eliminate it: 1. No connection with a previous case, but a frequent history of dietetic imprudence. 2. Absence of "rice-water" stools, which remain turbid with feces or covered with bile or blood. 3. Presence of colicky pains, but absence of painful tonic cramps of legs and feet. 4. Absence of cyanosis and collapse, as a rule, and of urinary suppression. 5. No cholera spirilla in the stools.

Arsenic-poisoning and other forms of *gastro-enteritis* must be discriminated by the history, the character of the stools, the absence of violent muscle-cramps and of the effects of great loss of fluid (cyanosis, shrunken body, profound collapse, etc.). Chemical tests are not to be neglected if the history points to any form of corrosive poisoning.

Prognosis.—This is dependent mainly upon the type. Thus "cholerine" is very rarely fatal, while, on the other hand, the asphyxic form is almost as rarely survived. It is impossible to state the average mortality, since it varies with each epidemic, but it has been found to range from 20 to 80 per cent. Many deaths occur during the latter part of the first day or during the algid period; still more during the stage of reaction, the dangers of the latter period being as follows: asthenia, cholera nephritis with uremia, and the various complications (*vide supra*). The personal circumstances which render an attack grave are old age, alcoholism, previous ill-health, and debility. On the other hand, the death-rate may readily be lowered by prompt and judicious treatment.

Treatment.—Prophylaxis.—Prevention is of greater importance than cure, and is easily accomplished as compared with the eradication of the disease. It has been owing in great measure to the efficient quarantine system of the United States that cholera has not gained a foothold on our shores since 1873.

Individual Prophylaxis.—In the first place, those nursing the sick can prevent the spread of cholera by prompt and thorough disinfection of the vomitus and stools, as well as of the receptacles containing them and anything that may be soiled by them. The dejecta may be disinfected by pouring upon and mixing with them an equal part of a 5 per cent. solution of carbolic acid or an equal volume of a freshly prepared solution of chlorid of lime. The discharges thus treated must be covered and allowed to stand from fifteen minutes to half an hour, and then emptied into a pit in the earth containing quicklime, with which they should also be covered. It is of the utmost importance to guard against a pollution of the water-supply by these pits. Soiled clothing, linen, etc. should be promptly disinfected, and bedding had better be burned; none but the attendants should be permitted to enter the sick-room. The dishes used should be disinfected immediately after use or before leaving the sick-chamber. Shakespeare further recommends that the remains of the patient's meals should be disinfected and destroyed. After handling the patient or anything that he has soiled the attendants should first disinfect and then carefully wash their hands, these ablutions being performed invariably before eating. After vomiting and after an evacuation of the bowels the mouth and the parts around the anus should be wiped with a cloth wet with a solution (1 : 2000) of mercuric chlorid. If convalescence supervene, the patient should be kept isolated for a week and the stools should be disinfected during that time.

Persons exposed should use boiled milk and water only. Certain forms of food must be avoided, especially salads and unripe fruits; also alcoholic stimulants. All uncooked food may be pernicious. Such persons should lead regular lives, avoiding fatigue, excesses, etc., and intestinal disturbance must be met speedily by the use of antiseptics, opiates, and astringents. In India, Haffkine[1] has used a protective virus with encouraging results. Thus, "of 1735 persons not inoculated in a certain section, 174 took the disease and 113 died, whereas of 500 inoculated but 21 were affected and 19 died." He has made, altogether, 70,000 injections in 40,000 patients without a single accident, and claims that the results have been entirely favorable. Behring and Ransom have also succeeded in obtaining an antitoxic serum. Klein concludes against Haffkine's anticholera inoculations, showing that there is no certainty as to the protection against the specific poison in the intestines, even though there may be protection against the effect of intracellular poison. Klemperer has produced immunity by using a toxin.

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[1] *Münch. med. Woch.*, Jan. 29, 1895.

Treatment of the Attack.—(*a*) **Premonitory Diarrhea.**—When the prodromal period exists it must be quickly combated, and if this were attended to appropriately few cases of cholera would follow. In the instances which are not preceded by premonitory diarrhea opportunity to prevent the attacks does not present itself. In this stage a double indication is presented—"to restrain the development of the bacilli in the intestine and to neutralize the cholera-poison." To meet this Cantani proposes tannic acid by irrigation (enteroclysis). He injects into the intestine ½ to 2½ quarts (liters) of water, or infusion of chamomile containing ʒiss to ʒv (6.0 to 20.0) of tannic acid, gtt. xx to xxx (1.20) of laudanum, and ʒv–xij (20.0–50.0) of gum arabic. The temperature of the liquid should be 80°–104° F. (26.6°–40° C), in order not to chill the patient. Injections should be repeated four times a day, and in grave cases after each evacuation.[1] To this should be added a regulated liquid diet, with rest and recumbency. For the same purpose acetate of lead and opium, or large doses of bismuth with or without Dover's powder, have been much employed with good results.

(*b*) **Stage of Serous Diarrhea.**—The chief indication is to restore to the blood the watery elements withdrawn by the diarrhea. Not a moment is to be wasted. Opium, and preferably the salts of morphin, should be administered hypodermically, the dose not being small, but gr. ¼ to ⅓ (0.0162–0.0216) to be repeated at intervals of about eight hours. To opium given *per oram* or in the usual way there is a serious objection—namely, its slowness of action. Cantani advocates the injection of an artificial serum (*hypodermoclysis*) containing 1 dram (4.0) of sodium chlorid and gr. xlvj (3.0) of sodium carbonate per quart (liter) of sterilized water warmed up to 104° F. (40° C.) into the subcutaneous connective tissue. This solution may be introduced through the cannula of an ordinary aspirator, the fluid flowing by gentle pressure. Shakespeare recommends for hypodermoclysis a fountain syringe with a long flexible tube furnished with a cock; with another shorter tube, one end attached to the cock, the other having a needle-pointed cannula, a little longer, stronger, and with a somewhat wider caliber than the ordinary hypodermic needle (Fig. 12). The tube and cannula are first perfectly filled with a fluid, and then the cannula is inserted well in between the skin and deep fascia of the flanks, buttocks, or interscapular region. The fluid should be made to flow slowly, allowing fifteen to twenty minutes for the introduction of one quart. This is preferred to intravenous injection, in which the liquid is diffused slowly. The indications presented by the premonitory stage must be met as above stated.

The *vomiting* is to be relieved by bits of ice, small amounts of brandy and water at brief intervals, cocain, or by *lavage*. In this stage remedies by the mouth should be avoided, since they aggravate the gastric disturbance. Heat should be applied externally with a view to assisting the peripheral circulation as well as the reaction, and, on the other hand, to obviate collapse. Warm baths have been recommended for this purpose. Stimulants must be used to fulfil the same indications. They are of superior value even to the above-mentioned measures, and are to be

[1] *Annual of the Universal Medical Sciences*, 1893.

given hypodermically, and either brandy, ammonia, or strychnin may be employed in large doses.

(*c*) **Stage of Algidity.**—If this develop, the case is desperate. In this stage the following measures and procedures, which have been detailed in the treatment of the preceding stage, are to be persevered with:

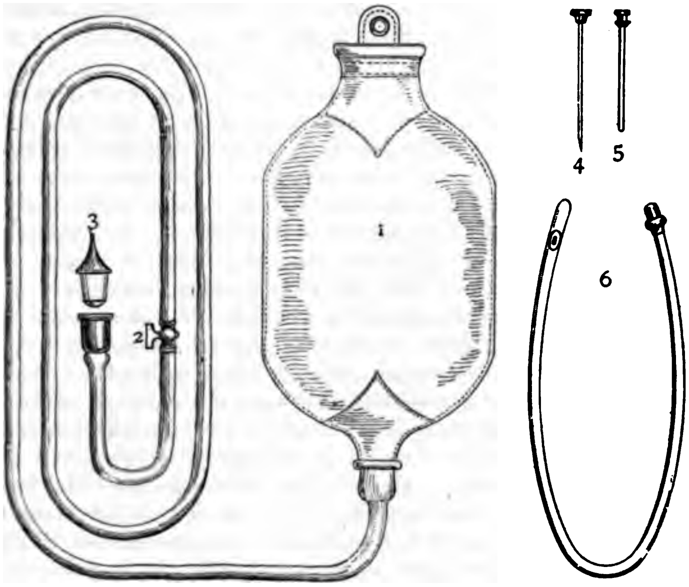

Fig. 12.—1, fountain syringe; 2, cock; 3, attachment for cannula; 4, needle; 5, cannula; 6, soft-rubber rectal tube, with two lateral openings, one a half inch from the end (not visible), the other two inches from the end. The latter is to be introduced by a combined rotatory and pushing motion to the depth of ten inches in enteroclysis, and the fluid then allowed to enter the colon slowly.

enteroclysis and hypodermoclysis, hypodermic stimulation, and the external application of heat. Additionally, intravenous injections of fluids have been strongly urged by its champions. For this purpose the following standard of saline fluid may be chosen: sodium bicarbonate 1 part, sodium chlorid 6 parts, boiled water 1000 parts. The temperature of the fluid when injected varies according to circumstances from 100⅖° to 104° F. (38°–40° C.), more frequently the latter (Shakespeare). The quantity demanded may be 1 or 2 quarts (liters), and the injection may need to be repeated in from one to three or four hours. Despite the physician's best efforts, patients in this period usually succumb.

(*d*) **Stage of Reaction.**—During this stage the tannic acid may be replaced by a solution of salt in water (10 or 15 per cent.) for enteroclysis (Cantani), and it may be well to continue hypodermoclysis in some instances. Further than this, the treatment is essentially symptomatic. Food of the blandest sort and in small quantities must be allowed at frequent intervals if we would avoid enteritis and other unfavorable complications. Tonic remedies should be given cautiously, and rest and careful nursing insisted upon. Complications must be met in accordance with general principles.

YELLOW FEVER.

(*Febris flava; Gelbfieber, Ger.*)

Definition.—Yellow fever is an acute, highly infectious (but non-contagious) endemic and epidemic disease. It is characterized by a sharp period of invasion, followed by a period of remission, and the latter in turn by a relapse and certain symptoms peculiar to the affection (black vomit, jaundice, suppression of urine).

Historic Note.—Yellow fever is endemic only within certain geographic limits, where it also prevails epidemically when the conditions are favorable. According to general belief, it first appeared in 1647 in the Barbadoes (West Indies). Subsequently, it was conveyed along the channels of commerce until it became widely disseminated, and chiefly in seaport towns. In 1699 an English vessel carrying slaves transported the disease to Mexico from the Atlantic coast of Africa. Guitéras classified the areas of infection thus: (1) The *focal zone*, in which the disease is never absent, including Havana, Vera Cruz, Rio, and other Spanish-American ports. (2) *Perifocal zones*, or regions of periodic epidemics, including the ports of the tropical Atlantic coast in America and Africa. (3) The *zone of accidental epidemics*, between the parallels of 45° N. and 35° S. latitude. Yellow fever was brought to the United States (Boston) in 1693, and since then has invaded in epidemic form numerous sea-coast cities, being carried thence to a number of inland towns. The belief that the disease never originates outside of certain territorial limits was advanced for the first time by the College of Physicians of Philadelphia (1797), and the efficacy of rigid quarantine regulations in preventing conveyance of the poison by vessels having yellow-fever cases on board was pointed out by the same organization.

Pathology.—The *skin* is jaundiced (hepatogenous) and often large or small ecchymotic spots are observed, but neither the internal viscera nor the blood shows characteristic lesions in cases of average intensity. In severe forms congestion, hemorrhage, and degeneration are the changes noted, especially in the liver and the gastro-intestinal mucous membranes.

After death the liver is anemic, as a rule, but in the early stages of the disease it is markedly hyperemic. Its color varies, ranging from pale yellow to an orange hue, and punctiform extravasations cause mottling of the surface. Its size varies little from the normal. Parenchymatous degeneration of the hepatic tissue is common, though in places it may be entirely normal. The liver-cells are swollen, containing fat and granular matter with indistinctness or absence of nuclei.

The gastro-intestinal mucosa is the seat of numerous minute hemorrhages, similar spots of extravasation being found on the various serous membranes of the body (meninges, pericardium, pleura, etc.). Hemorrhagic infarctions may be found in the various internal viscera. The lesions of acute catarrh are seen in pronounced form in the gastric mucosa, which may also present erosions. The black-vomit material is found in the stomach, and less frequently also in the smaller intestines, which present the evidences of acute catarrh of their mucous walls.

The *spleen* is dark and friable, but is not enlarged. The *kidneys* show the lesions of parenchymatous nephritis, the microscope revealing cloudy swelling of the epithelium of the tubules with fatty degeneration, and the tubules themselves being occupied by casts, chiefly granular. The heart-muscle looks pale, and may be the seat of granular and fatty degeneration. The *brain* and its *meninges* are hyperemic, and degenerative changes have been described in the sympathetic ganglia (Schmidt).

The *blood* is dark, and many of the red corpuscles, having disorganized, set free hemoglobin, as in malaria. Certain significant lesions of a general character—such as a fatty degeneration of the walls of the small blood-vessels and the capillaries—have been noted by competent observers, and these, by allowing filtration of blood-serum through the vessel-walls, may account in great measure for the concentration of the blood.

Etiology.—Bacteriology.—J. Sanarelli has described a specific organism of this disease, and named it the bacillus icteroides. This micro-organism is not detectable in all cases during life. Wasdin and Geddings[1] found it present in blood taken from the ear in 92.85 per cent. of cases; while Walter Reed,[2] Surgeon United States Army, and his corps of assistants, were unable to find the bacillus icteroides in peripheral venous blood at various stages of the disease; but usually found such blood free from bacteria. Cultures made at 11 autopsies from organs and fluids of persons dead of yellow fever were likewise negative. Pothier[3] isolated the bacillus icteroides in 3 out of 51 autopsies.

Mode of Infection.—Transmission through the agency of the mosquito was pointed out by C. J. Finlay[4] in 1881. Mosquitoes were permitted to prey on persons sick of yellow fever, and later to bite susceptible subjects, in whom from five to twenty-five days developed a mild form of the disease. Partial or complete immunity[5] against yellow fever was obtained after the bites of infected insects.[6] The mosquitoes were identified as "Culex fasciatus." Surgeon Reed[7] and his assistants succeeded in producing the disease in 65 per cent. of their experiments, which consisted in subjecting susceptible persons to the bite of the infected female culex.

Among *predisposing causes*, *season* heads the list. The disease prevails chiefly in summer, being completely arrested by one, or at most two, severe frosts. *Age* and *race* have some degree of influence, children being more liable than adults, males than females, and whites than blacks. The poison is not transferred by *fomites*. The march of an epidemic may be interrupted or even completely arrested by apparently trivial agencies—*e. g.* watercourses, rows or clumps of shrubbery. One attack usually bestows permanent *immunity*, and natives of an infected district are far less liable to the disease than newcomers.

Clinical History.—Incubation Stage.—This varies greatly, ranging from one day to two or even three weeks. During the incubation certain general symptoms may appear, such as languor, headache, anorexia, etc.

Invasion Stage.—The *onset* is abrupt, an initial *chill* usually occurring, but it is very seldom severe or prolonged, a reactionary fever following promptly and the *temperature* rising to 103°, 104°, or even 105° F. (40.5° C.) The temperature is apt to be highest at the beginning, and then falls gradually, hyperpyrexia being rare. The chill and fever are accompanied by headache and pains in the loins and legs, often of great severity, and a little later restlessness, mental confusion, and a delirium that is sometimes violent in character may develop. In the majority of instances, however, the mind remains clear. The *pulse* is accelerated, but not in proportion to the height of the temperature; it is full and strong at the start, and may fall while the fever is rising. The *face* is flushed, with slight icteroid addition. The *eyes* are suffused and intolerant of light. The *tongue* may or may not be coated, and nausea and vomiting may occur, the latter being one of the most characteristic symptoms of the disease. Associated with these symptoms there are epigastric oppression and burning sensations, with decided tenderness. The *vomitus* may be blood-streaked or contain chocolate-colored particles, and occasionally unaltered blood is vomited. Constipation is usually present, the stools showing a deficiency of bile. The *urine* is diminished in amount, dark-colored, and often contains a slight amount of albumin: this *early transient albuminuria* is a very characteristic symptom. The *initial stage* may last from six or eight hours to two or three days, or even longer, and is longer in the milder forms of the disease. With the termination of this stage the fever remits and the other symptoms disappear with surprising rapidity, the pulse becoming remarkably slow.

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[1] "Report on the Cause of Yellow Fever," Marine-Hospital Service, U. S. A., 1899.

[2] "Etiology of Yellow Fever," *Philada. Med. Jour.*, Jan. 10–27, 1900.

[3] *Jour. Amer. Med. Assoc.*, April 16, 1898.

[4] *Annales de la Réal Academie*, vol. xviii., pp. 147–161.

[5] *Med. Record*, May 27, 1899.

[6] Eighth Congress of International Hygiene and Demography, Budapest, 1894.

[7] *Philada. Med. Jour.*, Feb. 16, 1901.

Stage of Remission.—From this moment convalescence may begin and proceed to full recovery without interruption, the happy event being often marked by critical discharges. In most instances, however, the patient presents certain symptoms and signs of ill-health during this period (more or less prostration, epigastric distress with tenderness, mental dulness or even stupor, and a yellowish tint of skin and urine), which lasts from a few to twenty-four hours, when another stage with its more striking symptoms supervenes.

Stage of Secondary Fever or Collapse.—The patient becomes extremely weak, presenting the signs of profound *collapse*. The surface of the body is cool (extremities often positively cold), the skin in nearly all instances assuming a yellow or bronzed tinge, from which the disease receives its name. The *pulse* is rapid and compressible, and soon vomiting becomes very distressing. *Hemorrhage* into the stomach generally occurs, the blood being acted upon by the gastric secretions, and producing the material which is expelled as the characteristic "*black vomit.*" Occasionally unaltered blood may be vomited; the stools also may be tarry. In the worst cases hemorrhages from other mucous surfaces are common (epistaxis, hematuria, metrorrhagia, etc.), and cutaneous hemorrhages also now occur. In this stage the *tongue* becomes dry, brown, or even black; less frequently it is smooth, red, and fissured, and sordes may often be observed on the teeth and lips.

In most cases the *urine* is deficient, containing albumin and casts (with careful centrifugation), and in rare instances there is complete anuria. The latter may precede the development of grave *nervous symptoms*, as convulsions, or even coma, which may be uremic.

In some instances the *temperature* rises during this period (secondary fever), and in favorable cases terminates by lysis, or it may assume the typhoid form and result fatally. In all cases that pursue a favorable course convalescence is slow and gradual, and may be uninterrupted by relapses, but this is an unusual course of affairs. The duration of the entire attack (composed of three stages) is variable, though as a rule it covers about one week.

Clinical Varieties.—Many different varieties have been described, each characterized by one or more prominent features, but none seem more justifiable than Finlay's[1] classification, in which he distinguishes three forms: (1) the *acclimation fever*, or *non-albuminuric yellow fever;* (2) the *plain albuminuric yellow fever;* (3) the *melano-albuminuric yellow fever*, characterized by the presence of blood or "black vomit" in the stomach or intestines.

Diagnosis.—The symptoms that justify a diagnosis in the initial stage, provided an epidemic be prevailing, are the sudden onset, headache, severe lumbar pains, peculiar facies and pulse, nausea, and vomiting of bile. In the early stage intense capillary congestion of the surface of the body is diagnostic and indicative of a severe form of the disease. In the third stage the coexistence of jaundice, the black vomit, and suppression of urine, with evidences of collapse, makes the diagnosis easy.

Serum-diagnosis.—Woodson and P. E. and J J. Archinard have applied the Widal reaction (agglutination-test) in 100 cases, and claim that the serum-diagnosis of yellow fever is practicable and important, and may be used on the second day. A dilution of 1 : 40, with a time-limit of one hour, is preferable.

Differential Diagnosis.—*Pernicious malarial fever* has not the deep jaundice, the slow pulse, the peculiar temperature-curve, the intense capillary congestion of the surface of the body, the black vomit, the early albuminuria, and the clear mind—all symptoms that mark yellow fever. On the other hand, the organism of Laveran is pathognomonic of pernicious malarial fever, as is the effect of quinin upon the disease. Kemp has made a microscopic, spectroscopic, and chemical study of the black vomit of yellow and malarial fevers, and found that the pigment in each case was derived from the blood, which had been acted upon by the gastric juices. The vomitus in malarial fever, however, contains in addition considerable quantities of bile-pigment and bile-salts, which are wanting in that of yellow fever. Further, in the latter the vomited matter is much more highly acid.

Prognosis.—Different epidemics show widely different death-rates, and the most potent factor is the particular type of the disease in individual epidemics. Some have been characterized by the lighter forms of the affection, and in such the death-rate has been low (1 per cent.). In other epidemics the type has been so virulent as to make the mortality list high, even to 100 per cent. In general, mild epidemics give a mortality of 5 to 10 per cent., and severer forms one of 30 to 50 per cent. The death-rate is lower in private than in hospital practice.

Among the gravest symptoms *intense capillary congestion*, coming on during the first stage, deserves special emphasis. Equally serious, in most cases in which they occur, are *suppression of urine*, *intense jaundice*, and *uremic toxemia.* The black vomit is not as fatal a sign as the symptoms previously mentioned.

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[1] *Edinburgh Medical Journal*, Edinburgh.

It has been noted that a larger number of men, proportionately, than women and children succumb to the disease, and that it is less fatal among negroes than among whites.

Treatment.—The measures that are employed in yellow fever may be considered under three main heads: (1) Prophylaxis; (2) general management; and (3) medicinal measures.

(1) **Prophylaxis.**—Reed claims that the present quarantine laws against yellow fever are needless and the detention system absurd. The effective way to prevent carrying of the fever poison is the destruction of the mosquitoes—on vessels at sea as well as in infected houses and districts on land. Well persons must be protected against the bites of the *culex.* It is unnecessary to disinfect articles of clothing, bedding, or merchandise supposedly contaminated by contact with those ill of the disease. The patient must be isolated.

Protective inoculation with the serum obtained from a horse or an ox vaccinated with the bacillus icteroides has proved effectively prophylactic in a severe outbreak in an insanitary prison.[1]

(2) **General Management.**—The sufferer from yellow fever must be put to bed at once, and an abundance of fresh air (without exposure to strong drafts) must be supplied, The medicaments and the nourishment are to be administered through a tube or spout-cup, so as to obviate raising the patient's head. Body- and bed-linen should be kept scrupulously clean, being changed frequently, and the patient must not be allowed to leave his bed on any account. The *diet* should be of the lightest sort and entirely liquid, beginning with peptonized milk, koumiss, or light broths, and in small quantities.

(3) **Medicinal Measures.**—At the outset it is well to gently stimulate the various excretory organs, and mild laxative diaphoretics and diuretics answer this purpose. Hydrotherapy may be employed to maintain the nervous tonicity and reduce the temperature, but when the spontaneous fall of temperature sets in this method must be promptly discontinued. During the first stage the neuralgic pains, which attack principally the head, loins, and nerve-trunks, are to be relieved by morphin given hypodermically; and for the same symptom Bemiss highly recommends quinin by the rectum (gr. xx—1.296). Intestinal antiseptics may also be used throughout the attack (salol, betanaphtol, etc.).

During the stage of *remission* the powers of the system are to be fully maintained by a suitable dietary and by tonics and stimulants if required.

In the last stage, supportive measures must not be forgotten, everything that aids the vital powers being brought into prompt requisition. *Rectal nutrient enemata* should be employed if marked gastric irritability prohibits feeding by the mouth. *Stimulants* are demanded, and these should also be administered per rectum if not retained by the stomach, or they may in some measure be administered hypodermically. The stomach is, as a rule, tolerant of iced champagne.

If irritability of the stomach be present, ice and hydrocyanic acid may be tried. Sodium bicarbonate (gr. x to xx—0.648 to 1.296) in Vichy, Apollinaris, or Seltzer water is a most useful remedy, and Sternberg has used it in combination with mercuric chlorid with success in the following formula:

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[1] *Philada. Med. Jour.*, Sept. 18, 1898.

℞. Sodii bicarb.,	ʒiv (16.0);
Hydrarg. bichlorid.,	gr. ss. (0.032);
Aquæ puræ,	Oj (480).—M.

Sig. For a severe case two teaspoonfuls every hour, day and night; for a mild case, every hour by day and every two hours by night; administer always ice-cold.

Perhaps the chief indication for the use of sodium bicarbonate is the extreme acidity of the various secretions, especially the gastric and renal. Sternberg contends that by fulfilling this indication we prevent in great measure the occurrence of acute nephritis and suppression of the urine. Hemorrhages and other symptoms must be treated by the usual means. During convalescence tonics are to be administered, and the customary diet can gradually be resumed.

Serum-therapeutics.—Prof. Sanarelli records favorable results from the use of his antitoxic serum. The experience of Sanarelli has been confirmed by other observers. Morcour[1] points out that we need to try the serum only in grave cases, since mild cases recover with simpler methods and careful nursing. Wasdin, however, used Sanarelli's serum in 3 cases and noted no advantage over other treatment. Matienzo,[2] after a series of experiments on guinea-pigs and human beings with American serum, concludes: Intravenous and subcutaneous injections produce general reaction: no effect is produced upon the disease or even the individual symptoms. The reaction obtained in convalescence proves that the antitoxin does not cause the cure.

CEREBRO-SPINAL MENINGITIS.

(*Spotted Fever; Cerebro-spinal Fever.*)

Definition.—An infectious disease, caused by the diplococcus meningitidis. It is characterized anatomically by inflammation of the meninges of the brain and spinal cord, and clinically by an irregular course, a moderate febrile movement with somewhat characteristic and profound nervous symptoms (excruciating headache, pain in the back and upper part of the spine, contraction of the muscles of the nucha, hyperesthesia, delirium, and ofttimes coma). The disease may occur sporadically or in epidemics, or may even assume pandemic proportions.

Historic Note.—Cerebro-spinal meningitis was first recognized and described as late as the beginning of the present century (1805) by Viesseux of Geneva. During the next decade numerous limited epidemics were observed both in Europe and the United States, and subsequently recurring epidemic and pandemic visitations were noted, though at comparatively long and variable intervals of time. In nearly all the large cities in this country it may be said to have become endemic, and in Philadelphia since 1863; yet the affection is, without doubt, becoming less and less prevalent.

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[1] *Proceedings Third Pan-American Medical Congress*, Feb. 4, 1901.

[2] *Med. News*, Jan. 13, 1900.

Pathology.—The cases that prove speedily fatal do not present gross characteristic changes, but by the aid of the microscope leukocytes are discovered immediately around the cerebral vessels, and round cells in the cortex of the brain. In some cases the characteristic evidences of encephalitis are already noticeable. On the other hand, the cases in which death occurs after the disease has been fully developed show the lesions of suppurative inflammation of the meninges of the brain. The arteries, veins, and sinuses are much engorged; the ventricles are distended with liquid, but the pia mater is principally affected, its vessels being greatly enlarged, and a more or less copious sero-fibrinous or sero-purulent exudate occurring into the meshes of its network. The longer the duration of the case the more purulent is the exudation. The ventricles of the brain are filled with a similar exudation, and red blood-globules may be present at an advanced stage. The color of the exudate is at first almost clear (being composed of serum); it then changes to a milky turbidity, to a pale yellow, and, lastly, takes on a greenish-yellow color ("leek-green"). The subarachnoid space may be occupied by a uniform layer composed of fibrin and pus, which exhibits the greatest thickness along the longitudinal fissure.

The brain-matter is congested, and sometimes softened in spots, and on section the gray matter may present punctate extravasations. When resolution occurs recovery may be complete, but more frequently the pia mater remains thickened.

The exudation may follow the auditory and optic nerves along their lymph-sheaths, and pus has been found in the internal ear as well as in the chambers of the eye.

The membranes of the spinal cord manifest lesions identical with those of the brain. They are vascular engorgements, followed by sero-fibrinous, and later still by sero-purulent, exudation beneath the arachnoid. The changes are more marked on the posterior than the anterior surface of the cord, and the exudate increases in amount in passing from above downward, in severe cases sometimes assuming the form of a sheath which completely surrounds the cord throughout its entire length. The pia mater is congested, and may be thickened, shaggy, and in places adherent to the cord, of which the gray matter may be the seat of serous infiltration, and rarely of softening.

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Etiology.—**Bacteriology.**—The diplococcus meningitidis is the specific cause of epidemic cerebro-spinal meningitis. The special organism can be isolated from the spinal fluid, the meninges of the brain and cord, the blood, the joint-lesions, and the nasal mucus.

The meningococcus, like the gonococcus, occupies a position within the polynuclear leukocytes, and like the latter is biscuit-shape. The bacterium takes the usual stains. It develops upon agar-agar and upon Loeffler's blood-serum, manifesting characteristics of growth that simulate those of the pneumococcus. Councilman, Carl Fränkel, Boston, and others, by refined methods, have, however, been able to differentiate these organisms. Netter,[1] and other writers, hold that primary cerebro-spinal meningitis may also be due to the pneumococcus. Welch suggests that the meningococcus and the pneumococcus are possibly varieties of the same bacterium, while Netter regards the meningococcus as a degenerate form of the pneumococcus. Among the associated microbes are the pneumococcus, streptococcus pyogenes, staphylococcus aureus, bacillus coli communis, and the tubercle bacillus.

Predisposing Causes.—(1) **Age.**—Most cases occur in children and young adults, though no age enjoys perfect immunity.

(2) **Climate.**—The disease is unknown in tropical climates, but has occurred in all parts of the temperate zone, and is most prevalent in the more northerly portions of the latter.

(3) **Season** is not an important factor, though the disease prevails largely in cold weather.

(4) **Unhygienic Influences.**—Those who live under unfavorable sanitary influences are especially liable, and hence the disease often appears in ill-ventilated and overcrowded habitations—among the poorer classes, among soldiers crowded together in barracks, and among prisoners. Prolonged marching, and excessive physical or mental exertion, may heighten the susceptibility to the disease. In certain epidemics the disease has raged exclusively in villages.

Modes of Conveyance.—Precisely how the contagion is transferred from an infected person to a healthy one is not known, but the disease is probably not contagious. There is evidence to show that the poison may be conveyed by *fomites*, though this seems to be limited to the cases furnishing intensely virulent poison. The organism, owing to its feeble vitality, probably could not lead a saprophytic existence. As to the manner in which the virus gains entrance to the system, our knowledge is imperfect also.

Clinical History.—The period of **incubation** is brief, though unknown. The *prodromal symptoms* are variable in different epidemics, and may even be absent when the invasion is sudden, a patient in vigorous health often being stricken down as though by a blow. In some rapidly fatal cases there is a short prodromal period, during which the patient complains of lassitude, headache, rachialgia, muscle- and joint-pains, and sometimes nausea and vomiting. In ordinary forms the prodromes may last from a few hours to a week or more, and the patient's complaint may be limited to cervical and occipital pains lasting a day or two; then, without any initial chill, the *invasion-period* supervenes. In milder, and usually in sporadic, cases the symptoms consist chiefly of languor and debility, headache, pain in the back and limbs, vertigo, vomiting, and sometimes diarrhea.

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[1] *Twentieth Century Practice of Medicine*, vol. xvi., p. 191.

Most cases begin *abruptly*, between noon and midnight. The most distinctive and violent features are chill (often severe), *fever* of a moderate grade, a full and somewhat *accelerated pulse*, *raging headache*, and *vomiting*. In children the ushering-in symptom may be a *convulsion*. These symptoms are followed soon by pain in the back and cervical portion of the spine—an early and characteristic symptom. Attempts at flexion or rotation of the head increase the pain in the neck, and in like manner movements of the body augment the spinal pains. Later, the muscles in the cervical region contract, at the same time becoming rigid, and produce the condition of opisthotonos. The patient may be unable to swallow on account of the excruciating pain excited by the act.

The *temperature* is but moderately elevated. In a certain percentage of the cases it rapidly rises to 104° or 105° F. (40.5° C.), but soon falls to 102° or 103° F. (38.8° or 39.4° C.), at which level it is maintained with irregular undulations until defervescence, which takes place by lysis. In fatal cases death is preceded by a sudden great elevation of temperature to 108° and even 110° F. (43.3° C.). In the very young the thermometric range is lower than in adults.

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The *respirations*, as a rule, increase in frequency and are sometimes quite irregular; but marked dyspnea, with slowing of the respirations, may be observed during the advanced stage, being due to pressure exerted by the exudation upon the respiratory center. Cheyne-Stokes breathing and sighing respirations may be present.

Nervous Symptoms.—The *headache* is racking and often persistent, though it is subject to remissions; and is intensified by light and sounds, being so violent as to cause the patient to groan even while profoundly comatose. There is vertigo in nearly all instances. The pain referred to the spine may be general or limited to either the lumbar or cervical region (rarely the dorsal), and the general myalgic pains are often intense, especially in the extremities and the abdominal region. With the cephalalgia and abdominal pain may be associated vomiting. *Hyperesthesia* is a prominent symptom, the gentlest touch being extremely painful; *anesthesia* may also be noted, though less frequently, and usually follows the hyperesthesia. Any voluntary muscular movements, however, excite pain. In some cases *delirium* appears early, and in others rather late, while in the worst types death often occurs before delirium develops. On the other hand, in a small percentage of cases this symptom is absent throughout the entire course, and always its character and intensity exhibit a remarkable variety. It may be mild or it may take the form merely of incoherent answers to questions. Active delirium, however, is common and is accompanied by hallucinations, during which the patient shouts loudly, and, unless restrained, gets out of bed. This form of delirium occurs in paroxysms that are most apt to appear at night, and in the female it is sometimes hilarious or hysteric. An erotic tendency, with priapism or seminal emissions, has rarely been observed in males. The "maudlin" delirium of the drunkard is sometimes seen, but sooner or later somnolence appears and may deepen quickly into coma, the latter symptom perhaps being temporary, though more often it continues until recovery or death. As before stated, vomiting is common, though it may appear late in the disease; it is doubtless of cerebral origin.

Symptoms of motor irritation are among the prominent phenomena of the disease, twitching of single muscles or groups of muscles often being seen, and occasionally muscular tremors. Muscular contraction is an almost constant feature. After a few days a tonic spasm of the muscles of the extremities sets in, as the result of which the arms are bent upon the chest, the forearm upon the arm, and the thumb upon the palm; the thigh is also flexed on the abdomen and the leg on the thigh. The opisthotonos previously alluded to may be followed by trismus, which can be considered a mortal symptom. Convulsions do not occur in adults, but are common in children; occasionally, however, there are paralysis, especially of the muscles of the face, and paretic hemiplegia.

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Complications.—Many of these have already been mentioned in the portrayal of the symptoms, particularly those taking the form of destructive inflammations of the eye and ear and the paralyses of the cranial nerves. The purulent inflammations of the serous sacs which were referred to in discussing the pathology (pleurisy and pericarditis) are among the frequently associated conditions, and secondary bronchitis is also common.

Pneumonia (lobar and lobular) is a frequent complication.

Hemorrhagic nephritis is a rare complication.

Special and Atypical Forms.—(1) **Mild or Rudimentary.**—In this type the characteristic signs are either undeveloped or wanting, and the diagnosis is possible only during the prevalence of epidemics, which furnish typical cases. The symptoms vary and are indefinite, but perhaps the most constant and significant are severe headache, languor, vertigo, nausea, and occasionally vomiting. Fever and contraction of cervical muscles are absent, as a rule. The duration of rudimentary cerebrospinal fever is brief, the more noticeable symptoms rarely exceeding three or four days.

(2) **The Abortive Form.**—Here the initial symptoms are severe, but after two or three days they rapidly subside, leaving the patient convalescent. The disease is cut short by the acquisition of immunity, and not as the result of medical interference.

(3) **Intermittent Form.**—In this variety the symptoms, however intense, remit or almost wholly intermit every day or second day; these remissions are followed by a decided exacerbation or recurrence of the distressing features of the disease. Intermissions may occur at the beginning of a case, though more often they occur at an advanced stage and tend to prolong its course. There is not observed the strict periodicity that is seen in malaria, and neither is the temperature-curve typical of the latter disease nor are the malarial organisms found in the blood.

(4) **Typhoid Form.**—In a certain though small proportion of the cases the special features are characteristic of the "typhoid state," but their course is more protracted than is usual.

(5) **Fulminant or Apoplectic Form.**—The symptoms characterizing this most malignant type of the affection are rather inconstant. There may be severe chill, loss of consciousness, followed by deep coma and death, the whole course occupying the space of a few hours only. I saw two such cases in the same family: the first, a girl of five years, was stricken at 2 P. M. and died at 9 P. M.; the other, a boy of seven years, was taken ill on the following day about the same hour, and died at 10 P. M. Other instances pursue a somewhat slower course, though manifesting the most striking malignancy. These begin with intense chills, violent headache, vomiting, early stupor, great prostration, contraction of muscles of the neck, moderate fever, and a feeble, progressively slowing pulse until it sometimes reaches 50 or even 40 beats per minute. The eruption,

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[1] *Amer. Jour. Med. Sci.*, July, 1899.

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Deafness is by no means an infrequent symptom, there being an early intolerance of sound and a marked tinnitus aurium. Later, suppurative inflammation of the middle ear, followed by rupture of the tympanum and otorrhea, may occur. The internal ear may be similarly involved, and in such cases the gait may become uncertain from implication of the semicircular canals. The deafness may after recovery be found to be permanent, though, as a rule, it is incomplete.

Cutaneous symptoms appear, some of which possess considerable diagnostic worth. Pallor and lividity of the skin and visible mucous membranes often characterize the period of invasion, and shortly after the onset herpes facialis appears in more than half the cases. This symptom is significant for diagnosis. The separate lesions are extensive, and often coalescence of two or more is witnessed. Herpes facialis belongs in a peculiar sense to cerebro-spinal meningitis—herpes labialis to malaria, and less frequently to pneumonia and meningitis. A petechial eruption is common, and has been most frequently met with in the early epidemics, and more frequently in America than in Europe. To this symptom the pirations be irregular, atropin may be combined with the opiate; and if the heart threatens to fail, strychnin may be administered. In young children we must rely upon the bromids rather than the opiates. In older children we may employ opium if we do so cautiously, and I have found the deodorized tincture of opium and paregoric to be the best preparations.

For the tonic contraction of the muscles, especially when associated with violent cerebral symptoms, cannabis indica should be tried. Convulsions call for warm baths or ether-inhalations. Mercury has been, and still is, firmly advocated by certain authors, and, cerebro-spinal meningitis being an infectious disease, this drug may be given for its antiseptic virtue (mercuric chlorid gr. $\frac{1}{24}$ (0.002) every four hours to an adult; calomel, gr. $\frac{1}{12}$–$\frac{1}{16}$ (0.005–0.004) every four hours to children). Belladonna and ergot have been employed to diminish the congestion of the cerebro-spinal capillaries. They should be administered in the early stages. Antipyrin, acetanilid, and phenacetin are objectionable, since they depress the circulation.

Stimulants are required if signs of heart-exhaustion appear. They may be freely exhibited in accordance with the customary rules.

After effusion of the exudate has taken place the narcotics are to be replaced by agents that promote absorption, as potassium iodid.

The **local means** are also important. When tub-baths are not available, cold should be used locally, since it is both of value and very grateful to the patient. An ice-bag is to be put on the head, and, if possible, long ice-bags placed along the spine. In rare cases of sthenic type we may employ small blisters at the nape of the neck or over the mastoids: these should be applied early, though they are also useful during the stage of effusion. In the usual form of the disease it is better to apply the thermo-cautery lightly over the mastoid region. If the patient be not too much enfeebled, we may abstract a small amount of blood by means of leeches or by a few wet cups placed behind the ears. Quincke's lumbar puncture and laminectomy with free drainage have been practised, but their value as curative measures is still doubtful.

Convalescence is prolonged, and requires to be diligently and judiciously treated. We must rely upon the generally accepted tonics—iron, cod-liver oil, arsenic, and strychnin; the potassium iodid and the mercury also being continued for their influence in promoting the absorption of the exudate. Special attention is, however, to be paid to the hygienic management of this period. An abundance of fresh air, sunshine, and easily assimilable food must be furnished at all hazards, and electricity and massage, judiciously employed, will hasten recovery.

LOBAR PNEUMONIA.

(*Croupous or Fibrinous Pneumonia; Pneumonitis; Lung Fever.*)

Definition.—An acute infectious disease caused by the ***Micrococcus lanceolatus***, which produces a specific inflammation of the parenchyma of the lung and marked constitutional disturbances—chill, extreme prostration, and fever which terminates by crisis. There are different forms of lobar pneumonia, classified according to their clinical or pathologic peculiarities, as primary lobar pneumonia, secondary lobar pneumonia, and lobar pneumonia with the formation of new connective tissue, etc. I shall describe the third form separately (p. 514).

Pathology.—Usually the lesions are confined to the whole of one lobe; less frequently to the whole of one lung, and rarely to parts of both lungs. From Jürgensen's analysis of 6666 cases the following statement, showing the different situations of the lesions and their relative frequency, was taken: Right lung, about 54 per cent.; left lung, about 38 per cent.; and both lungs, about 8 per cent. In the right lung the lower lobe was involved in 22 per cent., the upper in 12 per cent., the middle in nearly 2 per cent., and the whole lung in about 9 per cent. In the left lung the lower lobe was involved in about 23 per cent., the upper in about 7 per cent., and the whole lung in about 8 per cent. Both lungs were implicated in 8 per cent.

The lesions of pneumonia are those of three stages: (*a*) Stage of congestion or engorgement; (*b*) Red hepatization (consolidation); and (*c*) Gray hepatization.

(*a*) **Stage of Engorgement.**—The part or parts implicated are of a dark-red color, and firmer to the feel, but less resilient and crepitant, than normal. The cut section drips a blood-stained serum, and dark blood exudes from the distended capillaries. The air-cells do not collapse, though they are not solid, since excised pieces float; but the weight of the lung-tissue is much increased and the air-sacs are distended with the corpuscular exudate. Collapsed portions may be observed which may readily be insufflated from the bronchus, and areas of extravasation may occasionally be noted near the pulmonary pleura.

On microscopic examination the alveolar epithelium is seen to be swollen, the capillaries greatly distended, and the air-cells filled with alveolar epithelial cells, red corpuscles, and a few leukocytes. Similar elements occupy the small bronchi.

(*b*) **Red Hepatization.**—The affected tissue is solid, airless, and firm, resembling, as the term indicates, liver-tissue. It is reddish brown (mahogany) in color, presenting a dry, mottled appearance, and when, as is usual, an entire lobe is involved, it is more voluminous than normal and its surface is often furrowed by the impress of the ribs. Being airless, the affected portion does not crepitate, and its weight and specific gravity are increased. It cannot be inflated; is extremely friable, and its lacerated surface presents a finely granular aspect, this latter appearance being due to the minute plugs of inflammatory matter (fibrin) which fill the air-spaces. The air-passages and small bronchi are distended with similar material, and granular masses can be removed from the air-cells of a cut or lacerated surface by carefully scraping the latter. If death takes place during this stage, the ante-mortem, dry, inflammatory exudate soon softens, and may flow from the cut section as a grumous, viscid fluid; the consolidated tissue sinks rapidly in water. The pulmonary pleura is covered with a fine sheet of fibrin, and in cases complicated by marked pleurisy the fibrinous, inflammatory exudate forms a thick coating upon the pleural membrane, and the sac may contain liquid effusion.

Microscopic examination shows the air-spaces filled with clotted fibrin, in whose meshes are held red blood-corpuscles, pus-cells, and changed alveolar epithelium. The interlobular connective tissue may be infiltrated with leukocytes and fibrillated fibrin, but the blood-vessels in the walls of the alveoli remain pervious. The pneumococci (micrococci lanceolati), less frequently also streptococci and staphylococci, are detectable.

(*c*) **Gray Hepatization.**—In this stage the fibrinous exudation becomes decolorized, the surface at first resembling granite in color, and later appearing uniformly gray. Associated with this change, and following it, there is fatty and granular degeneration of the inflammatory exudate, in consequence of which the latter becomes moist and soft. The exudate loses its granular character, while at the same time the friability of the lung-tissue is further increased, and from the surface of the cut section there flows usually a grayish-white or yellowish-white purulent liquid. Not less than one-half of the fatal cases die in the early part of this stage. The pleura that invests the involved tissue is usually covered with a fine fibrinous exudation.

Microscopic examination shows the air-cells stuffed with leukocytes, while the other histologic elements (fibrin, red blood-cells, etc.) have disappeared; and the full development of gray hepatization marks the beginning of resolution, though the latter process may in reality begin with the commencement of the former. The exudate is now softened into a liquid material, with disintegration of cellular elements, and is absorbed by the lymphatics. *Resolution* usually corresponds in time with the occurrence of the crisis, though it may begin later. Pratt[1] found larger phagocytic cells in all stages of the disease; it is likely that they play an important part in resolution. Among unfavorable terminations are—

(1) **Purulent Infiltration.**—Here the lung-tissue becomes very soft, friable, and is bathed in purulent material; and microscopic observation shows the pus-cells densely infiltrating the interalveolar tissue and filling the air-spaces as well. This impairs the nutrition of the lung-tissue, and may thus cause rupture of the septa, producing

(2) **Abscess.**—This is to be attributed to subsequent infection by streptococci, and hence is a complicating lesion. The abscesses vary in size within the widest limits, most frequently being situated near the base of the lung, and may occupy the periphery and rupture into the pleural sac, causing pyo-pneumothorax. In most instances the abscess-cavity has a fistulous connection with a bronchus, but occasionally the abscesses become encapsulated in fibrous tissue, their contents undergoing first caseous, and then calcareous, degeneration. Rarely they open into the pericardium, and still more seldom externally. They may be small and multiple, in which case they sometimes coalesce, forming large abscesses.

(3) **Gangrene** may rarely follow, but is due to a specific cause, and hence does not belong peculiarly to the pneumonic process.

(4) **Induration.**—A. Fränkel states that in a few instances (about 1 per cent.) pneumonia ends in induration, and is found upon section to be smooth and its tissue resistant. The surface of the cut section sometimes shows a peculiar transparency, with characteristic yellow specks, due to the collection of cells which have become fatty. Microscopically, the alveoli are seen to be blocked up by connective tissue resembling polypi and containing vessels. It is probably due to secondary infection.

[1] W. H. Welch's [illegible], p. [illegible]

(5) Pneumonia, particularly of the apex, may terminate in phthisis. Tubercular infection commonly occurs in unresolved pneumonias.

Changes in Other Viscera.—The *heart* often appears pale and is flabby, but upon microscopic examination the muscular cell-fibers of the organ are not found to be degenerated, except in rare and usually protracted cases. The cardiac chambers, particularly the right, are distended with firm, tough clots, which are usually removable *en masse* from the great vessels in the form of arboreal casts. The blood tends to coagulate, owing to the fact that its fibrinous elements are vastly increased.

Pericarditis occurs in about 5 per cent. of the cases, and is relatively more frequent in left-sided or double pneumonia. *Endocarditis* is more common, especially the ulcerative form, which was present in 11 out of 100 autopsies (Osler). With malignant endocarditis the lesions of meningitis are often combined, but as a separate complication it is rare.

The *spleen* is congested, moderately enlarged, and softened, and the *liver* is likewise hyperemic and somewhat swollen. In the *kidneys* are found the lesions of parenchymatous inflammation, and with remarkable frequency also those of chronic interstitial inflammation. A catarrhal state of the gastro-intestinal mucosa (often with jaundice) is common; and a frequent complicating change is croupous inflammation of the colon.

Etiology.—Bacteriology.—The generally accepted specific cause of pneumonia is the *Micrococcus lanceolatus* of Fränkel. It is a lance-shaped (slightly elliptic) coccus, united in pairs (a fact to which it owes its name of diploeoccus), and is present occasionally in the nose, Eustachian tubes, and larynx of healthy individuals. Netter found it in 20 per cent. of the specimens of buccal secretion taken from well persons, and to the presence of this germ is to be ascribed the form of septicemia induced in animals by inoculation with saliva. It is present in about 80 per cent. of all instances of pneumonia, and in persons who have had the disease it is detectable for many months or even years. It is generally present in pure culture, but may be associated with pyogenic organisms. It is probable that Friedländer's bacillus (discovered in 1883) and other micro-organisms (Eberth's bacillus, streptococcus of erysipelas, bacillus pestis) may also have the power to cause the disease; and Wassermann[1] suggests that specific forms of pneumonia may coexist in the same individual, as, for example, lobar pneumonia and influenzal pneumonia. The organism grows upon all the culture-media except potato, between the temperatures of 24° and 42° C. (McFarland). The *micrococcus lanceolatus* (Fig. 13) can be readily demonstrated in the sputum by treating a cover-slip preparation "with glacial acetic acid. and then, after washing off the acid, dropping on anilin oil and gentian-violet, which is to be poured off and renewed two or three times."

The Pneumococcus in Other Diseases.—It has been found in pure culture in pleuritis (including empyema), pericarditis, meningitis, peritonitis, endocarditis, synovitis, bronchopneumonia (principally in adults), acute abscess, and other conditions.

The **mode of infection** is probably by inhalation. The first and chief effects of the germ are local—in the lung, though it may reach more distant portions of the body. To the widespread distribution of the pneumococcus is due, in part, the septicemic process sometimes observed.

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[1] *Deutsche medicinische Wochenschrift*, Leipzig, Nov. 23, 1893.

Usually, then, the disease is a local one at the start, but soon the toxins of the *micrococcus lanceolatus* become diffused throughout the system, producing a general disturbance. *Secondary infection* with other specific organisms (streptococci, staphylococci, etc.) commonly occurs in the various organs of the body.

Predisposing Causes.—(1) **Endemic Influence.**—That endemics of pneumonia, often of serious type, may occur in solitary buildings (barracks, tenement-houses, institutions, etc.) cannot be successfully denied, and we may attribute these outbreaks to defects in the local sanitary conditions.

(2) **Epidemic Influence.**—From time to time pneumonia prevails extensively, and appears to spread throughout a considerable percentage of the entire population of urban and rural districts. It may also originate in the endemic form in tenement-houses and institutions, and increase in its scope until it assumes an epidemic character. The epidemic form of pneumonia is at times confined to private homes (house epidemics), and in the winter of 1894 I saw, with Dr. W. K. Mattern of Philadelphia, 3 cases develop in rapid succession in one family. A Sister of Charity, after nursing two of the patients faithfully for a period of ten days, was also attacked and died of the disease. It is possible that the house-epidemic form may spread by *contagion*. An instructive epidemic is reported by W. B. Rodman, who states that 118 cases of pneumonia, with 25 deaths, occurred in a prison population of 735. B. Robinson[1] insists upon his view that pneumonia is contagious.

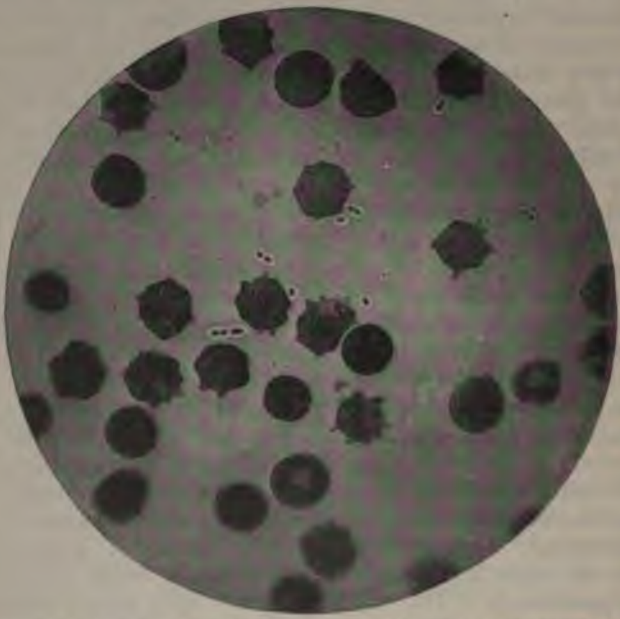

FIG. 13.—Diplococcus pneumoniæ, from the heart's blood of a rabbit; × 1000 (Fränkel and Pfeiffer).

(3) **Geographic Distribution.**—Pneumonia may be said to be an almost universally distributed affection. It prevails, however, more exten-

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[1] *Lancet*, April 16, 1898.

sively in certain countries than in others. Thus, Delafield[1] points out the fact (based on the eighth and ninth census reports) that in the United States the disease is of more frequent occurrence in the South than in the North. Climate, *per se*, does not, however, exercise a notable influence.

(4) **Season.**—Of 5905 cases collected by Seitz in Munich, 36.8 per cent. occurred in the spring, 32 per cent. in winter, 15.7 per cent. in autumn, and 15.3 per cent. in the summer. The period of maximum frequency of the affection in temperate climates is usually from the beginning of February to May, inclusive, and the next most frequent period is from December until February. In London most cases appear between the end of March and the end of June (Herringhan). The period of greatest frequency will be sometimes found to correspond in time with the period of the greatest vicissitudes of temperature and humidity.

(5) "**Catching cold**" is often followed by pneumonia, but frequently there is no such history. In this condition the mucosa of the respiratory passages is so altered as to become more susceptible to infection with the pneumococcus, and hence the so-called "cold" is a predisposing cause. Such facts as these also explain why pneumonia occurs with undue frequency in persons following certain occupations exposing them to those external influences that are apt to excite "cold."

(6) **Traumatism.**—Following injuries, especially of the chest, pneumonia occurs quite frequently. Contusions of the thorax lower the vital power and resistance of the tissues.

(7) **Age.**—Lobar pneumonia is common at all periods of life, and during the first two years of life it is not infrequent. Between two and twenty years of age there is less liability, and between twenty and forty it is again increased; while from forty to sixty years susceptibility again diminishes. After the latter period it augments rapidly.

(8) **Sex.**—Males are, on the whole, more commonly attacked than females, the discrepancy in the relative number of cases being greatest from the twentieth to the fiftieth years of age, and being due to the different degrees of liability to exposure in the two sexes.

(9) **Unhygienic Surroundings.**—The disease is more frequent among the lower than the higher classes—a fact due to the improved hygienic environment of the latter.

(10) **Circumstances connected with Individuals.**—The alcoholic is especially prone to this disease, any or all habits that tend to depress the nervous system acting as predisposing causes. Certain chronic diseases may exert an influence (chronic Bright's disease, organic heart-affections, carcinoma, diabetes, etc.); but, contrary to what is observed in other acute infectious diseases (typhoid fever in particular), susceptibility is not so great among immigrants and new-comers as among the natives and the older residents.

(11) **Prior Attacks.**—One attack undoubtedly leaves the system more susceptible to the disease, so that repeated attacks may occur in the same individual. And yet while it is true that persons have had numerous attacks—ten or more—this predisposing influence has probably been overestimated by most writers.

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[1] "Diseases of the Lungs," *American Text-Book of the Theory and Practice of Medicine*, Pepper, vol. ii. p. 540.

Immunity.—The results of the investigations of Behring and Kitasato with the blood-serum of animals which had been immunized against tetanus and diphtheria led Drs. G. and F. Klemperer to experiment upon the lower animals with Fränkel's diplococcus. They found that the rabbit could be rendered immune by intravenous or subcutaneous injections of large amounts of the fluid bouillon-cultures or of the glycerin-extract. From 10 to 20 c.c. of serum taken from a non-receptive animal were injected into the veins of an animal that was suffering from typical pneumonia (artificially produced), whereupon the symptoms subsided rapidly and the animal entered upon a speedy recovery. The same serum, used in a similar manner upon healthy receptive animals, rendered them non-receptive. The important truth that the serum of the blood of patients during convalescence from pneumonia contains an antitoxin which, when injected into the venous system of infected animals, is found potent to cut short the disease, has also been demonstrated by these observers. They have employed the blood-serum of pneumonic patients *after* the crisis, injecting it into other patients *before* the crisis with a view to inducing the latter, and success has attended their efforts in 6 cases. The question of serum-therapy for this important affection in man is not finally cleared up, and is still beset with difficulties; but that the pneumococcus engenders a virus—pneumotoxin—which produces elevation of temperature, etc. has been clearly demonstrated by the Klemperer brothers. Again, that this substance, acting upon the albuminous elements of the body, generates an antipneumotoxin which circulates in the blood and neutralizes the pneumotoxins as they are formed, inducing the crisis, has also been clearly proved. Antipneumotoxin, however, has not as yet been isolated.

Clinical History.—*Prodromes* are rare, and when present consist merely of a slight general indisposition, lasting a day or more. Rarely, there is cough, thoracic oppression, and slight chest-pains (simple bronchitis), that may or may not be connected with the pneumonic process. When this is the case, however, the invasion may be marked by sudden, great thoracic oppression or by a gradual development of the local and general symptoms.

Usually the *invasion* is *very abrupt*, and marked by a severe *rigor*, which has a duration of from half an hour to an hour, during which period the patient feels most uncomfortable, and is, indeed, very ill. The initial chill may occur at any hour of the day or night, the *fever* rising immediately and rapidly, and the temperature often mounting to 104° F. (40° C.) or even higher in the course of a few hours. The *skin* becomes harsh and dry, the face flushed, and the cheek on the side affected often shows a circumscribed deep-red spot. *Prostration* is pronounced, and headache and other nervous disturbances (restlessness, delirium) accompany and follow the ushering-in symptoms.

The *thoracic symptoms* follow closely upon the termination of the chill. Inspiration, particularly if deep, causes a stabbing *pain* in the affected side; the respirations are hurried, somewhat jerking and shallow (panting), while the pain persists, and later *dyspnea* may become marked, with accelerated breathing. *Cough* sets in early, and is dry and painful during the first day or even longer, and may be attended with expectoration, which generally presents a characteristic *rusty* or *blood-stained* *appearance.* The *physical signs* rarely appear before the end of the first day, and sometimes as late as the third (central pneumonia); in the latter form the local symptoms, as cough, dyspnea, and sometimes pain, are either wanting or feebly expressed during the first three or four days, and the clinical picture is composed of the general features only.

Anorexia is usually complete; *thirst* is excessive, and there may be *vomiting* at the onset, the bowels being generally constipated, though diarrhea may not infrequently be present. The patient in most instances lies upon the affected side until the pain has in great part subsided, and then he is apt to assume the dorsal position, exposing to full view an anxious countenance, with a characteristic *flush* upon the cheek, while the alæ nasi are seen to dilate forcibly during inspiration. Very frequently *herpes* on the lips or nose appears about this time, and forms a valuable diagnostic symptom. The nocturnal remissions are slight, the temperature being of the continued type, and the fever continues high—104° to 105° F. (40.5° C.)—for from five to ten days, and generally terminates by crisis. The pulse is somewhat quickened, but the *pulse-respiration* ratio is not maintained. The other general features last until the crisis occurs, or even increase in severity, but do not outlast this period; many of the local symptoms, however, and particularly pain, are greatly improved before the crisis is reached.

As will be seen hereafter, the general course of pneumonia is modified by a variety of interfering conditions that have relation to complications, individual circumstances, severity of the type, etc. In the instances in which the crisis is reached convalescence is rapidly established. The crisis may be accompanied by special symptoms, as copious sweating or diarrhea.

Leading Symptoms in Detail.—Local or Respiratory Symptoms.—Increased frequency of the respirations is a characteristic symptom, the rate varying from 40 to 60 per minute in adults, and in children from 60 to 90 or more. It is panting in character, particularly when pneumonia occurs in old subjects, and both inspiration and expiration are brief, though sometimes separated by a rather long pause. Expiration is usually accompanied by an audible "grunt," indicating great oppression, and while actual dyspnea is a frequent symptom, it may be absent or as the case progresses may become either increased or greatly diminished according to the severity of the type.

The *chief causes* of the rapid and labored breathing are the involvement of a large portion of the lung, associated general bronchitis, pericarditis or extensive pleurisy, cardiac failure, collateral congestion with edema, fever, and the intense pain in the side.

The *pulse-respiration ratio* is disturbed, the relation now being 1 to 2, or even 1 to 1.5, instead of 1 to 4, as in health (see Fig. 14).

Pain in the affected side is in most cases developed within a few hours after the initial chill, and after lasting two or three days gradually disappears. It is stabbing in character, and usually referred to the region immediately below the nipple or to the axilla, and rarely to other points (abdomen, flank). In most instances it is not severe until greatly intensified by the cough. which always aggravates this symptom, as does deep inspiration. The pain is due to implication of the pleura covering the inflamed lung, and may be entirely absent, especially in the aged and those showing marked toxemia.

The *cough*, like the chest-pain and respiration, is somewhat characteristic, being frequent, short, dry, and voluntarily repressed, because it is attended with increased suffering. Yet there are cases that run their entire course without cough, and this especially in the aged and in drunkards.

The Sputum.—At first mucoid and frothy, it soon becomes of a characteristic *rusty* color. It consists of a frothy, fluid mucus containing an abundance of small viscid masses of a yellowish- or reddish-brown color, from admixture of blood. The chief peculiarity of the sputum in fully developed cases is its *viscidity* and *tenacity*, often adhering to the receptacle even though the latter be inverted; owing to its adhesive quality it is ejected from the mouth with considerable difficulty by the patient. About the time of the crisis the sputum usually becomes more abundant, distinctly purulent, and its expulsion easy, but rarely it may be absent after the crisis. In severe types of the disease it may, at the outset, consist largely of pure blood, and in adynamic forms it is often thinner and darker in color (*prune-juice*). There are cases in which there is an abundance of muco-purulent expectoration when extensive associated bronchitis occurs, and, on the other hand, instances are met with in which nothing is expectorated save a little light-colored mucus. In old persons or in those previously enfeebled there may be no expectoration whatsoever. The amount is therefore exceedingly variable, not only in different cases, but also in different stages of the affection.

Under the microscope the sputum is seen to contain red blood-corpuscles, alveolar epithelium, the *Micrococcus lanceolatus* (usually with other micro-organisms), pus-corpuscles, and small fibrinous casts.

General Features.—The Fever.—As I have already stated, the fever rises rapidly during the initial chill, so that in eight to twelve hours the temperature reaches 104° or 105° F. (40.5° C.). It then remains high until the crisis, pursuing the continued type, with nocturnal remissions amounting to a degree or over, while the daily fluctuations correspond with the normal, except that they are now somewhat exaggerated. In children the rigor is almost always replaced by convulsions. The temperature has a lower average range in persons previously debilitated, in old people, and in drunkards, than in healthy adults and children. During the febrile period there may be observed a pronounced fall of temperature—pseudo-crisis—but the temperature again rises to its former height. This may occur quite early, though more often it precedes the true crisis by a day or two; and rarely it may take place repeatedly, and the temperature-curve bear a strong resemblance to the remittent or even the intermittent type, regardless of any malarial infection. The temperature may be unusually high, 106° F. (41.1° C.) or even 107° F. (41.6° C.), these striking elevations sometimes preceding the crisis (*perturbatio critica*), and hyperpyrexia may be the signal of approaching dissolution. It is especially characteristic of pneumonia, however, that the fever terminates by *crisis;* hence a mere glance at the temperature-chart may serve to complete the diagnosis in doubtful cases (see page 143). The crisis may occur anywhere from the end of the third to the fourteenth day, but in the majority of instances it is on the seventh or

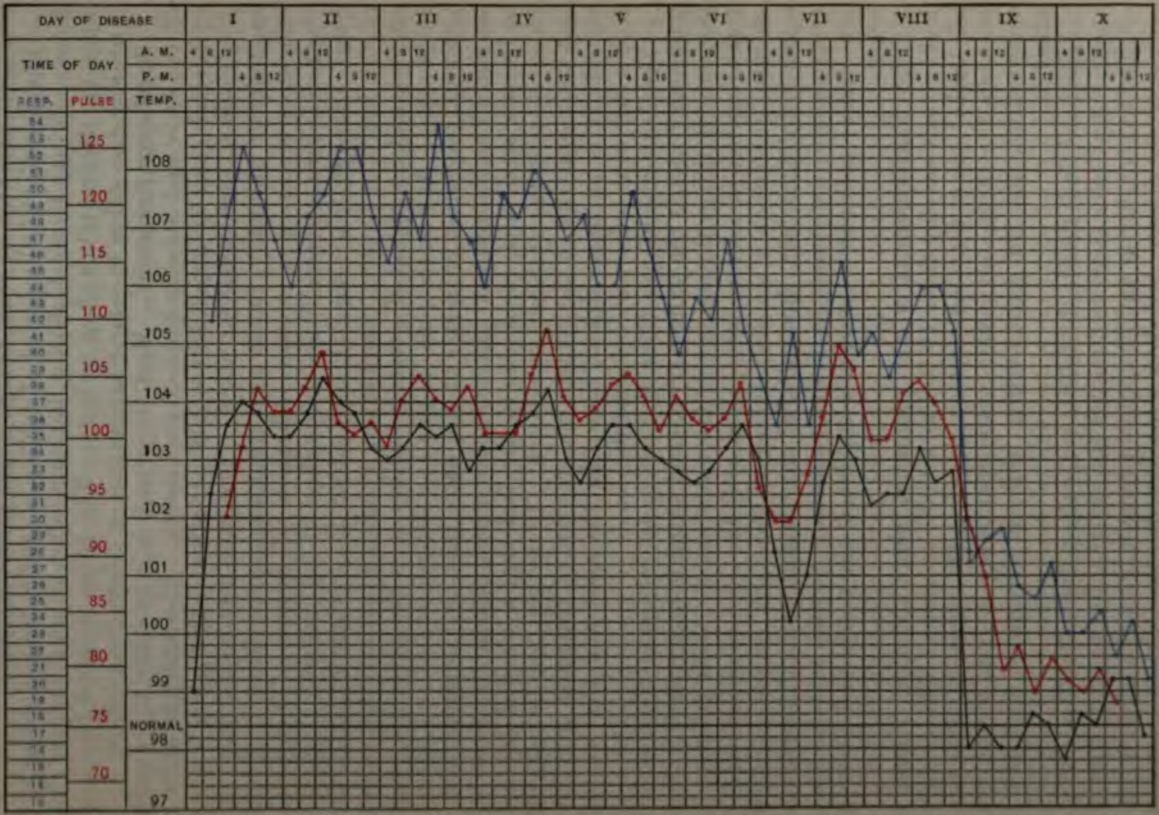

FIG. 14.—Chart of a case of lobar pneumonia with favorable course. A. T., aged thirty-two years; lower right lobe affected. Black, temperature; red, pulse; blue, respirations.

the ninth. The temperature usually falls during the night, and the drop is accompanied by copious perspiration, so that by the following morning the thermometer is found to register at the normal, or more often a subnormal, point (96–95° F.—35° C.). This fall in temperature may also be interrupted by fresh though slight exacerbations.

The *duration* of the period of decline is usually from eight to twelve hours. It may be much shorter, but more often is much longer, just as when the decline takes place by lysis. The latter mode of termination is usually due either to some complication or delayed resolution.

Circulatory Symptoms.—Most important is it to study the condition of the *heart* and *pulse* in cases of pneumonia. The average pulse-rate in typical cases is about 100 to 108 per minute, and when it exceeds 120 there is just cause for alarm. The rate may be increased either suddenly or gradually, but in any event augmented frequency implies danger, since it is a certain indication of failure of heart-power. Cardiac failure is generally due to the effect of the pneumotoxin upon the heart, although less commonly also either to previous organic disease of the heart, or to some complicating condition (pericarditis, collateral edema), and the period of greatest liability is in the advanced stage of the disease. At first the pulse is small; a little later full and bounding, although the latter character of the pulse may be associated with low tension (Van Santvoord). In extensive consolidation the pulse is apt to become small and rapid, due to the fact that a lessened amount of blood reaches the systemic circulation. Dicrotism may be noticeable, and an irregularity in the volume and rhythm of the pulse may be observed; it is an unpropitious sign. In the aged and the weakly, a feeble, frequent pulse may be present.

The *heart-sounds* are clear, and owing to increased tension in the pulmonary vessels the pulmonary second sound is accentuated. This is the state of things throughout in favorable cases. With failure of the right ventricle (a not rare event) there arise the signs of dilatation of this chamber (extension of cardiac dulness to the right, epigastric impulse, a low systolic murmur, shortening of the diastole, or fetal heart-sounds, cyanosis, and indistinctness of the pulmonary second sound). A soft, low-pitched murmur is sometimes audible in the mitral and pulmonary zones.

The *blood-appearances* are somewhat characteristic. There is a leukocytosis varying from 10,000 to 40,000 or more. The researches of Lache[1] show that leukocytosis is of some value in determining between the crisis and pseudo-crisis, continuing in spite of the fall of temperature, etc., in pseudo-crisis, while it disappears with the true crisis.

Stienon[2] finds that in the febrile stage the polynuclear forms predominate, but as soon as these diminish the eosinophiles begin to increase.

The red corpuscles and hemoglobin remain little changed during the course of the disease, but show a marked decrease almost immediately after the actual crisis.[3] Slight leukocytosis may indicate a mild infection, but as a rule it is a bad prognostic sign. Leukopenia occurs in the malignant cases; on the other hand, leukocytosis of high degree, while indicating a severe infection, "it, at the same time, shows a good reaction."[1] The blood-plates are also increased in number (Hayem). Da Costa has collected 9 cases of phlegmasia alba dolens in pneumonia.

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[1] *Berliner klin. Woch.*, 1898, Nos. 36 and 37.

[2] *La Presse méd.*, July 13, 1895.

[3] Sadler, *Fortschritte der Medicin*, 1892; Leichtenstein, *Ueber der Hämoglobin-gehalt des Blutes*, etc., Leipzig, 1892.

Cerebral Symptoms.—*Headache* sets in early and may be a prominent and persistent feature. In many cases, and particularly in children, the disease is ushered in by convulsions, this symptom occurring more often in the apical than in the basilar form of pneumonia. *Delirium* may come on during the acme of the disease (rarely, it may start as an acute mania), and may assume a maniacal form, but oftener in my experience consciousness has been retained. In the drunkard *delirium tremens* usually develops, and may anticipate the symptoms referable to the lungs; and I fully agree with Osler in stating that it should be an invariable rule, if fever be present, to examine the lungs in delirium tremens. These cases may often be appropriately termed "*walking pneumonia*," since they go about until excitement gives way to a coma that deepens into death. In *adynamic forms* a low, muttering delirium is frequent and is sometimes accompanied with more or less coma.

In the so-called *cerebral* pneumonia the nervous phenomena are quite pronounced, and simulate closely cases of cortical meningitis. It is often associated with excessively high fever, except in the aged, when the cerebral symptoms are also well marked, but the fever is moderate. Most writers contend that apical pneumonias are apt to assume the cerebral type, but, according to my own experience, this dictum is correct as relating to children only. Double pneumonias are commonly characterized by severe cerebral symptoms, yet I have seen instances in the adult without unusual nervous phenomena.

The Cutaneous Symptoms.—As stated before, herpes is common and its diagnostic importance is considerable. *Naso-labial herpes* is but little less frequent in this disease than in malaria, being present in about one-third of the cases. It usually comes out from the second to the fifth day of the disease, and rarely may appear upon the cheek, lobe of the ear, the genitals, forearm, or upon the mucosa of the tongue. Sweats are not common during the height of the disease, but usually accompany defervescence, when they may be copious. The deep-red circumscribed spot upon one cheek (*mahogany flush*), usually on the side of the affected lung, has already been mentioned. Urticaria has been observed, though rarely.

Digestive System.—The mucous membrane of the mouth is dry, the tongue has a coating of a yellowish-white color, becoming dry and brown in cases representing a low form, and anorexia and thirst are present. *Vomiting* is not uncommon at the outset, and may be repeated, while constipation is the general rule and diarrhea the frequent exception. The above symptoms spring from the marked fever. Splenic enlargement of slight degree can usually be detected on palpation, but the liver is not perceptibly increased in size.

Urinary Symptoms.—The urine is febrile, diminished in amount, and high-colored, the urea and uric acid being greatly in excess. On the other hand, the chlorids are, according to the older authors, either diminished in amount or absent during the febrile stage, presumably for the reason that they pass into the inflamed lung-tissue. They are not, however, constantly absent, and sometimes they are not even lessened, in pneumonia; moreover, their disappearance is not peculiar to this disease. The above-mentioned facts justify two important inferences: (1) The absence of chlorids is a symptom of little diagnostic value; and (2) their reappearance in the urine toward the close of pneumonia is of small prognostic worth. Slight (febrile) albuminuria is common.

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[1] E. Becker, *Deutsche med. Woch.*, Aug. 30, 1900.

Physical Signs.—Stage of Congestion.—The density of the lung is increased, but the involved tissue is not consolidated and the pleura is not yet covered with fibrin.

Inspection.—The movements of the affected side (especially if the base be involved) are defective, the degree of expansion being much diminished. In double pneumonia the costal type of breathing, combined with a vigorous play of the abdominal muscles, is observed.

Palpation.—There is a slight increase in the tactile fremitus over the congested area.

Percussion.—The note may be normal, though more often it is briefer, higher-pitched, or even distinctly tympanitic.

Auscultation.—The breath-sounds are weak, and sometimes become broncho-vesicular upon deep inspiration, while over the unaffected lung-tissue they are exaggerated. If, as often happens, inflammatory products due to associated bronchitis occupy the small bronchi, subcrepitant râles may be audible. The crepitant râle, however, is rarely heard until the close of the first stage or until fibrin coats the pleural surfaces, and I cannot agree with the view of certain authorities who claim that it is of vesicular origin.

Stage of Consolidation.—*Inspection.*—There is little or no expansive motion of the chest over the affected area, while upon the unaffected side it is increased. The volume of the thorax on the diseased side is increased, as shown by mensuration, but the intercostal depressions are not effaced.

Palpation renders clearly perceptible the defect or absence of expansion. Vocal fremitus is usually much increased, though in exceptional instances it is diminished or absent—a circumstance which can, as a rule, though not invariably, be attributed to an associated pleurisy with more or less effusion. Frequently a friction-rub is felt before complete consolidation is established.

Percussion.—Varying degrees of dulness are obtained in this stage, and before the lung-tissue becomes thoroughly solidified the note may have a tympanitic quality. After complete consolidation there is usually marked or absolute dulness posteriorly, unchanged by full inspiration, while the note may be more or less tympanitic anteriorly, where the vibrations are more apt to reach the air in the larger bronchi. A sense of resistance is offered to the pleximeter-finger, but not to the same degree as in the case of a pleurisy with effusion. When the latter condition is associated and in massive pneumonia the percussion-note will be flat. Deadness is less marked in old people in whose ribs senile changes have taken place, which render them more resonant, or in cases in which the consolidated areas occupy the central portions of the lung. Above the solidified part Skodaic resonance is usually obtainable.

Auscultation.—Bronchial or tubular breathing is heard, as a rule, over the solidified lung, but it may be absent in consequence of the plugging of the large bronchi with exudate (so-called *massive pneumonia*). Bronchophony is usually obtainable over the portion of the lung affected, though this may also be absent, and for the same reason as in the case of the bronchial breathing: it sometimes takes the form of egophony. Subcrepitant râles, due to associated bronchitis, are sometimes heard with unusual distinctness (owing to the consolidation), and the crepitant râle at the end of inspiration is best heard at the beginning of consolidation, when the pleura receives its coat of fibrin and while the lung is yet capable of sufficient movement to produce fine pleural friction. A distinct friction-rub may also be heard occasionally.

Stage of Gray Hepatization.—With beginning resolution the solid contents of the air-cells liquefy and are removed, so that air now re-enters the air-cells and permits a consequent increase in the movement of the lung.

Inspection.—The normal expansile movement of the affected side gradually returns.

Palpation.—Tactile fremitus progressively diminishes.

Percussion.—The dull or tympanitic quality of the note is gradually lost, though the fact must be emphasized that the abnormalities in the note vanish more slowly than the other abnormal physical signs. Some degree of deadness often remains long after apparent recovery.

Auscultation.—With increased movement of the lung there may be a reappearance of the crepitant râle, due to interplay of the pleural surfaces, and the softened exudate in the air-cells gives rise to subcrepitant râles, heard both on inspiration and expiration, with coarser râles over the bronchi. Bronchial breathing gradually gives place to broncho-vesicular, and the latter in turn to normal breathing.

The Pneumococcus Septicemia.—The pneumococcus infection may cause severe toxic features and even speedy death without any, or with but little, involvement of the lung-texture. The general invasion symptoms, such as the chill, high fever, and nervous symptoms which always predominate, however, are present and persist until death ends all. Death is preceded by signs of cardiac failure or more rarely by coma. In some of these cases localization of the morbid process may occur in organs other than the lungs, as the cerebral meninges, the endocardium, pericardium, and the pleura. An assured diagnosis in these atypical forms of the pneumococcus infection can be arrived at by a bacteriologic examination of the exudate obtained by aspiration.

Complications.—Doubtless many of these are due to the primary infection by diplococci.

Pleurisy is, of necessity, associated in all instances in which the consolidation reaches the pleura. It is to be looked upon as a direct result of the pneumonic process, since in a great proportion of cases examined the presence of the diplococci has been demonstrated. Cases are met with, however, in which the truly pneumonic symptoms are overshadowed by the intensity of the pleuritis, and to these the term *pleuro-pneumonia* has been applied. In this form there is often a copious effusion which is exceedingly rich in fibrin—a circumstance which distinguishes it from other forms of acute pleurisy. There may be the ordinary grade of pleurisy on the side affected by the pneumonia, and a severe grade on the opposite side, and when effusion occurs under the latter exceptional conditions it is apt to be purulent. Indeed, empyema has of late been shown to be a frequent complication of pneumonia, but, as far as my own observation goes, it would appear to rank as a sequel rather than a complication, coming on as it usually does several days after the crisis. Its development is accompanied by replacement of ordinary dulness by flatness with great resistance, and by the disappearance of râles and breath-sounds, normal and abnormal. Other characteristic features of empyema are present, but in the event of doubt surrounding the diagnosis the needle should be introduced.

There is a prompt rise of fever, the temperature leaping to 103° or 104° F. (40° C.) quickly, after which it is decidedly remittent in type, but there are no hectic chills. Fistulous connection with a bronchus, however, and the establishment of *empyema necessitatis* are common events in this form of the disease, and may be preceded by diurnal chills, sweats, etc.

The occurrence of *septic phenomena* is a certain indication of secondary infection by streptococci. The pus is absorbed very rarely, and more frequently becomes encysted. I saw one instance in which the effusion measured 8 liters, while ordinarily the amount ranges from 2 to 5 liters. Removal of the effusion by aspiration is promptly followed by the disappearance of the fever, but reaccumulation generally occurs.

Finally, if defervescence takes place by lysis or if the "critical" decline is absent, a residual purulent or sero-fibrinous effusion may be considered as the likely cause. This latter complication is attended by a paroxysmal cough which is excited by movement, and is not usually accompanied by expectoration, while the temperature rises, though not so high as when the effusion is purulent.

Acute general bronchitis may pre-exist or may arise as a complication, and often proves formidable, intensifying the fever and increasing the dyspnea, the tendency to heart-failure, and the cyanosis. The expectoration of mucus is freer than in uncomplicated pneumonia, and over the bronchi moist râles intermingled with sibilant and sonorous râles are audible.

Pericarditis.—This is one of the most important complicating affections. It results from a direct extension of the adjacent pleuritis, and hence is more common in left- than in right-sided pneumonia, and children are more prone to it than adults. Although generally of the plastic variety, it is not infrequently sero-fibrinous, and rarely the effusion is purulent. The *diagnosis* can be made in the same way as when other conditions attend its development, but it may be readily overlooked by the careless observer. I would say, however, that the occurrence of increased dyspnea, with or without precordial pain, should serve as a signal and lead to a physical examination.

Endocarditis.—This is far more frequent than pericarditis, and particularly in the ulcerative form. Out of 209 cases of malignant endocarditis collected by Osler, 54 cases occurred in pneumonia, and my experience fully agrees with his statement as to its comparative frequency in this affection. There are no reliable symptomatic indications of this condition, and of those symptoms that do appear the physical signs are least trustworthy. Frequently murmurs are entirely absent; and, on the other hand, the presence of a murmur, even though it be loud and harsh, is by no means diagnostic of the complication. Some claim that a rough diastolic murmur is quite significant; this has not, however, been present in any of my cases. The development of septic manifestations, especially irregular fever, chills, and sweats, renders the case highly suspicious, and when in addition there arises distinct evidence of embolic processes the diagnosis becomes highly probable. If, now, the symptoms of meningitis supervene, little doubt remains as to the character of the complications, since meningitis and endocarditis are often combined in pneumonia.

Netter, Weichselbaum, and Bignami have shown by microscopic examination and cultivation that acute endocarditis may be caused directly by the diplococcus of pneumonia, and, from the presence of this bacillus in the right ventricle, that it is far commoner than the forms due to other causes.[1]

Chronic Endocarditis.—This predisposes to acute endocarditis, both simple and ulcerative, but, independently of the acute form, pneumonia arising in the course of chronic endocarditis is apt to be attended by cardiac failure, with venous stasis as the consequence. The murmurs of chronic valvular disease often disappear with the development of pneumonia.

Cardiac clots (ante-mortem) may form, but are rare. They result from weakness of the ventricular wall, especially in the right heart; and are most apt to arise, therefore, in cases in which the preagonal period is much prolonged. Venous thrombosis is rarely seen, and embolism of the larger arteries is a rare complication. Cerebral embolism, causing aphasia and even hemiplegia, has been observed, but seldom.

Acute purulent meningitis is a comparatively rare but very grave complication. It is often related etiologically to pneumonia, and its symptoms are not clearly defined; particularly is this true when it develops during the invasion-period and the basilar meninges are not involved. Hence its diagnosis is often impossible. The presence of intense and persistent headache, rigidity of the nucha, wild delirium, followed by stupor deepening into profound coma, affords a basis for a probable diagnosis. Its frequent association with ulcerative endocarditis has been pointed out above.

Peripheral neuritis is among the rare complications of this disease.

Parotitis is also sometimes seen, and may cause a fatal termination of the case. I have seen two instances, however, in which this was a complication, and both ended in recovery. It is thought to be associated usually with endocarditis, but not so in my cases.

Arthritis.—This may closely simulate rheumatism at the start. It soon, however, takes the form of a purulent arthritis, and may be associated with other suppurative inflammations (meningitis, endocarditis). The pneumococci have been found in the affected articulation.[2] *Rheumatism* may be rarely associated also, particularly in children.

Gastro-intestinal Complications.—*Croupous gastritis* may rarely intervene, but *croupous colitis* is a more frequent concomitant, giving rise to tympanites and marked diarrhea, and it may prove a serious condition.

Peritonitis occurs, but with great rarity.

Jaundice may be observed in all types of the affection, though, on the whole, it is more frequent in serious than in mild forms of the disease. It is rarely intense, and has no symptoms as a rule; it is most probably an obstructive (hepatogenous) jaundice. I have observed cases in which the evidence of a certain degree of obstruction was unmistakable. N. V. Pétrov has reported 13 cases complicated with icterus, and in all observed local lesions (mainly catarrhal) of the duodenum and the biliary canals.

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[1] *Practitioner*, London, Aug., 1894.

[2] Bernheim, *La Médecine moderne*, Paris, Feb. 21, 1894.

Acute nephritis, generally of a mild grade, is a rare sequel or complication, and its recognition is entirely dependent upon the discovery of albumin and casts in the urine.

Clinical Varieties and Anomalous Types.—(1) **Typhoid Pneumonia.**—This relates to an adynamic, serious type of the disease with *typhoid symptoms*, and not to typhoid fever. It is often secondary to low fevers, to septicemia, diabetes, and chronic nephritis, and is also the variety met with in drunkards and in persons previously enfeebled. The onset is somewhat gradual. The physical signs may be ill defined, but the general features are always striking and characteristic. Prostration is extreme; there are delirium and often stupor; the temperature may or may not be high; while the respirations and pulse are almost always frequent. The skin is dry, and may show a dusky tint or slight jaundice. The tongue is dry, often brown, and vomiting is common; the sputa may be rusty or decidedly hemorrhagic. Splenic enlargement is often clearly perceptible. When recovery ensues convalescence is tedious.

(2) **Epidemic Pneumonia.**—This is often of malignant type. The symptoms exhibit noticeable variations, according to the special etiology and to different epidemics. The pneumonias of *epidemic influenza* are complicated with or preceded by general bronchitis. The heart-power often becomes exhausted early, and then follow congestion and edema of the lungs. The physical signs are often slight.

The so-called *serous pneumonia* often complicates influenza; it is ascribed to streptococcus infection (*streptococcus pneumonia*). Septic phenomena often arise, such as the accustomed irregular fever and sweats. The physical signs, for a time indefinite, when fully developed, resemble those of bronchopneumonia. There may be a tendency to migration from one to the other lung, as in a case under my immediate observation. There may be a late-appearing rusty expectoration, and in some cases the sputa are muco-purulent throughout. If death does not supervene, the course is protracted, and the fever may terminate by lysis. In two of my cases the pneumococcus was detected in the sputum in increased numbers. It is said that in mixed infection the micrococcus lanceolatus is abundantly present. In so-called "*larval pneumonia*" the general symptoms are mild and the local signs ill defined. The epidemic outbreaks that occur in institutions, tenement-houses, jails, etc., belong to this variety.

(3) **Latent Pneumonia.**—To this class belong *central pneumonias* (*vide* General Symptomatology). In these instances the sputum is to be stained and examined microscopically, when the pneumococcus will be found. When pneumonia arises in the course of emphysema a physical examination often gives negative results, the dilated air-cells not being filled with the exudate: hence dulness is less marked, and tubular breathing is often absent. The sputum is gummous and rusty, as a rule. Before the crisis occurs consolidation usually advances to the periphery.

(4) **Migratory Pneumonia.**—By this is meant an extension of the specific inflammation to other parts of the lungs. Such extension may prevent the occurrence of the usual crisis, and often occasions an exacerbation of the general pneumonic features.

(5) **Bilious Pneumonia** ("*Malarial Pneumonia*").—When lobar pneumonia occurs in persons who are subjects of malarial poisoning, the initial chill is prolonged and the fever paroxysmal or remittent. Jaundice and vomiting are common.

(6) In **children**, the first symptom is often a convulsion. Cerebral symptoms (delirium, stupor, coma) may appear early. The upper lobes of the lungs are frequently involved. Unless the objective indications be examined for, the disease is frequently overlooked. The characteristic sputum is rarely seen in juvenile pneumonia.

(7) In **old persons** pneumonia runs a peculiar course and is dangerous. The initial chill is often absent or replaced by moments of chilliness. There may be nausea and vomiting. Prostration is profound; there is fever, but it does not range high and is apt to be irregular. Nervous phenomena, sometimes prominent, are not uncommon, but the local symptoms (cough, expectoration, and pain) are mild or wholly absent. The physical signs are defective owing to impairment of the respiratory movements; dulness on percussion (with a tympanitic quality), tubular breathing, and a few subcrepitant râles may, however, be noted. This affection, as it occurs in old people, is a most deceptive one, the cases generally ending fatally after an illness of an apparently mild degree of intensity.

(8) **Ether-pneumonia.**—Opinions are divided as to the frequency of occurrence of pneumonia after ether-narcosis. The aggregate number of cases from all sources (57,842) gives a percentage of 0.07. My own statistics, embracing 12,842 cases, give a percentage of 0.23.[1]

Among the principal causes are—(*a*) Season. According to my investigations, over 80 per cent. of the cases occur during the winter and spring months. The patient is sometimes carried from a heated operating-theatre through a cold corridor to a room or ward with a lower temperature. (*b*) "Catching cold," or exposure, as may obtain during protracted operations. (*c*) Bronchitis, coryza, or other morbid state of the respiratory mucosa at the time of anesthesia. (*d*) Dried secretions or incrustations of foreign matter that are loosened by the ether and drawn downward into the lungs, particularly if the head be not kept comparatively low and turned from time to time. (*e*) Abdominal operations give the highest percentage of cases of ether-pneumonia, and my studies show that this is partly due to the more protracted etherization, thus rendering the bronchi more susceptible. Moreover, coughing excites great pain, and hence is restrained, with retention of the bronchial secretions.

The *clinical features* are aptly compared with those of secondary pneumonia (*vide* p. 160). The *diagnosis* rests principally upon the typical physical signs. Owing to the extreme latency of the condition, and the danger that the symptoms may be regarded as being septic in nature, I would emphasize the importance of a physical examination of the thorax upon the sudden accession of *fever*, particularly if associated with thoracic *pain*, however slight, following an operation.

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[1] "Ether-pneumonia," *University Med. Mag.*, August, 1898.

Relapses.—These are among the rarest of events, and are usually rudimentary. *Recurrences* are ordinary, however, second, third, fourth, and even more attacks occurring in the same individual.

Course and Duration.—In cases that recover the febrile stage lasts from three to thirty or more days. In most instances, however, defervescence occurs on the seventh or ninth day, and resolution is completed about one week later, making the total duration from twelve days to two or three weeks. Convalescence may be delayed when complications outlast the primary disease or when sequelæ arise, and fatal cases are apt to terminate on the seventh, eighth, or tenth day of the disease. The course of pneumonia is often greatly modified by complications.

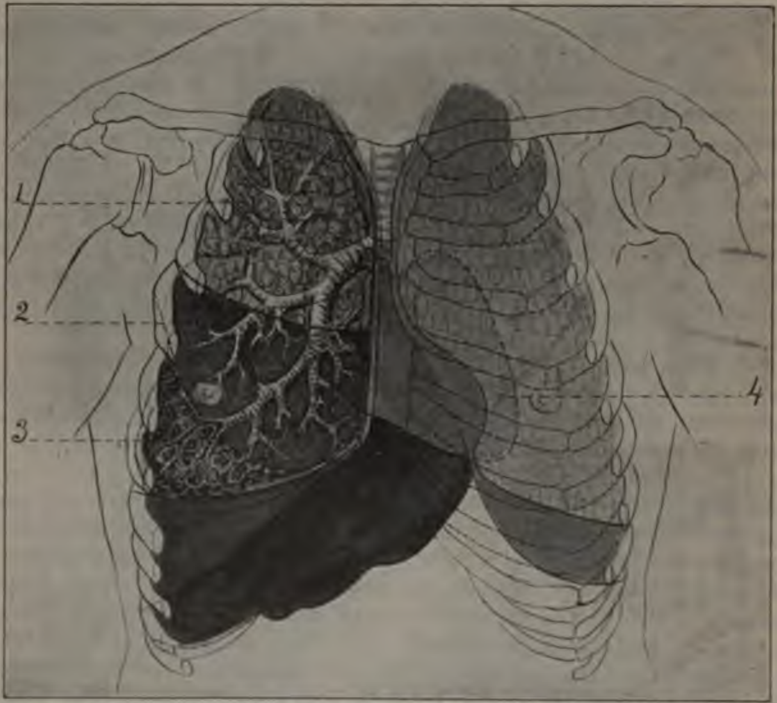

FIG. 15.—Lobar pneumonia: 1, unaffected area (upper lobe); 2, consolidated area (middle lobe): 3, resolving area (lower lobe); 4, heart in normal position.

Termination.—(*a*) **Delayed Resolution.**—The process of resolution may not begin until the fourth, sixth, or even tenth week. Usually defervescence by crisis has taken place long before the physical signs indicate resolution; the fever may, however, fall by lysis. When resolution occurs it may lead to complete restoration of the functional and anatomic entirety of the lung-tissue, or very rarely proliferation of the interstitial connective tissue may arise during the period of postponed resolution, producing (*b*) **chronic interstitial pneumonia.** (*c*) **Abscess**; (*d*) **gangrene**; and (*e*) **tubercular phthisis** are also sequelæ. For their clinical description, *vide* Diseases of the Lung; also p. 288.

T. A. Bowes and H. M. Fischer each give notes of a case of gangrene that recovered.

Diagnosis.—The diagnosis is determined by special local and general symptoms, together with the physical signs. Of these, the abrupt onset with rigor, the course of the fever with termination by crisis, the stabbing chest-pains, the dyspnea, the peculiar type of breathing, the abnormal pulse-respiration ratio, the cough, the rusty expectoration, and the signs of lobar consolidation, are the most characteristic. Repeated physical examinations of the chest will often detect consolidation, in the absence of the accustomed symptoms. Again, when in the course of certain chronic affections (cancer, Bright's disease, diabetes, and organic affections of the heart) fever is developed, physical exploration of the thorax is imperatively demanded.

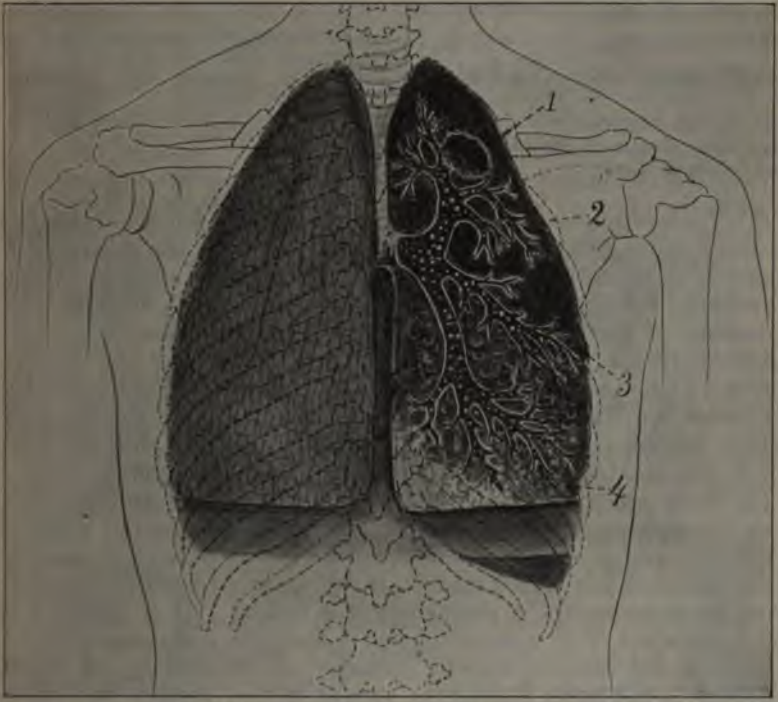

FIG. 16.—Acute pneumonic phthisis, posterior view: 1, cavity; 2 and 3, consolidation; 4, infiltration; the white spots indicate râles.

Differential Diagnosis.—This relates to (*a*) acute pneumonic phthisis, (*b*) pneumo-typhoid, (*c*) meningitis, (*d*) broncho-pneumonia, (*e*) acute pleurisy with effusion.

(*a*) PRIMARY LOBAR PNEUMONIA.	ACUTE PNEUMONIC PHTHISIS.
There may have been prior attacks.	Inherited predisposition or previous tuberculous disease.
Sudden, with severe rigor and rapid rise of temperature.	Generally more gradual—repeated fits of chilliness (rarely severe rigor), often following exposure or "cold."
Fever of continued type, terminating by crisis.	Fever of remittent type, often becoming intermittent, without crisis.

(*a*) PRIMARY LOBAR PNEUMONIA.	ACUTE PNEUMONIC PHTHISIS.
No drenching sweats, except at time of crisis.	Drenching sweats present and oft repeated.
Herpes common.	Absent.
Not much emaciation.	Rapid emaciation.
Pulse-respiration ratio considerably disturbed.	Less so.
Sputum rusty-colored, viscid, and sticky; contains pneumococcus.	Sputum may be blood-tinged; is more purulent and copious, and contains numerous bacilli and yellow elastic tissue.
Duration of febrile stage shorter.	Duration longer.
Physical signs, as a rule, first referable to base of lung.	First referable to apex.
Usually limited to one lobe or the lower portion of one lung.	Usually extension from apex to base.
Signs of consolidation, followed by resolution.	Signs of consolidation, followed by cavity-formation, with large gurgling râles at apex.
Apex of opposite lung not involved.	Apex of opposite side generally invaded.
Prognosis not hopeless.	Hopeless.
Tuberculous disease of other organs does not follow as a rule.	Often does.

(*b*) *Typhoid pneumonia* must be diagnosed from *pneumo-typhoid*, and the blood in the two conditions may be of service in the discrimination. Leukocytosis usually exists in pneumonia, and there is hypoleukocytosis in typhoid; but this fact is only of value when there is marked increase or decrease of the leukocytes, since figures about normal may occur in either condition. In pneumo-typhoid, after the end of the first week, however, undoubted symptoms of typhoid fever arise, and often before this period the Widal test will clear the diagnosis. On the other hand, typhoid pneumonia is characterized especially by great physical prostration, feeble heart-action, and other symptoms of the typhoid state.

(*c*) *Meningitis* is sometimes mistaken for pneumonia, and particularly when the latter occurs in children. The initial symptom of pneumonia in the very young is often a convulsion; whereas, though in meningitis this symptom is not uncommon, it is more apt to manifest itself later. When headache occurs in pneumonia it is frontal. It is almost invariably complained of in meningitis, but is occipital, and is associated with rigidity of the cervical muscles. Before the occurrence of pressure-symptoms in the latter disease the patient is very restless and morose; his reflexes are exaggerated and there is marked hyperesthesia. The temperature-range is lower, more irregular, and there is no crisis, while the pulse is more variable and often irregular in meningitis. In pneumonia with latent local symptoms the pulse-respiration ratio is greatly altered and the type of respiration peculiar (*vide ante*). The important rule, to examine for the physical signs in doubtful cases, must not be neglected, and if the subject be young the apex region in particular.

The differential diagnosis between pneumonia and broncho-pneumonia and pleurisy with effusion will be found on pages 523 and 557.

Prognosis.—The mortality from pneumonia in hospitals averages about 25 per cent. It is less in private practice—about 15 per cent. The death-rate, however, is greatly modified by the type of the individual epidemic, and by so many conditions and incidents that a precise statement as to the percentage of fatal cases cannot be ventured. The above mortality-rates have been based upon all of the accessible statistics at my command. Wells collected 223,730 cases, which gave a mortality of 18.1 per cent.

The elements that enter into a correct prognosis are in the main identical with those in other acute infectious diseases, and concern (1) the severity of the type of infection, (2) the presence or absence of complications, and (3) circumstances peculiar to the individual.

(1) **Severity of the Type of Infection.**—In sthenic cases this is shown by (*a*) the temperature-range, (*b*) the degree of heart-power, (*c*) the intensity of the nervous symptoms, and to some extent by (*d*) the size of the area of lung-induration. It is a matter of common observation that the absence of leukocytosis is indicative of a grave type. In case the diplococcus be found in the blood, the prognosis must be considered very grave, as it has never been found there during life, except in cases that are in themselves very grave or seriously complicated. A continuance of marked leukocytosis with a drop in temperature would point to a pseudo-crisis. (*a*) *The Temperature-range.*—A continued high temperature, as, for example, 105° F. (40.5° C.), on two or three consecutive days without material remissions, is ominous. (*b*) *The Degree of Heart-power.*—A steadily rising pulse-rate after the fifth day indicates real danger, since it points indisputably to gradual cardiac failure. The same thing is shown by a diminution in the intensity of the second pulmonary sound, particularly the giving out of the right ventricle. (*c*) *The Intensity of the Nervous Symptoms.*—Active delirium is not favorable at any stage, and is particularly unfavorable if it develop early. When it assumes the form of delirium tremens the case has usually passed beyond the hope of recovery. (*d*) *The Size of the Area of Lung-induration.*—I have observed that extension of the consolidation at an advanced stage belongs to serious types. The same may be said of double basic pneumonias.

Typhoid pneumonia, being of low type, gives an unfavorable prognosis, notwithstanding an absence of high temperature and of extensive inflammation of the lung-texture.

(2) **Presence or Absence of Complications.**—Cases in which there is involvement of a single lobe or two lobes, if it occur on the right side and without complications, generally terminate in recovery. In nearly one-half of the instances complications occur, and these greatly increase the death-rate. Among the most common is *pleurisy*, which, unless accompanied by considerable effusion, does not add fresh danger; when pleurisy attacks the non-affected side, however, it does. *Empyema*, following pneumonia, generally terminates in recovery unless septic phenomena are superadded. Extensive *bronchitis* is a most perilous complication in my judgment. *Pericarditis* decreases the chances for recovery, but by no means to the same extent as *endocarditis*. *Cardiac clots* may form, but usually the patient is already moribund. *Abscess of the lung* and *gangrene* form highly unfavorable complications. *Congestion* and *edema* of the uninvaded portions of the lungs render the outlook bad, and these, together with cyanosis, are apt to be dependent upon failure of the right heart. *Acute meningitis* is exceedingly grave. Fenwick, as the result of an analysis of 10,000 cases, found that the

quantity of albumin in the urine is of considerable prognostic value. *Gastro-intestinal* complications occurring at the outset are unpropitious.

(3) **Circumstances connected with the Individual.**—Of these *age* heads the list, and after the twentieth year the mortality increases progressively until the seventh decade. It has been claimed that nine-tenths of the deaths after the seventy-fifth year are from lobar pneumonia. Under the twentieth year, according to the analysis of 708 cases at St. Thomas's Hospital by Hadden, H. W. G. Mackenzie, and W. W. Ord, the mortality is 3.7 per cent.

Sex has little influence, though the disease is believed by some to be more fatal in females than in males. The *alcoholic* rarely escapes death.

Course.—In common with other clinicians, I have been impressed with the increased proportion of cases showing an irregular course, and I ascribe this change, in part at least, to influenza.

Modes of Death.—Death is commonly due to heart-failure, which results from two causes: (1) overwork, as when an extensive area of lung-tissue is involved (rarely); and (2) the direct effect of the pneumotoxin upon the heart (generally). The *complications* mentioned may prove fatal, however, and in one of my own cases thrombosis of the coronary artery killed the patient. This may be a not uncommon terminal condition.

Treatment.—**General Management.**—The patient should occupy a well-aired apartment, at a temperature of 65° F. (18.3° C.). Spolverini[1] points out that the pneumococcus in the sputum may remain virulent from fifty-five to one hundred and forty days, hence it is important to sterilize pneumonic sputum. The patient should not be allowed to leave his bed for at least one week after the occurrence of the crisis; and as pneumonia is a self-limiting affection, the principal object is to support the powers of life until the crisis is passed. *To this end nothing contributes so much as proper feeding.*

The **diet** should be light, chiefly liquid, but of the most nutritious sort. Alimentation should be especially vigorous when there is the slightest tendency to increasing debility. *Milk* should constitute the chief article of diet; meat-broths or meat-juices, egg-white, and light farinaceous substances may also be allowed. The food, particularly the milk, is to be administered at stated brief intervals and in definite quantities. When resolution is delayed stronger forms of nourishment (scraped meat, etc.) may be given. After the crisis a gradual return may be made to the usual forms of solid foods. Alcohol has considerable food-value, it lessens waste and improves the appetite and digestion. To accomplish this object, it should be given in small amounts, two to three ounces daily.

Cardiac stimulants are often indicated. It is well to begin their use as soon as the slightest tendency to cardiac failure is shown. The evidences of the latter conditions are to be found in the state of the pulse, the first sound of the heart, and the pulmonic second sound. When the pulse becomes more accelerated and feeble, the first sound of the heart less distinct, and the pulmonic second sound loses its accentuated character, or marked nervous symptoms appear, then alcoholics must be used. At first they are to be employed in moderate doses (½ ounce—16.0—of whiskey or brandy every three hours), to be increased if the effect be favorable proportionate to the urgency of the indication. There is a great tolerance of alcohol in this disease. In the pneumonia of drunkards its early and free use is to be recommended.

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[1] *Centralb. f. allg. Path. u. pathol. Anat.*, July 18, 1900.

If the alcoholic stimulants fail, other cardiants must be administered simultaneously. Of these, strychnin has been the most serviceable in my own hands, its mode of administration following the same rules as have been mentioned for alcohol—at first in moderate-sized doses, to be increased as occasion demands. Should urgent need of stimulation arise, however, either suddenly or more gradually, strychnin should be exhibited hypodermically. It is my custom in desperate cases to use subcutaneously as much as gr. $\frac{1}{15}$ (0.0043) every two or three hours. So soon as the condition of the heart denotes restoration of cardiac power the size of the dose is to be reduced, but the agent is not to be withdrawn until the disease has run its course. In no other disease does strychnin possess greater potency for good than in pneumonia, if wisely employed. For sudden heart-failure ether, administered hypodermically, is also very efficacious, and digitalis may be combined with the strychnin or given separately. In severe forms of pneumonia digitalis is invaluable during the advanced stages; it may be given in doses ranging from 5 to 15 minims (0.333–0.666) of the tincture every third hour. In cases in which extreme cardiac weakness with depression of respiratory forces supervenes the drug is to be administered hypodermically in the same dosage. Strychnin may be combined with the digitalis. The effect upon the pulse and heart-sounds should be the criterion of sufficiency. Recent experience enables me to speak strongly in favor of atropin administered subcutaneously in the threatened collapse that sometimes attends the crisis. Nitroglycerin is especially indicated when the renal secretion is scanty and the urine contains more than the usual trace of albumin. Ammonium, in the form of the aromatic spirits or the carbonate, is an excellent stimulant to the feeble heart of pneumonia.

Saline injections, given either intravenously or subcutaneously, increase the blood-pressure, and hence are valuable in overcoming circulatory depression, which is often threatening to life. The hypodermic method is preferable. From 1 to 2 pints (strength 0.7) may be injected, and allowed to flow under the skin from a rubber bag; and this may be repeated at intervals of eight to twelve hours if necessary.

Respiratory Stimulants.—Beginning cyanosis is the signal for the use of respiratory stimulants, of which the best are strychnin and atropin, and they should be given hypodermically. *Oxygen*, if given freely, often serves to tide over periods of marked cyanosis. The gas should be inhaled directly from the cylinder until relief is afforded, when it may be allowed to escape close to the patient's nose, or it may be taken from the nozzle every alternate ten minutes.

Hydrotherapy.—In meeting high temperature, marked nervous symptoms, dyspnea, cardiac weakness, etc. baths offer many superior advantages, and in pneumonia the effect of gradually-cooled baths upon the cardiac, respiratory, and nervous centers is especially desirable. It is of importance that the patient while being immersed put forth no muscular effort. He must be held and supported while in the bath, and gentle friction to the skin-surface must be made. The temperature of the baths should not be too low at the start: at the beginning it should be 90° F. (32.2° C.), and then be lowered according to the degree of sensitiveness of the individual patient. It is rarely necessary to go below 80° F. (26.6° C.), and in the aged, the very young, and in persons previously debilitated it is unwise to use any other than tepid baths. The duration should not exceed ten to fifteen minutes on the average, and more than three—or at most four—baths daily are not required. I have abandoned the rigid cold-bath method. Cool sponging, combined with the ice-cap or the wet pack, may serve as a substitute when full baths cannot be employed.

Abortive Method of Treatment.—Petresco has found that large doses of digitalis (ʒj–ij ; 8.0, of the digitalis-leaves in an infusion daily) administered at the onset will jugulate the disease. His experience covered 1192 cases, and showed the surprisingly low mortality-range of 1.22 to 2.66 per cent. This plan of treatment is rational, since it aims at meeting the chief pathogenic indication of pneumonia by passing through the lung-tissue an adequate proportion of leukocytes, and thus re-establishing the cardio-pulmonary circulation. In the few instances in which I have adopted the plan it has failed to cut short the disease, though the cases terminated favorably.

Venesection.—This is a good measure in sthenic cases (which occur with relative rarity in cities, but are not uncommon in rural districts), the temperature falling, the pain, the dyspnea, and the nervous symptoms being relieved and the pulse softened. Later in the course of pneumonia venesection may be resorted to if cyanosis and the signs of collateral pulmonary edema—due to a failing heart—arise, but at this period bleedings rarely yield good results.

Antiseptic Method.—This method is based upon etiologic indications, and is most rational. The best antiseptics are carbolic acid (♏j ; 0.066, every four hours), thymol (gr. ij–iij ; 0.129–0.194, every four hours), mercuric chlorid (gr. $\frac{1}{100}$; 0.0006, every four hours).

Antipneumococcus Serum and Serum obtained from Convalescents.—Washbourn, Pane, Fanoni, and others have reported favorable results from the use of antipneumococcus serum. On the other hand, many clinicians who have employed the serum are skeptical as to its therapeutic efficiency. It seems to possess considerable protective power, as shown by the Klemperer brothers (*vide* p. 140). J. C. Wilson[1] employed the serum prepared by Joseph McFarland in Mulford's laboratory in 18 cases of pneumonia, with 4 deaths. He observed that after the injections the temperature became lower, the pulse slower, and the patient felt better, but the duration of the attack was not shortened nor defervescence hastened.

Treatment of Special Symptoms.—The initial **pain,** which is of an acute, agonizing character, is relieved by the hypodermic use of morphin at intervals of six or eight hours. This counteracts the shock produced by the invasion-period, but it is to be omitted if the bronchi contain secretory products, since morphin dries these and favors their accumulation rather than their removal. Rarely is it necessary to continue this remedy after the second or third day of the illness.

Fever.—The fever of pneumonia is a temporary affair, and instead of being hurtful may prove beneficial, since it furthers tissue-metabolism, and this aids in the destruction of the specific poison of the disease. Fortunately, internal antipyretics for the purpose of combating high temperature are not so largely used at the present day as formerly. It is true that they possess the power to reduce temperature, but their use is attended with danger from their action as cardiac depressants; while, if it be true, as before stated, that pneumonia usually kills through the heart, it follows that cardiac power must primarily be conserved. Apart from the above-mentioned serious objection to internal antipyretics, it is to be remembered that they do not possess the important additional advantages to be derived from cool baths. In my opinion, their use should be limited to those cases in which cool baths or their substitutes (cold pack and cold spongings) fail to effect a reduction of fever. It must not be forgotten that unless the temperature exceed 104° F. (40° C.) it had better be let alone. When called for, the best among the antipyretics are acetanilid and phenacetin. The dose of these should be small—gr. v (0.324) of the latter and gr. ij–iij (0.129–0.194) of the former, to be repeated at intervals of four to six hours if required.

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[1] *Philada. Med. Journ.*, June 9, 1900.

Like internal antipyretics, arterial sedatives are to be used sparingly, and when used their effects must be carefully noted. In cases in which venesection is indicated the tinctures of veratrum viride and of aconite have been much vaunted as substitutes. The tincture of veratrum viride produces a good effect upon the congestion in the early stage, since it relaxes the arterial walls, and thus bleeds the patient into his own vessels, and "allows the return of the blood to the circulation when the stage of consolidation is reached" (H. C. Wood). The tincture of aconite, owing to its depressing influence upon the heart, should not be employed.

The **nervous symptoms** are successfully met, as a rule, by hydrotherapy (including the ice-cap), by the arterial stimulants, and by the use of morphin, as before recommended.

Cough during the early stage is controlled by the morphin needed to combat the pain. In the more advanced stages, if there be present numerous moist râles and a scanty expectoration, stimulant expectorants (ammonium muriate, terebene) may be employed with happy effect; but ordinarily they do harm rather than good. Pilocarpin may aid resolution when this is delayed (Reiss); the heart must be guarded.

Complications.—The management of the complications does not differ from that which is appropriate when they occur as independent affections, though all depressing measures must be positively omitted. I would add that in pleuro-pneumonia aspiration is not well borne, according to my observations; hence, unless urgently needed, I would postpone this procedure until the crisis has passed.

Among measures to prevent *ether-pneumonia* I would urge an appropriate toilet (a thorough cleansing followed by the topical use of an efficient antiseptic solution) of the naso-pharynx and mouth as a routine practice.

Local Measures.—When in doubt as to whether venesection should be employed or not, it must be remembered that early local bloodletting (cupping and leeching) is followed by relief from pain and dyspnea, but that these measures should be reserved only for robust persons. Counter-irritation by means of sinapisms is useful at the onset. The cotton jacket has certain advantages in maintaining the free, local action of the skin, and may be employed; before the days of hydrotherapy it was quite commonly used. The *topical use of cold* in the form of ice-bags has been practiced extensively by Lees of England and Mays of America with brilliant success, cold thus applied relieving the pain and dyspnea and diminishing pyrexia. In my limited experience with the application of cold I have found cases in which it could not be tolerated by the patient, and in such moderately cold wet compresses or the cotton jacket may be substituted. Lépine has used with success very dilute mercuric chlorid injections into the affected lung-tissue.

SECONDARY PNEUMONIA.

Pathology.—The lesions are identical in character with those of primary lobar pneumonia, but the areas involved have not always the same regular distribution. Congestion surrounding the hepatized lung-tissue is not infrequently extensive. We see, post-mortem, a tendency to commingling with small areas of lobular pneumonia. Both the streptococcus and the micrococcus lanceolatus are frequently found on microscopic examination.

Etiology.—Most instances are secondary to the acute infectious diseases, and it is probable that the specific causes of certain of the latter (Eberth's bacillus, Pfeiffer's bacillus, etc.) have the power to excite the morbid changes of acute lobar pneumonia. *Colon-pneumonia*, due to the bacillus coli, is the result of hematogenous infection either from the intestinal or from the urogenital tract. In the majority of instances, however, in which this disease develops in the course of the acute infectious diseases the latter are to be regarded as merely furnishing the opportunity for infection by the micrococcus lanceolatus.

Symptoms.—The rational symptoms are often absent. Close observation may, however, detect more or less dyspnea, cough, and increased fever, and rarely the attack is heralded by a rigor, followed by fever, the pneumonic type of breathing, pain, cough, and the characteristic expectoration.

The physical signs, when carefully observed, usually serve to enlighten the physician as to the nature of the affection. Hence it is a natural corollary that repeated physical examination is demanded in all cases in which there is danger of intervening lobar pneumonia.

Diagnosis.—This rests chiefly upon the physical signs, which are the same as in primary lobar pneumonia. Obviously, when the local subjective symptoms and the characteristic sputa are present a correct diagnosis is easily made. The fact must be emphasized that bronchopneumonia arises in the course of infectious diseases far more frequently than does lobar pneumonia.

Prognosis.—The occurrence of lobar pneumonia as an intercurrent affection adds greatly to the gravity of the primary disease. It is especially dangerous when it appears as a sequel during convalescence from acute infectious diseases.

The treatment is similar to that of primary lobar pneumonia, though less satisfactory.

INFLUENZA.

(*La Grippe: Epidemic Catarrhal Fever.*)

Definition.—Influenza is an acute contagious disease, probably caused by the bacillus of Pfeiffer. Its chief symptoms are due to catarrh of the respiratory and digestive tracts, together with profound muscular and nervous prostration, and grave complications (especially pneumonia) often present themselves. The disease may be endemic, though oftener it prevails in an epidemic or pandemic form.

Historic Note.—Every quarter of the globe has been the scene of visitations of epidemic influenza. More rapidly than any other disease belonging to the same class does it traverse a region of country, and hence within a remarkably brief period of time a whole nation may suffer. As a rule, influenza develops into epidemic proportions in the East, whence it spreads with unparalleled rapidity in a westerly direction. The first epidemic of the disease in the United States appeared in 1647, and was subsequently described; and, though it has since then frequently prevailed, the outbreaks have not observed any regular periodicity. The last true pandemic of the affection originated in Bokhara in May, 1889, reached St. Petersburg in the following October, Paris in November, and London in turn early in December. In America the cases began to appear about the middle of December, and rapidly multiplied into an explosive epidemic, which reached its maximum in January, 1890. Influenza reappeared in epidemic form, though less extensively, during April and a part of May, 1891, and again in a briefer and lighter form in the winter of 1891–92. During the winter of 1892–93 only a few sporadic cases occurred, but in each year since then it has prevailed in an epidemic form in certain sections of this country. During the early part of the present year (1901) a pandemic visitation of the disease occurred in the United States.

Pathology.—There are no special anatomic lesions that characterize the disease. In the rare instances in which death occurs in uncomplicated cases the catarrhal changes of the respiratory and gastrointestinal mucosa disappear after death. In the abdominal type of the affection there may be enlargement of the glands of Peyer and of the solitary follicles. The fatal cases exhibit lesions which are to be ascribed to the complications. Among the latter are pneumonia (either lobular or lobar), with which plastic pleurisy is usually associated, serofibrinous pleurisy, empyema, purulent pericarditis, nephritis, and rarely cerebrospinal meningitis.

Etiology.—**Bacteriology.**—Early in the year 1892 Pfeiffer discovered a bacillus which he believes to be the true cause of influenza—the *bacillus of Pfeiffer.* It is of about the same breadth as the bacillus of mouth-septicemia, and only one-half the length of the latter. When stained with Ziehl's carbol-fuchsin it may be observed as a small dumbbell, having knobbed ends connected by a rod-like shaft. These bacilli are obtained from the sputum. Pfeiffer has shown that they may penetrate the peribronchial tissue and pass out to the pleura. They have also been found in the blood. This bacillus can be cultivated in agar and other media, and when inoculated into rabbits it causes symptoms resembling those of influenza. The almost invisible cultures of the influenza bacillus become larger if inoculated with the staphylococcus aureus (Meunier). While the bacillus of Pfeiffer is generally supposed to be the sole pathogenic agent in influenza, this has not as yet been proved.

Modes of Conveyance.—A specific germ that is propagated with the unusual rapidity that marks the bacillus of Pfeiffer must be air-borne. Pepper suggests that the micro-organism may be almost universally distributed, and that under certain extraordinary atmospheric or telluric conditions it acquires a degree of virulence that renders all subject to its attack. There is no doubt that influenza is communicable by *contagion*, and evidence is becoming abundant to show that it may be transferred by *fomites*. In some epidemics the disease travels slowly, and follows principally the lines of ordinary human and commercial intercourse. Epidemics may take a course opposite to that of the prevailing winds.

Manner of Invasion.—How the contagion enters the system has not been positively determined, though it is probably with the inspired air through the respiratory tract. Some authors contend that it may enter through the alimentary canal, while still others believe that the primary point of infection is not rarely the conjunctiva.

Predisposing Causes.—These are few and unimportant, since all persons are liable to the contagion. *Age* has slight influence, the period of greatest susceptibility being from the twentieth to the thirtieth year. The very young are less liable than older subjects, and during an epidemic are apt to be affected last, while old persons (particularly if debilitated) are frequent sufferers. The same is true of those whose vitality is lowered by neuropathic heredity or chronic maladies, these being among the first to be affected during an epidemic. On the other hand, it is to be remembered that the healthiest are not exempt.

Immunity.—A primary attack of influenza does not bestow immunity, since *relapses* are very common, in 10 per cent. of the cases (Turney). Many persons, too, suffer from the disease with the reappearance of fresh epidemics, so that two, three, four, or even more attacks may be observed in the same individual (*recurrences*). Recent investigations have shown that a decided antagonism exists between influenza and malaria.[1] Epidemic influenza increases susceptibility to pneumonia and probably also to typhoid fever and appendicitis.

Clinical History.—**General Symptomatology and Course.**—The *incubation period* is quite brief, rarely exceeding two or three days. The *onset* is generally sudden, with either a severe rigor or repeated slight shiverings, accompanied by a rapid elevation of temperature which may touch 104° or 105° F. (40.5° C.), intense headache, distressing myalgic pains, and great prostration. The primary fever, however, varies greatly in severity, as does also the character of the symptoms—both local and general. Profound prostration characterizes the vast majority of instances during the invasion period. Depression of spirits, restlessness, insomnia (more rarely undue somnolence), and frequently delirium, are among prominent nervous phenomena.

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[1] "A Statistical Study of Influenza; its Potency to Lessen the Receptivity of the Body for Malaria, as well as to Increase the Receptivity for Pneumonia and, probably, Typhoid Fever," by the author—*Philadelphia Hospital Report*, 1895, vol. iv.

Rare Modes of Invasion.—The affection may set in (*a*) by vertigo, (*b*) by apoplectic features, (*c*) by bilious vomiting, (*d*) by an abrupt and profound prostration. Nose-bleed sometimes occurs.

The most striking symptom is *pain*, which in many cases is referable chiefly to the forehead, temples, occiput, eyeballs, and root of the nose. General neuro-muscular pains are often present. The principal seat of the pain is commonly the lumbar spine (rachialgia). I have frequently noted cutaneous hyperesthesia. The pains may take the form of neuralgia of individual nerves or of pleurodynic stitches, or there are localized areas of burning, boring muscular pain. The *temperature* may, as before intimated, mount quite high at the beginning, and if so it usually remits during the first night. It subsequently pursues a comparatively low range. The temperature-curve is markedly irregular, and often terminates by an apparent crisis. The *pulse* is small, feeble, irregular, and even intermittent, and I have sometimes observed it to be unusually slow, cardiac debility being prominent and at times reaching a dangerous degree. In many cases *dyspnea* is a rather conspicuous symptom, occurring independently of pulmonary complications. The same is true of *cyanosis*. Sweating may be troublesome.

Clinical Types.—Different types have been described based on the differences in the local manifestations. But it is to be mentioned that influenza is remarkably protean in its clinical features, and that the enumerated types quickly and frequently merge into one another. (*a*) *Respiratory Type.*—Local catarrhal symptoms usually develop in the course of one or two days. They are, as a rule, evidenced first by a suffusion of the conjunctivæ, with excessive lacrymation, frequent sneezing, and slight pharyngitis. A little later, in most instances, hoarseness and cough come on, the latter being hard, racking, paroxysmal in character, and resembling whooping-cough. The cough and other local symptoms are due to intense, dry laryngo-tracheal irritation. In most instances the expectoration is scanty, and in these the physical signs are very generally negative. In a smaller proportion of the cases there is considerable expectoration, and the physical signs of ordinary bronchitis are manifested. (*b*) *Gastro-intestinal Type.*—The catarrhal symptoms sometimes center in the digestive system, and most frequently in children. In such, vomiting comes on early and is apt to be repeated at longer or shorter intervals. There is diarrhea, more or less urgent, with sharp abdominal pain, as a rule. (*c*) The *cardiac* group of symptoms that occasionally supervenes comprises heart-failure and distress, with a rapid, feeble pulse. (*d*) *The typhoid type* presents a continued fever, with the signs of the typhoid state. Nervous symptoms are very marked, such as stupor or delirium, dry, brown tongue, etc. (*e*) *The rheumatoid type* manifests itself by violent pains in the muscles all over the body. There is no visible change in either the joints or the nerve-trunks. (*f*) Huchard[1] calls attention to *apyretic forms* in which there may be marked pulmonary congestion or actual lobar pneumonia without fever, without expectoration, and often without cough. The condition is often discovered upon auscultation. (*g*) There are ambulatory forms which are important because they tend to spread the affection. In cases of average severity *convalescence* is usually somewhat protracted, and in the severer forms decidedly so.

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[1] *Bull. Acad. de Méd.*, Feb. 17, 1900.

Complications.—(1) **Pulmonary.**—Severe bronchitis, particularly affecting the capillary tubes and leading to bronchopneumonia, is a common and very serious complication. As a secondary result we are apt to observe the development of collateral pulmonary edema, with its usual fatal termination; and whilst this complication is prone to develop in the so-called thoracic type of influenza, it is by no means limited to this class of cases. I have observed bronchopneumonia in cases in which the physical signs of bronchitis were not presented prior to its onset. It may originate apparently in the profound prostration of the nervous system—a condition which also annuls in great part the phagocytic action of the leukocytes. In nearly all instances, however, this form of pneumonia, as well as croupous pneumonia, may be definitely traced to exposure. The recognition of bronchopneumonia is in many instances difficult.

According to some authors, the nature of the condition is variable, and may at times be ascribed to *congestive collapse* and other conditions, rather than to the ordinary type of bronchopneumonia. *Congestion associated with edema* of the lungs occurs as a complication of influenza, as I have learned from personal observation. Acute enlargement of the bronchial glands may also be noted, and the recognition of this condition may be aided by careful percussion over the upper four dorsal vertebræ, where dulness will be obtained (*vide* Streptococcus-pneumonia as Complication of Influenza, p. 150).

Lobar pneumonia is also a frequent and very fatal complication. It may arise early and in rare instances insidiously, but it is much more apt to manifest itself after influenza has about exhausted its force upon the vital organs or during the early part of convalescence. The symptoms of invasion—severe chill, high temperature, followed by the usual physical signs—are *sudden in their onset* and lead rapidly to an extremely serious condition.

When lobar pneumonia develops early in the course of influenza (a rare event), its symptoms are modified, the preliminary chill and pain in the side being often absent, and more frequently still the characteristic crepitant râle. Subcrepitant râles, however, are audible, and the dyspnea is out of proportion to the area of lung-tissue involved. Most of the peculiar features just pointed out may also be observed in connection with the pneumonia that appears during convalescence. (See, also, Apyretic Varieties, p. 163.)

Plastic pleurisy is commonly an associated condition, especially in cases of lobular or lobar pneumonia. Other forms of pleurisy also occur, though less frequently (sero-fibrinous and empyema). *Gangrene* and *abscess* of the lungs may arise as terminal complications.

Cardiac Complications.—Heart-failure often manifests itself, and may prove fatal, though rarely. Purulent pericarditis is a rare complication, and is often secondary to pleurisy or pneumonia, while attacks of angina, which usually interchange with simple weak heart (often associated with arrhythmia), have been noted in certain epidemics (Curtin and Watson).

Gastro-intestinal System.—There may be severe gastro-enteritis, with frequent vomiting and purging and intense abdominal pains, and, more rarely, hemorrhages occur from the stomach and bowel. Catarrhal jaundice may appear, due to duodenal catarrh, but these gastro-intestinal complications are more apt to be met with in young children than among adults.

Nervous System.—The most frequent symptom is perineuritis, which probably causes much of the patient's sufferings. A soporose condition is often observed. Delirium of a most active form sometimes appears, and particularly when certain other complications have arisen, such as pneumonia, pericarditis, etc. Cerebro-spinal meningitis occurs as a rare complication. I have observed symptoms identical with those of meningitis appearing suddenly, and in the course of a day or two disappearing just as suddenly. The symptoms under such circumstances must be due either to the action of the specific poison upon the nerve-centers or to congestion, and hence a diagnosis of suppurative meningitis is to be made with extreme caution. In addition to the symptoms, we should have the existence of suppuration elsewhere in the body (otitis, purulent pericarditis, etc.) or the presence of pneumonia. Cerebral abscesses have also been noted (Bristowe). Kerr has reported disseminated lesions of the central nervous system following influenza.

Genito-urinary Tract.—Renal congestion, and even acute nephritis, may appear as a complication. A case of cystitis with hematuria has also been reported (Comby and Le Gendre).

The **diagnosis** of influenza except in ill-defined, sporadic cases rarely presents serious difficulty. Usually, the march of the epidemic, the abrupt onset, the brevity of the febrile stage, the rachialgia, the speedily developed prostration, which is out of proportion to the catarrhal manifestations, form a conclusive association of symptoms. In the obscure and irregular forms the sputa, if there be any, should be studied bacteriologically.

(*a*) *Climatic catarrhal affections* are sometimes hard to discriminate from sporadic cases of influenza. The former are usually attributed to sudden and great vicissitudes of temperature or exposure to strong drafts of air, while the latter come on independently of seasons of the year and of such agencies. Again, in influenza we usually observe the general features (nervous symptoms and debility) outweighing the local (catarrhal manifestations). Leichtenstern speaks of pseudo-influenza, or catarrhal fever (*influenza nostras*), believing that it bears the same relation to true influenza as does cholera nostras to Asiatic cholera. Its cause is unknown.

(*b*) *Typhoid fever*, particularly in its early stages, is often closely simulated by influenza with intestinal symptoms. Influenza, however, gives a different history, begins suddenly, does not show the typical temperature-curve of typhoid, may present splenic enlargement—but by no means to the same extent as typhoid—has no characteristic eruption, and does not give the characteristic sero-reaction.

(*c*) *Pneumonia* has quite frequently been mistaken for influenza, and especially when the thoracic symptoms in the latter have been unusually distinct. As already stated, lobar pneumonia may early complicate influenza in rare instances; but pneumonia is generally unilateral, while the lung-involvement in influenza is generally bilateral. In the former the physical signs indicative of consolidation are clearly marked; in the latter (unassociated with pneumonia) we often meet with those suggestive of congestive edema (impaired resonance, stationary crepitant and subcrepitant râles). The general features also present dissimilarities. Thus the nervous depression and the myalgic and neuralgic pains are more marked in influenza, while the pulse and respiration are apt to be less frequent than in pneumonia.

(*d*) *Cerebro-spinal meningitis* may manifest features that are almost identical with those characteristic of influenza. Thus during certain epidemics "grippe" patients may be stricken as by a blow; they suffer from intense headache—occipital and frontal—rachialgia, fever prostration, delirium, and stiffness of the muscles, with slight retraction of the head. There may be convulsions and vomiting at the outset. Here the history with reference to the character of the prevailing epidemic and the attendant circumstances must be carefully considered, but an absolute diagnosis is sometimes impossible unless a laboratory investigation of the discharges or lumbar puncture be made.

Sequelæ.—Among the pulmonary sequelæ are phthisis, chronic bronchitis, abscess and gangrene of the lungs (the latter two being rare), tachycardia, and angina pectoris. Chronic gastro-intestinal catarrh is not rare as a remnant. Chronic nephritis, and less frequently cystitis, may also be mentioned. Latent forms of tuberculosis and chronic nephritis are often kindled into active and progressive affections by intercurrent influenza.

Among *nervous* sequelæ, which are both numerous and important, are to be noted especially insomnia, neuralgia, migraine, melancholia, mania, meningitis, acute ascending myelitis, locomotor ataxia, peripheral neuritis, and peurineuritis. The organs of special sense manifest a great variety of sequelæ, such as otitis media, otitis interna, mastoid abscess, conjunctivitis, keratitis, iritis, irido-chorioditis, acute glaucoma, etc.

Prognosis.—The prognosis is, on the whole, good. Almost all fatalities are due to complications, especially *pneumonia*, and, less frequently, pulmonary congestion and edema, pleurisy, pericarditis, and cerebro-spinal meningitis.

The *circumstances connected with the individual case* often affect the outcome. Thus influenza runs a more severe course, and hence offers a correspondingly more serious prognosis, in those enfeebled on account of previous chronic disease (phthisis, valvular disease of the heart, emphysema, nephritis) and in the young and the old than at other periods of life. During severe epidemics of influenza the mortality-list in most chronic diseases is considerably augmented. Though epidemics vary as regards the mortality, the general average death-rate is a little under 1 per cent. In some epidemics it may reach 2 per cent., while in others it may be less than ½ of 1 per cent.

Duration.—The *duration* of the attack is brief, though subject to variations. In mild forms it is from two to four days, in the severe from seven to ten days; but complications and previous infirmities may greatly prolong the attack. The *duration* of *particular epidemics* rarely exceeds from four to six weeks.

Treatment.—**Prophylaxis.**—Experience has shown almost conclusively that the various drugs which have been counselled for their preventive effect (quinin, salicin) are devoid of value. The strongest persons are not immune, and those who are at either extreme of life or who are enfeebled by chronic organic disease, should be most carefully protected by proper wearing-apparel, and should not be carelessly exposed to unfavorable weather conditions. In this way we may lessen the totality of the cases, since the inmates of hospitals and prisons have been known to escape the disease. **Isolation** should therefore be carried out in hospitals and, whenever practicable, in private families, especially when the disease appears in households in which there are young children and aged persons. E. W. White has reported an epidemic of influenza that was successfully aborted by strict isolation of the patients. **Disinfection** of the catarrhal discharges, particularly the bronchial, which, as a rule, abound in the bacilli of Pfeiffer, is necessary. I must also insist upon disinfection of the naso-pharynx and mouth cavity.

Treatment of the Attack.—In considering the treatment of the attack the cases may be grouped under three heads:

(*a*) **Mild or Rudimentary Form.**—The cases belonging to this type require little besides careful hygienic management. However light the attack, the patient should remain in-doors and, if languid or prostrated, in bed for a period of two or three days. The *diet* should be light and nutritious (milk, eggs, rice, gruels, fresh vegetables, stewed fruit), and cooling drinks are to be preferred to hot ones, among the former lemonade or cold oatmeal water with lemon, and effervescent mineral waters (Apollinaris, lithia, Seltzer), being the best. The bowels should be moved regularly, avoiding, however, active purgation. Stimulants are not needful, but the use of light wines is not objectionable if desired by the patient. In all cases of influenza, even of the mildest grade, I prescribe moderate doses of quinin (gr. iv—0.2592, three or four times daily), and if there be much headache this drug may be combined with Dover's powder and monobromate of camphor (of the first two, gr. iij—0.194, each, and of the last gr. j—0.0648, in capsule), the dose to be repeated at intervals of three or four hours. To overcome the languor and debility, which are marked, I have found nothing so successful as strychnin.

(*b*) **Cases of Medium Severity.**—*General Management.*—This class of influenza patients betake themselves to bed, and should be kept there till convalescence is well advanced. During the febrile period the *diet* must be light, liquid, yet nutritious, and the food should be given every two or three hours. Although the patient has no desire for food, he should be urged to take it regularly. Moderate stimulation is also useful.

The *medicinal treatment* is, for the most part, simple and symptomatic. Calomel in moderate doses (gr. j every third or fourth hour) should be a remedy of choice for a day at least. An efficient diaphoretic, given within six or eight hours from the time of onset, may abort the attack. The neuralgia and myalgia may be relieved by the use of quinin, Dover's powder, and ergot; but if the pain be intense, morphin administered subcutaneously may be required. The temperature is somewhat reduced by these remedies, and especially by the quinin and Dover's powder, the latter acting as a diaphoretic. In addition, I am in the habit of ordering cool sponge-baths at intervals of two or three hours if the temperature be about 102° F. (38.8° C.). If not controlled in this manner, we may combine with quinin some antiseptic, such as salicylic acid or salol. I have sometimes found it necessary to add to the foregoing small doses of phenacetin (gr. ij—0.129). *Sleeplessness* may demand hypnotics, such as sulfonal, chloralamid, opium, and trional. It is necessary to utter a warning against the free use of coal-tar products, as acetanilid, antipyrin, and similar preparations, since they induce heart-failure.

The local catarrhal conditions (coryza, laryngo-bronchial irritation, true bronchitis, etc.) must be treated according to the special indications presented in individual cases. For the coryza inunctions of animal fats over the forehead and bridge of the nose are useful. A flannel cap may be worn if agreeable to the patient. Steam inhalations through the nares and mouth often act beneficially, both upon the coryza and laryngo-bronchial irritation. For the latter common condition the following formula will be found serviceable:·

℞. Codeinæ sulph.,	gr. iv	(0.259);
Ammon. chloridi,	ʒv	(20.0);
Syr. prun. virgin.,	f℥ij	(60.0);
Spts. junip. comp.,	q. s. ad f℥iv	(120.0).—M.

Sig. One teaspoonful every two or three hours.

If this prescription fail to mitigate the cough, we may resort to morphin hypodermically, but always in small doses. In the later stages, particularly if bronchitis be associated with free secretions, the oil of eucalyptus (♏iij to v—0.199 to 0.333), in capsule, every four hours, has in my experience proved useful. To obviate pulmonary complications I have been much gratified with the results from the use of strychnin (gr. $\frac{1}{30}$—0.0021), combined with vin Mariani (℥ss—16.0) at intervals of three or four hours. Chest-pains may be relieved by the use of turpentine stupes and sinapisms, both of which agents are also valuable in averting the more serious complications.

(*c*) **Severe Forms.**—*The general management* is similar to that recommended in cases of medium severity, excepting that freer stimulation is usually demanded. The medicinal treatment must also be more active than in the previous form, and often is heroic. Especially must quinin be given in full doses and continued, since it not only serves to reduce the temperature somewhat, but also to sustain the vital forces, to control the nervous symptoms and lessen the tendency to inflammatory complications. Nothing that exerts a depressing effect should be thought of, since the cardiac as well as the respiratory forces must be conserved. Should there be sudden cardiac failure, it must be promptly met by the various forms of stimulants, including strychnin and the cardiac tonics. In addition to alcoholic stimulants, the aromatic spirits of ammonia is usually borne well, and should be administered. Strychnin must be given in full doses hypodermically every third or fourth hour. The various inflammatory complications that may arise must be treated as under other circumstances.

The Convalescence.—In all grades of cases the convalescence from influenza demands most rigid supervision, and the greatest injury to patients at this time comes from going out too early. Usually the temperature is subnormal for several days—a circumstance due to the weakness of the patient—and so long as this condition obtains the patient is highly susceptible to a chill. Hence it is a good rule not to allow exposure to the external changes of temperature until the temperature has been normal for several days. The diet should now be more liberal, and tonics, such as gentian, iron, and quinin, may be administered and continued until complete restoration of the patient's health has taken place. In every way possible exposure to reinfection during the period of convalescence is to be avoided.

The treatment of sequelæ must be conducted according to general rules.

DENGUE.

(*Break-bone Fever.*)

Definition.—An acute infectious disease occurring epidemically in tropical and subtropical countries. Its chief symptoms are—a double febrile paroxysm (separated by an interval), arthritic and muscular pains, and a skin-eruption in about one-half the cases.

Historic Note.—The disease was prevalent in Java as early as 1779, in India in 1824, and later in the West Indies, Spain, and in some of the southern American States. Mild epidemics have visited Philadelphia, New York, and Boston (during warm weather), but, as a rule, it has not traversed regions beyond 32° N. latitude.

Its *pathology* has not been studied, death being the rarest of events.

Etiology.—McLaughlin, of Texas, has isolated from the blood and cultivated a micrococcus which he claims is the specific organism.

Predisposing Factors.—Its prevalence is favored by the summer season, and also to a slight extent by faulty hygienic conditions. On the other hand, age, race, sex, and social status are all without effect, most persons being susceptible, a fact that accounts for its marvellously rapid diffusion. As a rule, susceptibility is exhausted by one attack, but second and even third attacks have been observed during the same epidemic. The epidemics spread along lines of travel by land and sea, and most authors agree that the disease is contagious.

Clinical History.—There is a period of *incubation* that lasts from one to four days and exhibits no prodromes.

Invasion then is *abrupt* with a slight chill; fever follows, the temperature reaching its maximum—103° to 106° F. (39.4° to 41° C.) or over—at the end of the first or on the second day, and is accompanied by headache and by muscular and arthritic pains. The patient's sufferings are intense, the *pains* being described as "breaking"—a peculiarity to which the disease owes the popular name of "*break-bone fever.*" The *joints* become red, swollen, and tender. The *respirations* and *pulse* are much quickened; there is anorexia and sometimes slight nausea. Febrile albuminuria is rare, delirium and mental torpor also; but *prostration* may become marked, and an *erythematous eruption* (*initial rash*) com-hours if the temperature be about 102° F. (38.8° C.). If not controlled in this manner, we may combine with quinin some antiseptic, such as salicylic acid or salol. I have sometimes found it necessary to add to the foregoing small doses of phenacetin (gr. ij—0.129). *Sleeplessness* may demand hypnotics, such as sulfonal, chloralamid, opium, and trional. It is necessary to utter a warning against the free use of coal-tar products, as acetanilid, antipyrin, and similar preparations, since they induce heart-failure.

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The *initial fever* lasts three or four days, and ends with a deep remission accompanied by profuse sweating. All the symptoms now vanish save a slight soreness and stiffness, but after an interval of two or three days the characteristic symptoms (including a roseolar eruption) reappear. The *second* febrile paroxysm is usually milder and shorter than the first.

The **duration** of the disease is from seven to ten days, the attack being followed by a slow convalescence, which may be interrupted by a relapse. The slowness of the recovery is due to persistence of the pains, mental depression, and marked physical prostration.

Complications.—Meningitis has been rarely noted. Convulsions sometimes occur in children, and severe catarrhal inflammations of certain mucosæ (bronchial, gastric) may develop. Insomnia is common. Hyperpyrexia and pericarditis occur, though exceptionally.

Diagnosis.—The diagnosis of the usual form of the disease (epidemic) is an easy one after observation of the first few cases, but a more difficult task is the discrimination of sporadic cases from *rheumatism*. The course and degree of the fever, however, differ in dengue and in rheumatism, while the eruption belongs to the former alone. *Influenza* may closely resemble dengue. In the former condition the herpes is usually the only eruption; the joints are rarely involved; there is neither a remission nor a recurrence of the fever; and serious complications are more frequent. The discovery of the bacillus of influenza is probably decisive, and the existence of an epidemic of either condition strongly suggests the true nature of the disease. *Scarlet fever* has an erythematous eruption, but the fever is continuous and the arthritic symptoms are usually wanting. Dengue prevails only in tropical and subtropical countries as a rule.

Yellow fever has often been mistaken for dengue, and the two affections have been known to prevail together, as in the Galveston epidemic of 1897. To establish a differential diagnosis is often most difficult, as there are a number of points of similarity—time of appearance, geographic distribution, influence of severe frosts, and the character of the febrile paroxysm. To show contrast, however, I have arranged the following comparison of symptoms:

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[1] *Rev. de Méd.*, No. 6, 1894.

DENGUE.	YELLOW FEVER.
Affects all races.	Foreigners more especially.
Irregular rise of fever, followed by remission, then a second moderate rise. Duration 5 to 9 days.	The temperature rises regularly. Duration of fever 72 hours.
The pulse keeps pace with the fever.	Pulse falls while the fever is rising.
Eruption frequent.	Eruption quite rare.
Vomiting rare.	Vomiting frequent.
Urine never contains albumin (?).	Urine commonly albuminous, often with suppression.
Jaundice absent.	Jaundice present and early appearing.
Hemorrhages from mucous ·outlets, generally slight, and black vomit rare.	Hemorrhages common and severe. Black vomit an alarming symptom.
Prognosis favorable.	Often fatal.
Serum-diagnosis valueless.	Serum-diagnosis (Widal-reaction) present in 66 per cent. of cases.
Second attacks common.	No second attacks.

The **prognosis** is with rare exceptions favorable, dangers arising only in the serious forms, particularly those showing hemorrhages.

Treatment.—The case presents a double indication: (*a*) to harbor the patient's strength, and (*b*) to meet certain leading symptoms. The first is to be accomplished by enjoining rest in bed, by a generous though carefully regulated diet, and by the timely use of stimulants and tonics during convalescence. Among the symptoms that demand treatment is the fever, and when this is high hydrotherapy is indicated. For the intolerable pains morphin is to be administered hypodermically. Chloralamid and the bromids should be tried. Isolation should be practised.

THE PLAGUE.

(*Bubonic Plague; Black Death.*)

Definition.—A specific contagious disease, occurring chiefly in unsanitary surroundings and characterized by high fever and cutaneous symptoms (petechiæ, etc.). Its course is severe and rapid, and it occurs in epidemics.

Historic Summary.—An Oriental disease, the plague, has long been endemic in certain portions of India. Most European countries have in the past been visited by epidemics of the malady, and among the most famous was the truly pandemic prevalence of "black death" in Europe during the fourteenth century. Another virulent outbreak occurred in London in 1665, destroying more than 70,000 persons. In May, 1894, a severe epidemic prevailed in Canton and Hong-Kong, to which cities it had been imported from Northern India. In September, 1896, the plague appeared in Bombay and the Bombay Presidency; in November, the disease, "which had seemed to be abating, revived, and the mortality steadily rose" (Willoughby). Since then the plague has shown periods of decrease followed by others of decided increase, and the total plague-statistics for the Bombay Presidency from September, 1896, to January 13, 1899, are 214,197 cases and 169,240 deaths. In the autumn of 1899 2 cases were brought to the New York harbor, and in March 6, 1900, it appeared in the Chinese quarters of San Francisco, where 26 cases were officially reported. It has reached several European ports—Oporto, Hamburg, Glasgow, London.

Etiology.—Bacteriology.—During the epidemic at Hong-Kong, Kitasato and Yersin, working independently (1894), discovered the special organism of the plague (*bacillus pestis bubonicæ*). It stains deeply at the ends, giving the appearance of a pair of micrococci, but is really a short rod-bacillus with rounded ends. Pure cultures are readily made, and when animals (mice, rats, guinea-pigs, rabbits) are inoculated with these the clinical characteristics of the disease are produced.

Predisposing Causes.—These are embraced in the single phrase—unhygienic conditions. It is safe to assert that without these fostering influences the plague would not prevail.

Inside the body the bacillus has been found in the lungs (plague-pneumonia) (where it is often combined with the pneumococcus and staphylococcus), in the enlarged glands, in the pus from the buboes, the blood, and other internal organs. *Outside* the body, among infected materials, are dust, plague-infected flies, the excreta, food, and soil.

Modes of Transmission and Entrance into the Body.—According to Kitasato, the bacillus enters either through the digestive or respiratory tract, or the skin (*e. g.*, abrasions of the feet). The bubonic pest is spread by two principal factors—the rat and man (Simond). In most outbreaks of human plague rats had the disease both before and during the epidemic (Clemow). The rat is the carrier from house to house, and hence is more important in the transmission of the disease in local epidemics than man; but the latter is the ordinary agent of transport for long distances. Flies, fleas, ants, and other insects may act as carriers of the contagion, particularly from rat to man. Nuttall's studies warrant the belief that transmission of the poison by stinging-insects is extremely rare. Certain animals besides rats (mice, dogs, cats, rabbits, pigs, horses, etc.) may become infected and die of the pest, and both before and after death transmit the disease to healthy animals. Yersin established contagion of plague by keeping inoculated rats and healthy mice in the same place (Payne, in Allbutt's *System*). The disease, however, is more commonly transmitted by foci of the infection (houses, ships) and by fomites.

Clinical History.—Varieties.—Two distinct forms, either of which may prevail alone, are recognized: 1. *Pestis minor*, abortive or larval plague, often appearing as a forerunner of severe epidemics. It is characterized by swelling of the lymphatics, and by slight general disturbance, and lasts about a fortnight. It appears only in the *endemic* foci of the plague. 2. *Pestis major*, or severe epidemic form. To rapidly fatal cases belonging to this variety the term *pestis siderans* or "fulminant" has been applied. Between the mild form (*pestis minor*) and the epidemic form transitional cases are rarely observed, and the following description has reference solely to the latter. Certain writers recognize a third variety—*plague-pneumonia*, in which the usual pneumonic symptoms (chill, stabbing-pains, cough, rusty expectoration, and signs of consolidation) appear.

The Attack.—The *incubation*-period lasts from three to five or rarely seven days. *Prodromal symptoms* are sometimes observed, and may last from twelve to twenty-four hours. They are intense headache, vertigo, pallor, and a gait suggestive of intoxication. The physiognomy is stupid, and the patient may refuse or be unable to answer questions.

Invasion.—This may be *abrupt;* less commonly it is preceded by the above symptoms; and rarely bilious vomiting or hematemesis are the ushering-in symptoms. There occur a prolonged *rigor* or recurring shiverings. The *temperature* rises rapidly to 103°, 104°, or even to 106° F., and the *pulse* becomes rapid and thread-like, although variable in force and character. *Delirium* or *coma* tends to supervene. *Debility* may now be extreme, and the patient may die in the initial period. More commonly this threatened collapse is survived, and then (from the second to the fifth day) the most characteristic feature almost always appears—*buboes* or inflammation of the lymph-glands. The latter may enlarge gradually or rapidly, and are usually quite painful. *Resolution* may occur, or they may remain unchanged, particularly in fatal cases. *Suppuration* may occur, and rarely gangrene, forming the so-called carbuncle. The latter may also appear as gangrenous patches over the skin of the trunk and legs. Petechiæ and hemorrhagic diathesis, as shown by bleedings from the lungs, stomach, and intestines, arise in the worst forms. *Plague-pneumonia* is common in certain epidemics.

The *duration* in fatal cases is usually from three to five days. On the other hand, cases that tend to recovery may be prolonged, on account of suppuration of the buboes, to a few weeks or even months.

Diagnosis.—The report of the Austrian Plague Commission divides the disease into two main varieties, one affecting the lymphatic glands—a form easily recognized—and the other the lungs. It was found to be very difficult to "differentiate between an existent pneumonia and the characteristic plague-pneumonia in the initial stage.[1] A certain diagnosis rests upon bacteriologic evidence alone.

Prognosis and Mortality.—The death-rate is exceptionally high, ranging from 40 per cent. (rare) to 80 or even 90 per cent. Among favorable indications is suppuration of the buboes. On the other hand, a rapid disappearance of a group of swollen glands is a bad augury. Additional unfavorable symptoms are plague-pneumonia, intense toxic features, purpuric spots ("tokens"), carbuncles, and hemorrhages.

Treatment.—**Prophylaxis.**—The precautions to be taken by the individual relate to the abandoning of all unsanitary habits, the isolation of the sick, and the avoidance of prolonged contact with infected patients or dwellings. It would seem that doctors and even nurses and attendants in well-ordered and properly-ventilated hospitals rarely take the plague.

The prophylaxis of the public embraces—(*a*) Isolation of the sick and thorough disinfection of the sick-room, the bed and bed-linen, the vomitus, and the stools. Kitasato advocates steaming the bed at 212° F. (100° C.) for one hour, or exposure for a few hours to sunlight, and the burning of all infected articles. "After recovery the patient is to be kept in isolation for at least one month." The infected houses are to be thoroughly disinfected, and a pure water-supply procured. (*b*) Protective inoculation or treatment by "vaccination" of healthy persons seems efficient. Haffkine reports upon the epidemic of plague in Lower Damaun as follows: 24.6 per cent. of over 6000 persons who were not inoculated died of the disease, while but 1.6 per cent. of over 2000 inoculated acquired the disease. Immunity seemed to be directly in proportion to the strength of the vaccine. Haffkine[2] in a recent report states that at Hubli practically all of 50,000 inhabitants were rapidly inoculated. The difference in mortality of those inoculated and of those uninoculated averaged from 80 per cent. to 90 per cent. The dose was 2.5 c.c. The length of time through which immunity lasted was not well determined. Calmette recommends Yersin's anti-plague serum for prophylactic purposes in preference to Haffkine's vaccine, since the effects of the latter are sometimes unpleasant.

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[1] *Philada. Med. Journ.*, Jan. 28, 1899.

[2] *Proc. Roy. Soc.*, vol. lxv., No 418.

Treatment of the Attacks.—The diet should be liquid, concentrated, and nourishing, while free stimulation is demanded from the onset. *Medicines* are used merely to combat symptoms as they arise. Delirium and pain are to be met by morphin or hyoscin, and high temperature by cold or tepid sponging.

Local Treatment.—Cantlie does not believe in local measures before suppuration occurs, although he has observed good results to follow injections of mercuric chlorid and potassium iodid.

Serum-therapy.—Anti-plague serum exercises a specific action (Yersin). Of 26 cases treated, 2 died—a mortality of 7.6 per cent. A. Calmette also reports favorable results from the use of Yersin's serum in the epidemic of plague at Oporto.

ERYSIPELAS.

(*St. Anthony's Fire.*)

Definition.—A specific, acute contagious disease, characterized by a special inflammation of the skin and subcutaneous tissues, with a tendency to spread, high fever, moderate prostration, a disposition to mixed infection (suppuration, gangrene), and an average duration of fourteen days. It usually occurs in an endemic, though also in epidemic form.

Pathology.—Erysipelas is a specific inflammation involving the skin, subcutaneous and mucous surfaces, the latter, however, somewhat uncommonly. When inflammation extends to the subcutaneous connective tissue there follows, as a rule, suppuration. The specific cocci are found in the superficial lymph-vessels and spaces of the affected skin, being most abundant in the ever-advancing elevated margin. Beyond the border of the inflamed region they occupy chiefly the lymph-vessels, where they are finally overpowered by the phagocytic leukocytes. Microscopic examination reveals the changes of simple inflammation. Pericarditis and endocarditis affecting the bicuspid and mitral valves may be noted.

Etiology.—**Bacteriology.**—The specific cause of the disease is the *streptococcus erysipelatis* of Fehleisen, which is probably identical with the ordinary pus-producing streptococcus. Fränkel and Kirchner have investigated, experimentally, the *streptococcus erysipelatis*, and contend that their results offer convincing proof of the separate identity of the streptococcus pyogenes; but most observers hold that the former cannot be distinguished from the latter. The streptococci of erysipelas assume the form of a serpent or chain (chain-forming coccus of Cohn), and are very small, somewhat variable in size, and thrive on all kinds of culture-media. Their favorite situations are the lymph-vessels and the cutaneous connective tissue, where they are found in colonies composed of myriads of cocci. They are rarely found in the blood-vessels, and in blood-serum they are caused to disappear by the action of the phagocytes; yet in exceptional cases intra-uterine infection has occurred. G. E. Pfahler[1] found a diplococcus in eight cases, and thinks it may be the cause of the disease. That the streptococcus erysipelas is a saprophytic organism is shown by the fact that the identical cocci have been discovered in inanimate and decomposing animal and vegetable substances.

Predisposing Causes.—(1) **Season.**—In a paper on "Seasonal Influences in Erysipelas, with Statistics,"[2] I have shown, as the result of an analysis of 2010 cases collected from different sources, that the various seasons of the year exercise a potent influence upon the frequency of this affection. Thus month by month the cases increase, in slightly varying ratio, from August to April, the latter month giving the greatest number, and then there is a rapid decrease from April to August, when we find the smallest number. Again, one-half of all the cases occur during the months of February, March, April, and May, and 15.9 per cent. during the month of April alone. It was found that a low barometer and mean relative humidity invariably correspond with the annual period in which the greatest number of cases occur, and that the highest percentage of relative humidity corresponds with the months affording the fewest cases.

(2) **Age.**—From the notes of 1894 cases I found that in 25.8 per cent. the age of the patient was between twenty and thirty years. After fifty years the cases decrease rapidly, and more than 15 per cent. occur before the age of twenty. The great liability of newly-born infants is well known.

(3) **Sex.**—This factor was noted in 1767 cases, and a marked preponderance of the male over the female sex was noted (about 3 to 2).

(4) **Previous Attacks.**—Of 450 cases, there had been previous attacks in 39 (8.6 per cent.), in one instance four, and in another seven, while second and third recurrences were not uncommon.

(5) **Family predisposition** exercises a slight though decided influence.

(6) **Certain Antecedent Affections.**—Dr. M. Booth Miller examined the history of 301 cases, and found that acute coryza preceded the attack in 13 instances. Slight lesions of the Schneiderian mucous membrane may be assumed to exist in such instances. Testimony confirming the well-known fact that certain chronic diseases (chronic Bright's, phthisis, organic heart disease, chronic alcoholism) augment a receptivity to the complaint has also been brought to light by my researches. Cheadle has seen 5 fatalities in cirrhosis of the liver from erysipelas.

(7) **Slight Injuries, Abrasions, etc.**—Erysipelas will not develop on a surface which does not present a break, but with this present may do so though the latter be so trivial as to escape observation. Slight abrasions and fissures, either in the mucous membrane of the nose or in the skin of the face or ear, as well as all forms of slight injuries, are liable to furnish a door of ingress to the specific organism. Yet in 643 out of the 2010 cases mentioned above, previous lesions were noted in but 13. Women who have been recently delivered and persons subjected to surgical operations are peculiarly liable, and any deeply-seated focus of

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[1] *Philadelphia Medical Journal*, January 13, 1900.

[2] *Proceedings of the American Climatological Association*, 1893.

irritation (necrotic bone, chronic abscess, appendicitis) may give rise to repeated outbursts of erysipelas.

(8) **Antihygienic Surroundings.**—These doubtless predispose to the affection, as has been shown by the prevalence of erysipelas in hospitals and institutions in which the sanitary arrangements were markedly faulty.

Modes of Conveyance of the Contagion.—The latter may be air-borne. It has been collected from the air of rooms and wards occupied by erysipelas patients; but to what distance and precisely under what circumstances it can be conveyed is imperfectly known. It may also be transferred for a longer or shorter distance by fomites, by instruments, unclean hands, etc., the *infection* being a result either of *contagion* or *inoculation.* It is possible that intravascular infection may occur, the organism gaining an entrance through the lungs or digestive tract.

Clinical History.—I shall discuss only the so-called *idiopathic* erysipelas, the traumatic variety falling within the domain of surgical treatises.

Incubation.—This is somewhat varied, though it ranges usually from seven to fourteen days. The *prodromal symptoms* are, for the most part, general in character, consisting in headache, restlessness, cough and sore throat, anorexia, and slight or moderate pyrexia. These endure for a very variable period—from a few hours to several days.

Invasion Stage.—The symptoms are (1) local and (2) general.

(1) At first the affected part feels hot, tense, painful, and is tender to the touch. Very soon a small circumscribed area becomes red, swollen, firm, and shining, and simultaneously the subjective symptoms (pain, heat, etc.) become aggravated. The *point of election* is usually on the nose, but it may be on the ear, the face, or elsewhere about the head, and thence the inflamed, swollen zone spreads, chiefly in the direction of one or the other side of the head. Separating the diseased from the unaffected skin there is a sharp line of demarcation—an elevated brawny ridge; this ridge presents a "zigzag irregularity of outline, like the burned edges of a sheet of paper" (Warren). While the inflammation is advancing there may be noted, beyond the border of the latter, little red streaks and spots that grow in area till at last they become confluent. The degree of redness increases in intensity as the case advances, but any natural prominence or fold in the integument may prevent further extension of the inflammation (*e. g.*, naso-labial folds, border of the hairy scalp). In cases of *average severity* the face is much swollen, the eyes closed on account of tumefaction of the eyelids, the ears greatly enlarged (far better marked on one side than the other, as a rule), the scalp swollen and tender, and the facial lineaments often changed beyond recognition. In a minority of the cases the inflammatory process extends from the head to the arms, to the trunk, and even to the lower extremities (*erysipelas migrans*), and in such instances the face may be healed while the disease is yet extending over other portions of the body. When the disease is arrested the peripheral ridge ceases to extend and grows pale; the inflammation then subsides.

The epidermal layer may become elevated over circumscribed areas, giving rise to larger or smaller vesicles or bullæ (*erysipelas vesiculosum*). Suppuration may attack these large vesicles, whereupon they fill with pus (*erysipelas pustulosum*). From intense infiltration the part or parts may become gangrenous—*erysipelas gangrænosum*. Enlargement of the cerebral lymph-glands is common. Desquamation follows erysipelas, and the complexion is more delicate than before the attack.

(2) **General Symptoms.**—With the *onset* of the attack the patient is seized with repeated fits of chilliness; less commonly, a severe rigor occurs. Immediately, and more rapidly than before, the *temperature* rises to a height of 104° or 105° F. (40°–40.5° C.) on the evening of the first day. As a rule, the temperature reaches its maximum (105° to 107° F.—40.5° to 41.6° C.) on the third evening. Marked nocturnal remissions of temperature (2° to 5° F.—1.1° to 2.7° C.) are the rule, but the evening temperature may in rare instances be to an equal degree lower than the morning. At the end of a week the temperature declines rapidly to normal, and usually within twenty-four or thirty-six hours; sometimes, however, the course of the fever is prolonged and defervescence may be less critical. In erysipelas migrans a long and decidedly irregular temperature-curve is presented, and the same remark applies when complications are present. The *pulse* is frequent, of good volume, and soft. I have been able to confirm the observations of Da Costa, Strümpell, and others that the cutaneous inflammation in erysipelas (particularly erysipelas migrans) may advance to a slight extent even after the temperature has returned to the normal grade.

The *tongue* is furred, the anorexia intensified, and nausea and vomiting (due to sepsis?) occur. The *bowels* are usually constipated, though I have observed a few instances in which marked diarrhea developed at a late stage of the attack. The inflammation may extend to the mucous membrane of the throat and larynx, causing swelling and edema of the parts. It may also involve the serous membranes, though rarely. The *nervous symptoms* may or may not be conspicuous; but there are apt to be intense headache and restlessness, with some mental aberration at night. Actual nocturnal delirium appears in the severer forms, and in erysipelas occurring in drunkards delirium tremens may suddenly develop. The *urine* presents the usual febrile characters. Commonly it contains a little albumin, and rarely acute nephritis occurs as a complication. A polymorphonuclear leukocytosis, parallel with the severity of the infection, occurs in erysipelas. The blood must come from the warmed finger, however (Chantemesse and Ray[1]).

There is a direct correspondence between the intensity of the local and constitutional disturbances in this disease. Often in severe forms (such as are apt to arise in old, much enfeebled, or intemperate persons) of facial erysipelas the *typhoid* (adynamic) *condition* is developed.

Complications and Varieties.—An analysis of 1674 cases of erysipelas with particular reference to complications gave an interesting series of results, and one at variance with the notions of most authors. Some are given here in the order of frequency of occurrence: Abscess, 105; rheumatism, 20; delirium tremens, 10; lobar pneumonia, active delirium, phlebitis, pleurisy, each 7; acute nephritis, 6; synovitis and diarrhea, each 5; tonsillitis, 3; catarrhal pneumonia, otitis media, pharyngitis, edema of the larynx, acute bronchitis, each 2.[2]

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[1] *Presse méd.* July 1, 1899; *Saunders' Year-Book* for 1901.

[2] "The Complicating Conditions, Associated Diseases, and Mortality-rate in Erysipelas," by the Author: *The Int. Med. Mag.* for Oct., 1893.

The fact that acute articular rheumatism is a relatively frequent complication of erysipelas is worthy of special notice, for the reason that the attention of the profession has not hitherto been called to it. The symptoms of rheumatism usually come on several days after the onset of erysipelas. In a few instances pneumonia appeared early, being due most probably to special localizations of the specific streptococcus. To such cases the term "pneumo-erysipelas" may be appropriately applied. The cases—2 in number—in which acute nephritis developed during the first few days of the attack should in like manner be termed "nephro-erysipelas." Meningitis was present in a single instance only.

Apart from the *varieties* referred to above, three other forms—namely, *phlegmonous* or cellulo-cutaneous, and *relapsing* erysipelas and *erysipelas neonatorum*—should be mentioned. The first exhibits an inflammation of the subcutaneous connective tissue, which tends to suppurate. Relapsing erysipelas constitutes the chronic form of the disease, recurring at longer or shorter intervals, and usually in the same locality. It is commonly due to some deep-seated focus of suppuration. Erysipelas neonatorum is the result of infection of the stump of the umbilical cord. From the navel the inflammation spreads to the thighs and genitals. There is fever, followed in a few days by fatal collapse, as a rule.

Sequelæ.—The hair often falls, but it is usually replaced by a fresh crop. Abscesses in various parts of the body, particularly the eyelids, are of common occurrence (*vide* Complications), and chronic otitis media and chronic nephritis may date from an attack of erysipelas. *Per contra*, erysipelas is reputed to be curative of certain affections (eczema, lupus, carcinoma, sarcoma, rheumatism).

Out of 476 cases collected by me relapses occurred in 54 (11.3 per cent.), and in 1 of these instances 5 relapses occurred; in 2 others, 4[1].

The **diagnosis** is made with ease after the eruption has fully developed, and its appearance, seat, and behavior, particularly the manner of extension of the brawny, ridge-like edge (best marked on the forehead), are the features that distinguish it from every other disease.

Erythema produces superficial redness, but is not attended with heat, swelling, or fever. *Urticaria* assumes the form of pale-red circular wheals, which cause marked itching and appear in successive crops, often disappearing in the course of a few hours. *Acute eczema* of the face, when intense, may somewhat resemble erysipelas; but it lacks the peculiar border and mode of progression so characteristic of the latter disease. Again, eczema produces particularly troublesome itching, and the swelling is less than in erysipelas. *Chronic erythematous eczema* is met with later in life, is without fever, without any considerable swelling or pain, and excites intense itching. *Eczema nodosum* is characterized by its nodosities in the vicinity of joints.

Course and Duration.—In my own experience, based upon 1880 cases,[2] the average duration (including the prodromal stage and period of convalescence) in persons under forty years of age is fourteen days. The course of the disease is much lengthened by complications, the preexistence of chronic affections, and by age (after the fiftieth year).

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[1] *Journal of the American Medical Association*, July 22, 1893.

[2] "Points in the Etiology and Clinical History of Erysipelas," by the Author: *Journal of the Am. Med. Assoc.*, July 22, 1893.

The **prognosis** is favorable, and it is rare for erysipelas to assume a malignant type. Perhaps the chief dangers lie in certain complications, especially extensive suppuration, pneumonia, acute nephritis, delirium tremens, etc. Acute articular rheumatism is comparatively harmless; but *previous debility*, especially if dependent upon chronic diseases, as syphilis, chronic rheumatism, gout, tuberculosis, organic disease of the heart, and the like, increases the percentage of deaths considerably. Again, age has a positive influence upon the mortality, which it augments moderately after the forty-fifth year, and most decidedly after the sixtieth year. Of 2663 deaths due to erysipelas (United States Census Report), the death-rate per 100,000 inhabitants was as follows: under 5 years, 31.34; 5 to 15 years, 0.81; 15 to 45 years, 2.80; 45 to 65 years, 8.88; 65 and over, 38.55 (Wm. L. Rodman). When death occurs it is due to exhaustion.

The *mortality-rate* is low, as shown by the results of my own collective investigations into the subject.[1] I found the general average death-rate to be 5.6 per cent., while in cases from private practice it was 4 per cent. In persons over seventy years it was 46 per cent. The traumatic cases gave a mortality of 14.5 per cent.

Treatment.—The treatment of erysipelas falls naturally into four subdivisions: (1) *Dietetic;* (2) *Constitutional;* (3) *Local;* (4) *Prophylactic.*

(1) **Dietetic.**—Proper attention to the diet is of the first importance. It must be generous and composed of highly nutritious articles, and if the temperature be high, only liquid forms of nourishment should be administered in definite quantities and at stated, brief intervals. Rectal alimentation should be resorted to if the stomach rejects a suitable dietary, and I feel confident of the fact that liberal feeding is of greater service to the patient than any of the recognized forms of medicinal treatment. Lack of attention to the patient's diet during the primary attack tends to increase the frequency of relapse. In persons over fifty years of age, and in those in whom the vital processes have been lowered on account of previous chronic diseases, correct alimentation is of paramount importance, often abridging the otherwise much protracted course of the affection.

(2) **Constitutional Treatment.**—When, despite an appropriate diet, the pulse becomes very rapid and feeble, the heart's first sound indistinct, and the tongue dry or brown, indications for the use of stimulants are present and must be heeded. Alcohol may be given with a comparatively free hand, 12 to 16 ounces (360.0–480.0) of whiskey daily in divided portions. Strychnin gives prompt results, and may be used in association with alcohol. In marked gastric irritability champagne is to be preferred.

The tincture of the chlorid of iron was first extensively used in this disease by English authorities, and was formerly regarded by most clinicians as a truly specific remedy. In 74 cases of erysipelas which were treated by this remedy alone, the average quantity being 1 dram (4.0) daily in divided doses, in the Pennsylvania Hospital by Drs. Lewis, DaCosta, Longstreth, Meigs, and others, the death-rate was 4 per cent.[1] Other preparations of iron, however, are equally efficacious. Quinin is a valuable remedy in erysipelas, and during the past twelve years I have employed it in not less than 30 cases, confining its use to instances in which the temperature touched 103° F. (39.4° C.), and, with a single exception, in uncomplicated cases (22 in number) the nocturnal remissions were decidedly greater. In every instance iron in some form was administered simultaneously. J. M. DaCosta first used pilocarpin in erysipelas at the Pennsylvania Hospital. His experience showed that when given hypodermically (gr. $\frac{1}{6}$–0.010) in the very early stage, and repeated three or four times at intervals of two or three hours, it often aborted the attack. If we except this use of the drug, it is only in cases attended with high temperature with slight morning falls that pilocarpin should be employed; and the condition of the pulse and heart can be relied upon as a guide to its administration.

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[1] "The Treatment of Erysipelas," by the Writer: *Therapeutic Gazette*, July 16, 1894.

Numerous *antiseptic* remedies have been recommended.

Antistreptococcic Serum.—André, Robinson, Cox, and others have reported instances of its successful use. The serum is injected subcutaneously; its influence endures over several days, but it is important that the injections are repeated at forty-eight hour intervals. Marmorek's serum (care being taken that it is not too old) is to be preferred, and it is probable that it has immunizing power as well as a specific action as a prophylactic and curative agent.

Certain *symptoms* demand internal medication. When the fever, as sometimes happens, is alarmingly high, its reduction must be accomplished, and the best method is by means of cold spongings combined with the ice-cap, or cold or gradually cooled baths. Guaiacol applied externally has recently been employed for the purpose of reducing the temperature, and found highly efficacious.

For *marked nervous phenomena*, such as pain, sleeplessness, and active delirium, hyoscin hydrobromate (gr. $\frac{1}{100}$—0.0006) has been tried hypodermically in numerous cases at the Medico-Chirurgical, Pennsylvania, and Philadelphia hospitals, and has given promise of being a valuable remedy. It should not be employed when the heart-power is deficient, and to fulfil the same indications we may utilize the following: Sodium bromid, gr. v (0.324) every two hours, or gr. xx–xxx (1.296–1.944) at night; morphin, gr. $\frac{1}{2}$ (0.008), and chloral, gr. x (0.648), in combination every half hour for three doses; potassium bromid, gr. x (0.648), and tincture of cannabis indica ♏x (0 666), in combination; atropin, gr. $\frac{1}{80}$ (0.0008), and morphin, gr. $\frac{1}{6}$ (0.0108), hypodermically.

The treatment of the various *complications* must be conducted in accordance with general principles applicable to each.

3. **Local measures** have always held a prominent place in the treatment of erysipelas. The list of agents that have been used topically is long. In my paper previously cited it is stated that in the three series (247) that were treated at the Pennsylvania Hospital, together with a few collected from other sources, no less than fifty different remedies and preparations had been employed locally. Among those most frequently used were elm (37 cases); lead water and laudanum (20 cases); carbolic acid (1 to 40), injected subcutaneously (18 cases); zinc oxid (14 cases); mercuric-chlorid solution (14 cases); ichthyol ointment with lanolin (8 cases), etc. Many of these preparations were prescribed for their effect in excluding the air—a leading indication. This I am in the habit of meeting by the use of carbolized vaselin or cool carbolized oil.

A knowledge of the microbic nature of erysipelas has led to the local application of numerous antiseptic remedies, and it is along this line that the greatest advances in the treatment of the disease are to be expected. Mention has been made of the method of injecting carbolic acid. Here the aim is to check the spread of the inflammatory process by inserting the needle at numerous points just beyond the inflamed border. The method (introduced by Heuter) has been much practised by Henry at the Philadelphia Hospital, and more recently by Osler at the Johns Hopkins Hospital, and is especially applicable in erysipelas migrans. In the statistics before given a solution of mercuric chlorid (1 : 4000) was used locally in 14 instances, to which I can add the results of 12 others at the Medico-Chirurgical Hospital and in private practice. In a few cases it was injected beneath the skin, as in the case of the carbolic acid. More recently it has been recommended to scarify the affected part and follow with the application of a solution of mercuric chlorid. In view of the fact that the streptococcus is found chiefly in the more superficial channels of the corium, it follows that it may be attacked directly by the mercuric-chlorid solution when the latter is used after scarification; and this method of treatment is at once most promising and rational. In 8 instances (3 of which have been previously reported) it was attended with brilliant results.

(4) **Prophylaxis** embraces isolation and care of the skin of the whole body. Bathing with a boric-acid wash (3 per cent.), at intervals of several hours, so as to disinfect the desquamating epidermis, removes a source of danger. It is probable also that relapses are sometimes due to autoinfection. Frequent change of the body-linen is to be advised and encouraged, and removal to another room during convalescence may likewise prevent a relapse.

DIPHTHERIA.

(*Diphtheritis; Angina Maligna; Croup.*)

Definition.—An acute, contagious disease caused by the Klebs-Löffler bacillus, and characterized, anatomically, by a croupous-diphtheritic faucitis, less commonly rhinitis and laryngitis. Clinically, it is characterized by irregular fever, prostration, and albuminuria; also by the secondary development of toxemia, and often cardiac failure. It is commonly followed by peculiar paralyses. In large municipalities it behaves endemically, and from time to time epidemically.

Pseudo-diphtheria.—There are forms of inflammation occurring most frequently in the pharynx and adjacent air-passages (and also in many other parts of the body) that are attended with the formation of a pseudo-membrane, and are not caused by the Klebs-Löffler bacillus. These cases have been studied exhaustively by Prudden and others, who have usually found the streptococcus to be the specific cause of infection. The latter, however, has been found occasionally in the pharynx of healthy children and in the inflamed mucous surfaces met with in erysipelas and measles. "Pseudo-diphtheria," so called, is very common in scarlatina.

Pathology.—The **true diphtheritic inflammation** has for its chief pathologic peculiarity the production of a fibrinous exudate. When the inflammation is superficial and of a mild grade, a croupous membrane is produced which can be easily removed from the mucosa, which it covers. Its formation is accompanied by a necrotic process that does not extend below, but practically replaces the epithelial layer of the mucous membrane. In the severer types of the affection, however, the fibrinous membrane infiltrates all the layers of the mucosa, which undergoes necrosis more or less nearly complete. In the severest forms the submucous layer may also become necrotic. It is to be borne in mind that the production of the fibrinous exudate in croup or diphtheria is always preceded by coagulation-necrosis of the epithelium. The mucous membrane surrounding the exudate is hyperemic, more or less edematous, and the seat of muco-purulent secretions.

The Pseudo-membrane.—Its composition comprises fibrin, pus, disintegrated leukocytes, flakes of necrosed epithelium, bacilli, and sometimes red blood-corpuscles. The fibrin has two main sources: (*a*) "The fibrinogen of the inflammatory matter," which transudes through the capillary walls; and (*b*) Disintegrated, migratory leukocytes, which form branching fibrillæ. Weigert holds that the inflammatory exudation is coagulated by a ferment derived from the disintegrated leukocytes.

The Klebs-Löffler bacilli are found, chiefly and in varying relative numbers, in the meshes of the fibrillæ, but also in the granular fibrin and on the adjacent mucous membrane. Frequently other micro-organisms are associated (streptococci, staphylococci, etc.). The membrane presents a grayish-white color, and, if croupous in character, can, as before mentioned, easily be removed. When the mucosa is deeply involved the membrane is thicker, firmer, and more adherent, so that its removal entire cannot be effected without great difficulty, and without, as a rule, injury to the surface, as shown by bleeding, etc. The character of the pseudo-membrane is affected by the nature of the underlying structure; thus in the pharynx it is firmer and less easily separable than in the larynx and trachea, where a distinct basement membrane is found (Flexner). As the membrane becomes older its color is apt to grow darker, becoming yellow or even dark brown. It sometimes becomes gangrenous, and softens or disintegrates, with the production of a very offensive brownish, semi-liquid excretion. The advancing edge of the false membrane is usually thin. On the other hand, when the process has become arrested the edge is apt to look raised or wrinkled, and later it may be distinctly curled up.

The membrane may extend downward into the ramifications of the bronchi. In such cases there is apt to be a lobular pneumonia, and this latter condition may occur without extension of the membrane. Occasionally there is a lobar pneumonia. A generalized bronchitis extending to the smaller bronchi is common from the irritation of aspirated substances. In rare cases the membrane has spread into the esophagus and even into the stomach.

After separation of a croupous membrane repair consists merely in a restoration of the epithelial layer—a process which is initiated by the fragments of epithelium that remain along the edges of the diseased area, and proceeds centrally. On the other hand, in true diphtheria, with necrosis more or less nearly complete of the mucosa and even the submucosa, sloughing occurs, and the missing structures are replaced by cicatricial tissues.

The Heart.—The muscular structure and the nervous mechanism suffer most. The histologic changes may be of the parenchymatous variety, but only in mild instances; whereas in severer cases fatty degeneration is conspicuous. In still other cases the chief pathologic characteristic is an interstitial myocarditis, and rarely the lesions of pericarditis and endocarditis have been noted. The heart is by no means always involved.

The **spleen** is commonly enlarged, though not to an excessive degree. The **blood** is dark, its coagulability is greatly diminished, and Canon and Frosch have in a few cases found the bacilli in the blood of those dying of diphtheria. The red corpuscles are somewhat decreased in number during the course of the disease, while the white corpuscles are increased. Bouchut and Dulinsay consider the grade of leukocytosis of prognostic value, and claim that it varies directly with the severity. Grawitz has determined in numerous cases a higher specific gravity of the blood during diphtheria. The **lymphatic glands** of the neck become swollen as a rule, and are often greatly enlarged, but they show little tendency to suppurate. In pronouncedly septic cases in which a mixed infection is found by culture a good deal of tumefaction of the neck occurs, this sometimes even obliterating the normal contour from jaw to clavicle.

The Kidneys.—The kidneys show degenerative changes, the usual *kidney-lesion* being a hyperemic swelling with edema of the interstitial tissues, and often hemorrhagic spots in the cortex. Sometimes there is a marked glomerulo-nephritis, and more rarely a diffuse granular degeneration of the epithelium. Minute areas of necrosis have been observed in the internal organs, in which fibrin has been found deposited (Oertel). Welch and Flexner have produced, by artificial inoculation upon guinea-pigs, kittens, and rabbits, foci of cell-death in the lymph-glands throughout the body, in the spleen, liver, lungs, heart, and intestinal mucosa. When the dose is small and the animal lives several weeks, the paralysis which belongs to the disease may develop.

The **nerves**, in cases of paralysis, have shown parenchymatous and interstitial inflammatory lesions. In paralysis of throat-muscles (*i. e.* those near the locality of the pseudo-membranous inflammation) the latter show also round-cell infiltration and fatty degeneration of the fibers.

Etiology.—True diphtheria is caused by the Klebs-Löffler bacillus, and all cases of supposed diphtheria in which the bacillus is absent are to be regarded as non-diphtheritic. The etiologic is, therefore, quite different from the pathologic significance of this term. Recent researches have removed all doubt as to the specific nature of the Klebs-Löffler bacillus.

Bacteriology.—The bacillus diphtheriæ nearly equals in length that of the bacillus tuberculosis, and is twice the diameter of the latter. It has rounded extremities, which are also frequently bulbous, giving it the appearance of a dumb-bell. At times one end only is clubbed, or, more rarely, one or both ends appear pointed. The bacilli are immobile, do not form spores, and stain readily, the best agent being alkaline methyl-blue. Their manner of taking the stain is important. The bacilli show alternating segments of darker and lighter stained areas, and often minute dots showing a most intense and deep staining. They grow on most culture-media, but for clinical purposes Löffler's blood-serum is important (3 parts blood-serum and 1 part neutral or slightly alkaline nutritive bouillon, containing 1 per cent. of glucose). Inoculated on this, they outgrow all other organisms that may be present, and within eight hours or less show numerous spots, one-half to one millimeter in diameter, which have a dull surface and a dense white or somewhat yellowish color. There are usually present also smaller points which have different appearances and which are colonies of other organisms. The former are the colonies of the bacillus diphtheriæ, and from these microscopic preparations and (by further cultivation) pure cultures can be obtained. The bacilli are semi-anaërobic, and thrive at the temperature of the human body; a temperature of 122°–136.5° F. (50°–58° C.) causes their destruction in ten minutes.

Pseudo-diphtheria Bacillus or Bacillus Xerosis.—From many cases, often showing no lesions, an organism may be obtained that is identical in appearance, manner of culture, growth, etc. with the bacillus diphtheriæ, but inoculation with it causes no lesions. The works of Abbott, Roux, Yersin, and others seem to show that this is an attenuated form of the true bacillus, and varying grades of pathogenicity may be found between the two. The distinction from the pathogenic bacillus can only be made by determining the lack of infection after inoculation.

Site of Infection.—In the human family the seat of election of the bacillus diphtheriæ is usually the faucial mucosa, and less frequently other mucous surfaces and abraded skin. The bacilli do not penetrate the mucosa, and hence do not find their way into the lymphatic or circulatory system, but remain at or very near the site of the local changes.

The Toxins.—Toxins are absorbed from the diseased spots by the lymphatics and blood-vessels, and produce the general phenomena in uncomplicated cases. They have been isolated from artificial cultivations of the microbe, and when inoculated the chief ptomain of the Klebs-Löffler bacillus so modifies the solids and liquids of the body as to render the subject immune (Behring). Another, however, if employed in like manner, produces dangerous and even fatal symptoms (convulsions, paralysis, etc.).

It is certain that the bacillus can maintain an existence for months outside of the body, though its usual habitat is unknown unless it be the organic constituents of the superficial soil. The virulence of its products is modified by many individual conditions, and chief among these is a healthy and intact condition of the mucous membranes, which greatly reduces the susceptibility to the disease.

Associated Microbes.—With the Klebs-Löffler bacillus are frequently found other microbes, especially streptococci and staphylococci. These pass beyond the site of local infection, reaching the internal viscera and other structures, and, as will be seen hereafter, give rise to the serious septic element of the disease. W. Bloch and P. Sommerfield,[1] in studies on the pathogenicity of the Löffler bacillus, have verified the accepted statement with reference to the germ, their article being a good exposition of the present status of the bacteriology of diphtheria. From a study of 436 cases, the authors state that the Löffler bacillus was never found in culture, but always associated with other bacteria, among which streptococci played the greatest part. The two doctrines concerning the relation of streptococci to septic diphtheria are given, the one being that the streptococci increase the virulence of the diphtheria bacillus and cause sepsis by gaining access to the circulation; the other is that the diphtheria toxin, by its effect on the organism, prepares the way for an invasion by streptococci.

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[1] *Arch. f. Kinder.*, Bd. li. Heft 1.

Modes of Infection.—When the bacillus leaves the body of the sick it is contained in particles or shreds of the diphtheritic membrane, or in the expired air. Infection may then occur (*a*) *By direct contact* with the shreds of membrane thrown off—*e. g.* when the latter are ejected by coughing and lodge upon the conjunctivæ or faucial mucosa of bystanders. Under this category come the cases in which the deadly poison is transferred to the physician and attendants, with resulting infection, from the sucking of tracheotomy-tubes. (*b*) *By inhaling the air* surrounding the patient (contagion). Infection by contagion, however, does not extend beyond a radius of a few feet from the patient. (*c*) A very leading manner of conveyance of the bacillus from the sick to the healthy is by *fomites*. The contagion adheres tenaciously to a great variety of objects (toys, clothing, library books, letters, slates and drinking-cups in the public schools, etc.), and in this way the germs of diphtheria have been transferred over great distances and have given rise to the disease long after. The latter fact renders it difficult to trace certain cases to previous ones, to which they invariably owe their origin. (*d*) *Sewer gas, per se*, is to be regarded as non-pathogenic, or at least so far as this affection is concerned (Laws). It may, however, become a carrier of diphtheritic poison. (*e*) I regard it as highly probable that the disease may be communicated by *domestic animals* (fowls, cats, etc.).

As to the exact conditions under which infection occurs, our knowledge is as yet incomplete. We know definitely the usual point of local infection in man, and also that a catarrhal mucosa or an open lesion of a mucous surface invites infection. It is not certain, however, that even a slight lesion of the mucous surface is essential to infection, though it is very questionable whether the diphtheritic germs ever find lodgement in the perfectly healthy mucosa. Some writers claim still that the Klebs-Löffler bacillus may enter the blood through the respiratory system and give rise to primary constitutional symptoms, the local manifestations in the throat being secondary. I have met with a single instance that would lend support to this view.

Predisposing Factors.—(1) *Age.*—This is the most important factor, diphtheria being, in the main, a disease of childhood. Most cases occur between the second and seventh years, while the receptivity diminishes rapidly after the tenth year. Instances have, however, been observed up to the fiftieth or even the sixtieth year. During the first year of life also it is rare. (2) *Sex.*—This is without appreciable influence. (3) *Season.*—Cases are more numerous in winter and spring than at other seasons. (4) *Climate.*—Diphtheria is met with less frequently in tropical than in temperate and cold climates. Humidity favors the propagation of the diphtheria germ, and hence damp cellars also promote the spread of the disease. (5) *Unhygienic Conditions.*—Unfavorable sanitary surroundings tend to lower vitality, thus increasing the susceptibility to the specific virus. Most epidemic outbreaks have held more or less intimate relationship with decomposing organic matter, defective drainage and sewage, cesspools, etc., though it is to be especially remembered that the disease often prevails in sparsely-settled rural districts.

Immunity.—A single attack does not confer perfect immunity. Second and third attacks not infrequently occur in the same individual.

Symptoms.—**Incubation.**—The *duration* of this period is from two to seven or ten days, and in a small percentage of the cases it may be longer. In virulent epidemics and when the disease is produced experimentally the incubation-stage is short—from twelve hours to two or three days. The *prodromal indications* of diphtheria are not strikingly characteristic. They may either be acute in character or very mild; but usually the child will complain of feeling weary and indisposed to play, of being chilly and cold, and of pain in the head, back, and limbs. In young children the onset of diphtheria, as in other infectious diseases, may be marked by *convulsions*. There is nothing in this early stage of the disease to distinguish it from many of the other affections of children, such as simple pharyngitis or tonsillitis. There may be some fever, not very high—an elevation of one or two degrees at most. The child may often complain of discomfort in swallowing, and on examination the fauces will be found to be reddened, and in a short time the exudate will be found on the tonsils or soft palate. This is the usual type of **simple tonsillar diphtheria.**

Pharyngeal Diphtheria.—The symptoms are usually *slower of development* than in tonsillitis. The child is sluggish, looks heavy-eyed, languid, and pale for several days. The *fever* may not rise above 101° or 102° F. (38.8° C.). On examining the throat, however, it is found to be swollen and red, and if *lividity* is more pronounced than the swelling, it suggests the true nature of the disease. The *membrane* begins on the tonsils in the form of small patches of yellow exudate, scarcely distinguishable from the thick, cheesy plugs of inspissated dead epithelium and secretion which issue from the mouths of the follicles of the tonsils during the course of acute or chronic tonsillitis. The membrane spreads from the tonsils to the soft palate and half arches within a few days, and it may also appear on the pharyngeal wall. During this stage the throat may become much swollen and the tonsils greatly enlarged, frequently meeting in the median line. The *glands* immediately beneath the angle of the lower jaw on one or usually both sides become hard, painful, and slightly enlarged; the swelling of these glands is not usually great in mild forms, although their presence, in association with the foregoing symptoms, is an infallible indication of the disease. The child, as a rule, shows grave constitutional symptoms for a few days, and loses its appetite. The *temperature* is not characteristic, as a rule not being high, and the pulse is rapid and weak, being out of proportion to the general indications of the disease. In mild cases the symptoms abate by the end of the first week, and the pseudomembrane separates, leaving a red, inflamed surface behind. The child is prostrated for a number of weeks, and in about 20 per cent. of all mild cases the toxic effects of the disease may show themselves in the form of a neuritis, with its accompanying paralysis.

Variations in Manifestation.—Diphtheria may exhibit a number of variations as regards the seat of attack and the severity of the poisoning. In some epidemics the Klebs-Löffler bacillus seems to be more active and more numerous, or perhaps more virulent, than in others. The severity of the attack does not seem to depend on the amount of the pseudo-membrane, but rather, according to Rotch, upon three factors: (1) the virulence of the bacteria; (2) the local resistance; and (3) the general resistance. While false membrane is most frequently seen on the tonsils, spreading gradually to the soft palate and uvula, the mucous membrane of any part of the body (lips, tongue, conjunctivæ, vulva, or glans penis) may be the seat of the growth.

Malignant Diphtheria.—The symptoms are severe from the commencement. There are one or at most two days of slight illness, and then alarming symptoms manifest themselves, *cardiac failure* possibly setting in without a specially severe local lesion. *Vomiting* and *high fever*, resembling the onset of scarlet fever, may initiate the attack; and within a few hours we may find extensive swelling at the angles of the jaws, with a feeling of stony hardness, a very offensive, bloody discharge coming from the nostrils, accompanied with difficulty in opening the mouth. If the *throat* is examined, there will be found extensive swelling of the tonsils, even to meeting, the uvula and soft palate being edematous and covered with much sloughy-looking membrane. The *temperature* in severe cases soon reaches a point between 103° and 104° F. (40° C.), while the *heart-beats* become exceedingly feeble. In a day or two the cellulitis extends, the face becomes edematous, the skin pits all over the face, neck, sternum, and chest-walls. The patient soon becomes drowsy, cyanotic, and occasionally an erythematous rash appears about the face, neck, and chest, while a purpuric rash is not infrequent in malignant cases. Death occurs in such cases within one week from toxic poisoning. Malignant cases of diphtheria resemble very closely malignant scarlet fever, though the pulse in scarlet fever will be of assistance in the discrimination in the absence of the characteristic rash.

Nasal Diphtheria.—In all severe cases of pharyngeal diphtheria the inflammatory process is likely to extend to the nasal mucous membrane. In some cases the nasal mucous membrane is found to be the first involved, and it may spread to the tonsils, but in these cases the exudate will be found to involve the back of the soft palate and pharynx as well. In many cases of nasal diphtheria no membrane may be found during life; there may be only a purulent discharge with blood, the presence of which in the nasal passage obstructs breathing, giving rise to a bubbling sound, and rendering sleep troublesome and noisy. Many cases have also been reported of formation of pseudo-membrane in the nose with mild general symptoms (often insignificant), and from which bacilli identical with diphtheria bacilli were obtained by culture, the bacilli often persisting for months. Sometimes the cases have recurring mild attacks of pseudo-membranous inflammation of the nose, while the bacilli may be constantly present. It is probable that these cases may give rise to infections of like nature, and even of true diphtheria. In nasal diphtheria the symptoms are quite as severe as in faucial diphtheria, and in cases in which the soft palate, tonsils, and nasal mucous membrane are involved the general symptoms, the depression, and also the albuminuria, are well marked. In all cases of coryza with fever we should be guarded as to opinion, especially if an epidemic of diphtheria is prevalent at the time. The diphtheritic inflammation may spread from the nose to the conjunctivæ, with the formation of a false membrane, and much purulent discharge may escape from the eyes, the lids of which may be greatly swollen. In this place it is well to remember that in *measles* we sometimes have a form of membranous exudation occurring on the nasal mucous membrane and as a primary disease—"rhinitis fibrinosis"—which is not always diphtheria. This disorder runs a favorable course, the membrane being less adherent than in diphtheria. Ravenel has collected 77 cases, and in 33 out of 41 cases examined bacteriologically the Klebs-Löffler bacillus was found. Constitutional symptoms were either slight or wanting.

Wound-diphtheria.—The bacillus will not live on normal skin, but when the skin is cut or bruised, as after blistering or an eczematous condition, and when a moist, raw surface is present, the bacillus freely flourishes. Granulations also form a favorable soil. The diphtheritic germs may be introduced into the system during an operation, such as an excision of the tonsils, or even a vaginal examination; and in new-born infants the granulating surface left after sloughing of the cord may become the seat of diphtheritic inflammation.

Laryngeal Diphtheria or Membranous Croup.—In many cases the Klebs-Löffler bacillus produces its influence first on the mucous membrane of the larynx, and in these cases the mucous membrane of the nose and pharynx may never give evidence of a false membrane. In laryngeal cases the first symptom is a *cough* of a *harsh*, *metallic*, *ringing character*, and never to be forgotten when once heard. The *temperature* may be slightly above normal, or even, in many cases, normal. The toxic absorption is slight, on account of the locality affected, and the constitutional symptoms are usually mild. The *local symptoms*, however, are very alarming, as they are the results of laryngeal obstruction, there being marked *dyspnea* with retraction of the intercostal and supraclavicular spaces, and later of the epigastrium and lower chest. These are associated with an increasing *cyanosis*. The child is soon very restless, is forced to sit up to breathe, and for the same reason bends forward with its head thrown back. In these extreme cases, unless relief is soon gained, the child dies of suffocation. In many instances a slower form of suffocation may result from the extension of the membrane downward to the bronchi.

Complications.—Local complications may be mentioned, as when we have *hemorrhage* from the nose and throat in the more severe ulcerative cases. *Skin-rashes* are not unusual, especially the *diffuse erythema*. Sometimes *urticaria* will be noticed, and in severe forms *purpura* will mark the skin.

Broncho-pneumonia is the most serious pulmonary complication of diphtheria. It is not produced by the Klebs-Löffler bacillus, but by pyogenic cocci which have been taken in during respiration. Broncho-pneumonia is frequent, and most usually terminates laryngeal cases that have been operated upon.

Albuminuria is really a part of the disease, and can scarcely be regarded as a complication. It is the most constant symptom, and is almost as certain in establishing a diagnosis of true diphtheria as a bacteriologic examination. It is met with in both mild and severe cases, and the greater the amount of albumin the more severe the case. When acute nephritis complicates diphtheria it is usually not accompanied by edema or anasarca.

Dysphagia may, by its constant existence throughout the disease, produce a profound impression on the general nutrition. Involvement of the *conjunctivæ* is a rare but grave complication.

Otitis media occurs frequently, and may be a troublesome complication as well as a sequel.

The most frequent **sequelæ** are anemia, chronic naso-pharyngeal catarrh, peripheral neuritis and its associated paralysis.

Anemia may so prolong convalescence that the child will frequently be exposed to some intercurrent disorder. The *chronic naso-pharyngeal catarrh* may be so marked as to offer a favorable ground for new diphtheritic invasion. *Neuritis* and *paralysis* will not be noticed until the third or fourth week, the paralysis usually being first seen when the child attempts to swallow, and the food, especially if liquid, is regurgitated through the nose. This is due to a paralysis of the muscles of the soft palate, which will also be noticeable owing to a peculiar alteration of the voice. The paralysis may take a general form, such as is seen in multiple neuritis, the lower extremities being affected and the knee-jerk absent. It is frequently quite extensive; it may extend to the external ocular muscles and cause squint, to the ciliary muscles and cause dimness of vision from unequal accommodation, or to the muscles of the trunk in general, producing widespread paralysis. The child, unable to hold anything, may stagger about as if intoxicated, so much so as to suggest the existence of a cerebral tumor. The disturbance of vision and the absence of the patellar tendon reflex has in adults led to a mistaken diagnosis of *locomotor ataxia*. Loss of taste, deafness, and a disturbance of sensation are not infrequent. Thus, paralysis is to diphtheria what dropsy is to scarlet fever—a proof positive of the disease. To make one step more, in many sudden deaths occurring in early diphtheria we must recognize *paralysis of the heart* outside of all toxic influence, and the fact that in cases of sudden death, which are by no means uncommon during the disease, we have some sudden disturbance of the vagus brought about by means of its cardiac branches.

The **prognosis** in all cases of post-diphtheritic paralysis is quite favorable. *Myocardial weakness* tends to supervene as a sequel. It is evidenced by the sudden accession of pallor, nausea, sometimes by vomiting, and also by weak heart-sounds and a feeble, broken, irregular pulse, etc.

Diagnosis.—The diagnosis of a pharyngeal diphtheria (the usual typical form) is not difficult if an epidemic be prevailing. The false membrane on the fauces and the presence of albumin in the urine give us a practically certain diagnosis. The only unequivocal evidence of the disease, however, is the finding of the Klebs-Löffler bacillus in the membrane.

Differential Diagnosis.—From *follicular tonsillitis* we differentiate diphtheria by the seat of the membrane, that of the former being *in* the tonsils, while diphtheritic membrane is *over* the tonsils and *over* the soft palate. Moreover, in follicular tonsillitis the fever is high, the onset is sudden, and it is usually associated with gastric disturbance. Albuminuria is generally present in diphtheria, while it is present in follicular tonsillitis in exceptional cases only. Moreover, mild cases may not present albuminuria, or fail to show the presence of albumin until later in the disease. The histories of the two cases are quite different. (For differential diagnosis between diphtheria and follicular tonsillitis, see also Table, p. 718.) In many instances of so-called diphtheroid lesions the membrane is formed only by streptococcus pyogenes (*membranous angina*), and these cases are sometimes of an intense grade.

Croupous or *membranous angina* may offer some difficulty; yet in this disease there is no tendency to spread to the nasal mucous membrane or to the larynx; there is a diminished glandular enlargement; there is no albumin, and the onset is more sudden.

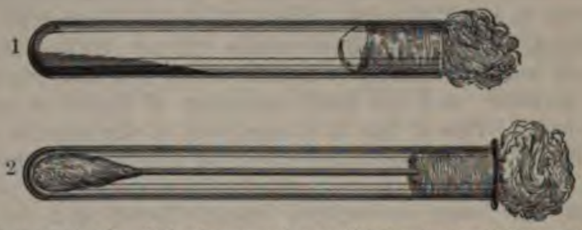

FIG. 17.—1, A tube of blood-serum; 2, a sterilized cotton swab in test-tube.

Rub the swab gently but freely against the visible exudate, and without laying it down, after withdrawing the cotton plug from the culture-tube, insert it into the latter, and rub that portion which has touched the exudate gently but thoroughly over the surface of the blood-serum without breaking its surface. Now replace the swab in its own tube, plug both tubes, and place them in the box provided by the health officials. This is to be sent to the bacteriologic expert. In laryngeal diphtheria the swab is to be passed far back and rubbed freely against the mucous membrane of the pharynx and tonsils.

Diphtheria frequently is associated with a rash, rendering it difficult to distinguish the condition from *scarlet fever;* but in diphtheria the rash is more truly an erythema, while in scarlet fever it consists of slightly raised points between which there may be an erythematous condition. The glandular swelling and sloughy condition of the throat, however, closely resemble diphtheria, and a positive diagnosis without a bacteriologic examination is often impossible. An immediate recognition of the disease is often possible from a smear-preparation of the exudate from the throat (see Fig. 17), the Klebs-Löffler bacilli being present in sufficient numbers to be readily distinguished by the microscopist. Park, who has had a rare experience with this affection, makes the following statement: "The examination by a competent bacteriologist of the bacterial growth in the blood-serum tube, which has been properly inoculated and kept fourteen hours at the body-temperature, can be thoroughly relied upon in cases in which there is a visible membrane in the tnroat if the culture is made during the period in which the membrane is forming, and no antiseptic, especially no mercurial solution, has lately been applied. In cases in which the disease is confined to the larynx or bronchi, surprisingly accurate results can be obtained from cultures, and although, in a certain proportion of cases, no diphtheria bacilli will be found in the first, yet they will be abundantly present in later cultures. We believe, therefore, that absolute reliance for a diagnosis cannot be placed upon a single culture from the pharynx in purely laryngeal cases." When a bacteriologic examination cannot be made the practitioner must regard as suspicious all forms of throat-affections in children, and carry out measures of isolation and disinfection. In this way alone can serious errors be avoided. Mistakes do not usually occur in a more pronounced membranous sore throat, but in the lighter types, many of which are in reality due to the Klebs-Löffler bacillus (Osler).

Prognosis.—Diphtheria is at the same time the most prevalent and most fatal of all the diseases with which the general practitioner has to deal. The mortality is enormous (30 to 40 per cent.), though it differs widely in different epidemics, and the most fatal variety is unquestionably the laryngeal. In laryngeal diphtheria the mortality may be as high as 75 per cent., and the younger the child the more unfavorable the prognosis, the strong and healthy seeming to share the same fate as the weakly. Of especially unfavorable prognosis are those cases that show large quantities of albumin in the urine, general adenitis, cervical glandular enlargement, excessive nasal discharge, a necrotic state of the throat, vomiting, and partial or complete suppression of the urine. Although the temperature in diphtheria is never very high, yet a sudden fall of temperature to subnormal and an irregular pulse, or bradycardia, are also a bad augury. Recovery from a severe attack in which there are extreme depression and much albumin is unusual, especially in a child under six years of age, though recovery takes place frequently in what would be regarded as hopeless cases. The results of Morse's extensive observations are opposed to those of Bouchut and Dulinsay, who claim that the degree of leukocytosis is of prognostic value (see p. 183). The cases of neuritis invariably recover. A child who has had diphtheria once is most likely to contract it again, and if he recovers is liable to suffer from its effects for years.

The *causes of death in diphtheria*, in their order, are as follows: membranous croup or laryngeal diphtheria; septic infection, which may be a slow death; sudden heart-failure—paralysis of the heart; broncho-pneumonia, following tracheotomy or occurring during convalescence.

Treatment.—**Prophylaxis.**—The best preventive measures against diphtheria are a clean nose and mouth. Insist upon a careful toilet of the nose in all children. The slightest appearance of a coryza must be overcome at once by the use of a mild antiseptic wash; all accumulations of crusts, dust, dried blood, etc. should be removed from the nose twice daily, especially in children attending school or during the prevalence of an epidemic. The child should be early taught to employ a small antiseptic gargle as a daily routine, using a weak solution of hydrogen dioxid, listerin, or even a mild dilution of alcohol. The teeth should be carefully cleaned daily, and all decaying teeth should be filled or removed. If it is true, as one authority claims, that over two hundred different species of bacteria find a happy home in the oral cavity, this fact should make all parents attentive to the proper physiologic condition of the mouths of their children.

All cases of sore throat should be examined for the Klebs-Löffler bacillus, and, if it is found, the individual should be isolated; and all cases of diphtheria should be kept isolated until the membrane has disappeared from the nose and throat. This is especially true in schools and asylums. Moreover, the throats of all persons exposed to this disease, and of those caring for diphtheritic patients, should be frequently examined for the Klebs-Löffler bacillus, and if it be found the person should receive immunizing doses of antitoxin. The fact that the Klebs-Löffler bacilli when found in healthy throats may not be active is no argument against isolation, because it is well known that if the same germs were to find such favorable soil as a broken or catarrhal membrane they would rapidly develop. The seed being there, the soil only requires preparation for its reception.

An unrecognized feature in the prophylactic treatment of the disease is seen in the uncertain period of convalescence. It frequently happens that long after all membrane has disappeared active bacilli may still cling to the throat. This condition may continue from two to six months, and even longer in deeply fissured tonsils; and the disease may be communicated by such throats in the act of kissing young children or adults with sensitive throats or with a broken mucous membrane of the mouth. For this reason the indiscriminate kissing of young children on the lips should be interdicted by the physician.

Sufficient importance has not been been given to the milder cases of diphtheria as to their isolation and disinfection, and this fact explains the occurrence of many house-epidemics.

Treatment of the Attack.—The treatment falls very naturally under several departments: (*a*) the hygienic measures to limit the diffusion of the disease; (*b*) the local management of the throat to destroy early the toxic germs; (*c*) medication to antagonize the effect of the toxins, and eventually to overcome the complications and sequelæ.

(*a*) **Hygienic Treatment.**—The patient should be in a room well exposed to sunlight and fresh air, as diphtheritic germs grow well in poorly-lighted and damp chambers. No stationary washstand should be allowed in the room, and Goodhart well says that many cases seem to have their origin in the proximity to foul-smelling drains. The physician should never consent to be responsible for the recovery of a patient in a room in which there is a washstand with its uncertain connection with the main sewer. If possible, the patient should use two connecting rooms, one during the day and the other at night, so that one while not in use may be thoroughly aired and disinfected. Even in mild cases the patient should be kept in bed throughout the attack, and in more severe cases also for some time during convalescence. This is especially important when there have been symptoms of cardiac depression during the acute stage. The general comfort of the patient is enhanced by two daily sponge baths of tepid salt-water or of alcohol and water.

Feeding.—Nursing infants may be fed on breast-milk obtained by a breast-pump, but should not be placed at the mother's breast (Holt). The feedings should be regular, yet lighter in quality and quantity than in health, remembering the tendency to vomit in all acute febrile affections, and the fact that gastric disturbance is closely associated with diphtheria. The rule must be, less solids and more fluids than in health.

Milk in some form being our main dependence, it should usually be diluted, and for young children partially if not wholly peptonized. The greatest difficulty comes in the latter part of the disease, when the child is septic and most likely has a strong objection to be disturbed. At this time vomiting is most easily provoked, and swallowing is rendered very difficult on account of the swelling and pain. We must not neglect the feeding even if it does cause discomfort, and here forced feeding by means of gavage is most valuable. Gavage is more desirable and likely to be more successful with children under three years than rectal alimentation. In older children, who object to the tube through the mouth, it may be passed through the nose with very little difficulty, and gavage by this route, even in intubated cases, will be extremely satisfactory. Concentrated broths, meat-juice, and even milk-punch or raw eggs, may be given in this way.

(*b*) **Medicinal.**—*Alcohol* no longer holds a debatable ground in the treatment of diphtheria: it is the most powerful drug in our possession to offset the ravages of the disease on the nervous centers and for the control of the circulation. Stimulation should be commenced as soon as there is a reasonable certainty as to the correctness of the diagnosis, and by commencing early with whiskey or brandy we may prevent the depressing effects of the poison of diphtheria as seen in the pulse and general condition of the child. The indications for alcohol are marked prostration, feeble pulse, and a weak first sound of the heart. The quantity must be adjusted to the age and gastric condition of the child, and usually one ounce (32.0) of good whiskey or brandy, well diluted, in twenty-four hours is sufficient for a child four years old. In very bad cases five or six times this quantity may be given, the only limit being the tolerance of the stomach. As a rule, the stimulant should be mingled with the food, as the child may rebel against taking both food and stimulant.

Strychnin stands next to alcohol in importance in the treatment of diphtheria, and usually it is given in too small doses. For a child four years old gr. $\frac{1}{30}$ (0.0021) may be given every six to eight hours, and may be administered in little tablets by the mouth or hypodermically.

Digitalis does not hold an important place in the heart-weakness of diphtheria, and yet it is strongly indicated on theoretic grounds. Clinically, it has been found to have an unfavorable action on the stomach before its good influence can be had on the heart itself. The same may be said of camphor and ammonium carbonate. The aromatic spirits of ammonia is valuable for rapid effects in syncopal attacks. In cases of threatened heart-paralysis occurring late in the disease Holt has found nothing so valuable as morphin employed hypodermically, the drug being given in full doses and repeated every two hours, keeping the child under its influence for some days.

Internal medication should be avoided until absolutely necessary, and such symptoms as vomiting or diarrhea are to be met with sufficient treatment only for their control.

(*c*) **Local Treatment.**—For the direct attack upon the membrane in the throat nearly all the remedies of the Pharmacopeia have been used. Gargling, swabbing, painting, spraying, and washing the throat, all have their advocates, and every physician has his favorite remedy or combination. And, as all adult pharyngeal diphtheria tends to recovery, it would seem reasonable that this form of treatment should not be neglected; yet since the acceptance of the antitoxin treatment medical opinion has suffered a decided change, especially as to the importance of local measures. The very best local application for pharyngeal or nasal diphtheria consists of hydrogen dioxid, diluted one-sixth, and used both as a gargle and spray as most convenient; this is usually sufficient in the early stage. The tincture of iron and glycerin is a valuable local remedy applied by means of a swab. The object of local treatment is a more thorough cleanliness—the prevention of systemic absorption of the ptomains. Hence a careful toilet of the nose and throat is important in preventing the spread of the disease. This part of the work is more easily directed than accomplished, especially in rebellious children, and we have frequently felt that new lesions were created in the mucous membrane of the nose and throat by an undue ardor in making applications. To avoid new lesions the spray alone should be used, and for the nose boric-acid solutions or hydrogen dioxid, 1:10, will be most serviceable. In this work the utmost tact and kindness must be maintained, for it is truly pitiable to force a struggling child, endangering the strength to accomplish so little. Warm, weak solutions, most thoroughly applied by means of the fountain syringe, will be better than the more frequent use of the hand-syringe. In older children who will use it a gargle of boric acid, listerin, or Dobell's solution, well diluted, may be used to keep the nose and mouth clean.

In laryngeal diphtheria the child should inhale an atmosphere laden with the vapor of slaking lime, or, whenever practicable, an atmosphere saturated with Löffler's solution (menthol 10 grams, dissolved in sufficient toluol to make 36 c.c., liq. ferri sesquichlorid, 4 c.c., absolute alcohol, 60 c.c.). J. Cordin warmly recommends mercurial fumigation for the relief of laryngeal stenosis. The development of the signs of actual stenosis, as shown by stridulous breathing, cyanosis, etc., furnishes an indication for either intubation or tracheotomy. According to my observations, the results of intubation have been quite favorable, and I would strongly recommend a trial of this procedure before resorting to tracheotomy (see temperature-chart, Fig. 18).

(*d*) **External Applications.**—External applications to the throat have no effect on the course of the disease. They are useful, however, in relieving the pain and the swelling in the lymph-glands. Careful massage of the neck with camphorated oil, as hot as the skin will tolerate, is very soothing; and soap liniment may be used in the same way, or if much pain exists chloroform liniment may be substituted. Poulticing for the relief of pain is not desirable, as it seems to favor suppuration. In older children the ice-bag has been used with good effect, and it soon brings grateful relief from the tension and subdues inflammation. All manipulations about the child, however, should be carried on as gently as possible, so that its rest may not be disturbed.

Serum-therapy; the Antitoxin Treatment.—This has now passed beyond the stage of uncertainty and experimentation, and must be regarded as one of the most positive advances made in practical medicine. Its utility rests upon the discovery that animals may be rendered immune to diphtheria, and that the blood of an animal so treated, when introduced into another animal, protects the latter from infection by the diphtheria

bacilli. The studies of Behring, Roux, Kitasato, and others have demonstrated that the use of the blood-serum of the lower animals, artificially rendered immune against diphtheria, has a powerful healing influence upon diphtheria that has been contagiously or spontaneously acquired by man. These experiments were first published in December, 1890. The principle was first shown to be true of tetanus, and, late in 1892, Behring further showed that the blood of an immunized animal had the power both of protecting and of curing susceptible animals which had been inoculated either with the toxins or the bacilli of diphtheria. In preparing the blood-serum it is very desirable, of course, to have a uniform strength or standard. One-tenth of one cubic centimeter of what Behring calls his normal serum will counteract ten times the minimum of diphtheria poison, fatal for a guinea-pig weighing three hundred grams. One cubic centimeter of this normal serum he calls an antitoxin unit. The serum prepared by his method is labelled in three strengths. No. I. is sixty times the strength of the normal serum; No. II. is one hundred times as strong; and No. III. is one hundred and forty times as strong. To a child of two years or over not less than 800 or 1000 units should be administered at the first dose; hence solution No. I. is rarely employed at the present day. Should a favorable result not be attained, then, on the following day, 1500 to 2000 units should be administered, and a third dose after a similar interval if necessary. The latter dose should be employed at the outset in very severe cases and in those not seen until they are far advanced. The sites to be selected for injection are various. In very young children either the buttock or thigh is to be preferred, while in older children the flanks or subscapular spaces may be chosen as well. The injections should be made deeply into the subcutaneous cellular tissue.

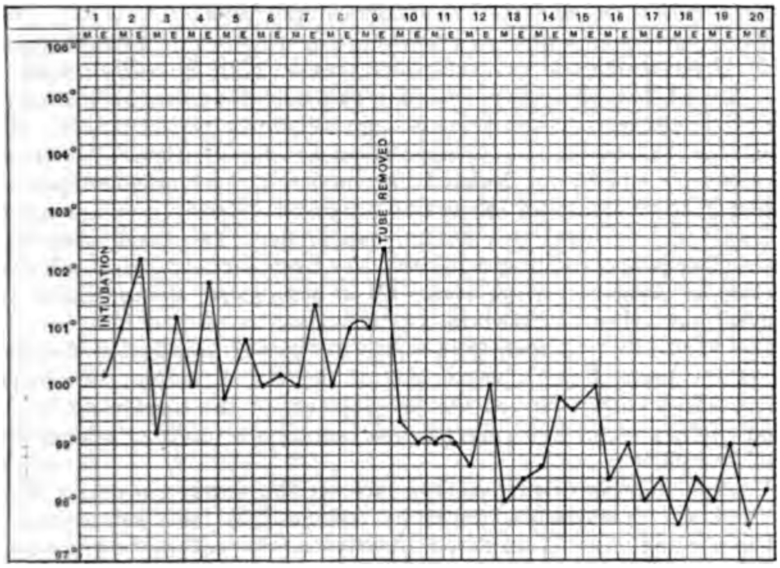

FIG. 18.—Temperature-chart of a case of diphtheria.

In fortunate cases the influence of the serum soon becomes apparent. Within twenty-four hours the faucial swelling diminishes, the membrane exfoliates, the temperature falls, the pulse becomes slower and stronger, and the general condition of the patient quickly improves. In cases of moderate severity and when injections are employed early the improvement in the throat and the constitutional symptoms is very decided; and the earlier the case comes under treatment the better are the results. There are, however, many cases of great severity in which the antitoxin has been used early, and yet has not shown any benefit.

A danger in serum-therapy may be the development of local abscesses, which, if full antiseptic precautions be taken, must be rare indeed. I have escaped them altogether. Certain skin-eruptions have been observed after injections, mostly urticarial, though sometimes scarlatiniform. The latter form has given rise to apprehensions of scarlatina. Widerhofer had one case which was isolated as measles, but never developed any symptoms other than the suggestive eruption. Rarely, joint-pains and swellings, with general prostration, supervene. Two fatal cases have been reported—one[1] that of a healthy boy five years old, the result of an injection of Behring's fresh serum as a preventive, dying within five minutes; the other occurred in Berlin.[2]

For establishing immunity in subjects exposed to infection the injection of 60 units (1 cubic centimeter of the No. I. serum) affords protection. In order to arrest the development of the disease during the period of incubation 100 units (1 cubic centimeter of No. II. serum) is probably sufficient.

A large number of preparations are on the market, many of which are good, yet great caution must be exercised in their selection.

The use and value of antitoxin in private practice are best shown in the following summary of the report of the American Pediatric Society's investigation of the subject:

1. The report includes returns from 615 physicians. Of this number more than 600 have pronounced themselves as strongly in favor of the serum-treatment, the great majority being enthusiastic in their advocacy.
2. The cases included have been drawn from localities widely separated from each other, so that any peculiarity of local conditions to which the favorable reports might be ascribed must be excluded.
3. The report includes the record of every case returned, except those in which the evidence of diphtheria was clearly questionable. It will be noted that doubtful cases that recovered have been excluded, while doubtful cases that were fatal have been included.
4. No new cases of sudden death immediately after injection have been returned.
5. The number of cases injected reasonably early, and in which the serum appeared not to influence the progress of the disease, was but 19, these being made up of 9 cases of somewhat doubtful diagnosis, 4 cases of diphtheria complicating measles, and 3 malignant cases in which the progress was so rapid that they had passed beyond any reasonable pros-

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[1] *Journal of the American Medical Association*, April 4, 1896.

[2] *Medical News*, April 18, 1896.

pect of recovery before the serum was used. In 2 of these the serum was of uncertain strength and of doubtful value.

6. The number of cases in which the patients appeared to have been made worse by serum was 3, and among these there is only 1 case in which the result may be fairly attributed to the injection.

7. The general mortality in the 5794 cases reported was 12.3 per cent., and, excluding all cases moribund at the time of the injection or dying within twenty-four hours, it was 8.8 per cent.

8. The most striking improvement was seen in cases that were injected during the first three days. Of 4120 such cases the mortality was 7.3 per cent., and, excluding cases moribund at the time of the injection or dying within twenty-four hours, it was 4.8 per cent.

9. The mortality in 1448 cases injected on or after the fourth day was 27 per cent.

10. The most convincing argument, and, to the minds of the committee, an absolutely unanswerable one, in favor of serum-therapy is found in the results obtained in the 1256 laryngeal cases (membranous croup). In one-half of these, in a large proportion of which the symptoms were severe, recovery took place without operation. Among the 533 in which intubation was performed the mortality was 25.9 per cent., or less than half as great as has ever been reported by any other form of treatment.

11. The proportion of cases of broncho-pneumonia (5.9 per cent.) is very small, and in striking contrast to results published from hospital sources.

12. As against the two or three instances in which the serum is believed to have acted unfavorably upon the heart might be cited a large number in which there was a distinct improvement in the heart's action after the serum was injected.

13. There is very little, if any, evidence to show that nephritis was caused in any case by the injection of serum. The number of cases of genuine nephritis is remarkably small, the deaths from that source numbering but 15.

14. The effect of the serum on the nervous system is less marked than upon any other part of the body; paralytic sequelæ being recorded in 9.7 per cent. of the cases, the reports going to show that the protection offered by the serum is not great unless injections are made early.

SEPTICEMIA.

Definition.—Septicemia is a systemic infection due to a microbic invasion of the blood and tissues, with or without a detectable seat of infection. Sapremia is a toxemia of saprophytic origin, while *septic intoxication* is an affection due to the absorption of poisons from foci of suppuration. Mixed infections are common, hence it is clinically impossible to differentiate cases of pure sapremia, septic intoxication, and septicemia in most instances.

Pathology.—After death the body putrefies early. The macroscopic changes in the viscera are sometimes few and often wanting. The muscles present a brownish color-tint. The pia mater is generally congested, and, together with the nerve-centers, may be the seat of ecchymoses. The blood is dark ("tar-like"); its coagulability is diminished, and, microscopically, it shows an abundance of micrococci and bacilli. The spleen is somewhat softened and its lymphoid elements more distinct, and almost invariably ecchymoses are found in the serous membranes, especially the pericardium and peritoneum.

In protracted septicemia more marked alterations exist, and among them may be briefly enumerated the following: endocarditis (rarely ulcerative); gastro-intestinal catarrh (of the duodenum and rectum in particular) with punctiform extravasations; enlargement of the lymphatics and spleen, with softening of the latter; cloudy swelling of the liver (rarely the so-called emphysema of the organ due to putrefaction); edema and catarrhal inflammation of the uriniferous tubules; congestion, sometimes associated with edema of the lungs; and inflammation of the pleura, pericardium, and peritoneum, with ecchymoses and trivial effusions.

Microscopically, the internal organs show numerous small foci of inflammation, some of which may be the seat of "coågulation-necrosis." Bacteria are found in abundance in various situations, such as the exudations, the capillaries of the inflammatory foci, and renal glomeruli.

Etiology.—Bacteriology.—Septicemia is due to micrococci which Koch has shown to be considerably smaller than pus-cocci, though no one form of bacterium has been found constantly present to the exclusion of all others. Besser, as the result of careful experiments, concludes that septicemia is caused solely by streptococci, while Rosenbach and others have found both staphylococci and streptococci. Doubtless in many instances of human septicemia the clinical manifestations are due partly to bacterial poisoning and partly to septic intoxication with the poisons (ptomains) developed by the organisms, and the ptomains probably kill the patient before the bacteria can propagate themselves throughout the system. Laboratory experiments teach us that in the lower animals septicemia can be produced both by chemical poisons and by bacterial infection, and these two types are observed in human beings. With reference to the bacterial form Warren[1] states: "Whether this process is caused solely by the multiplication of bacteria, or is dependent in part upon the liberation of intensely powerful poisons, or is due to some ferment-like substance capable of reproducing itself like the poison of the serpent, as are diphtheria and tetanus, much more extensive studies upon the human subject will be necessary to enable us to say."

Modes of Infection and Introduction of the Poison into the System.—(1) **Wounds,** either surgical or the result of injury, with which we have nothing further to do in this work.

(2) **Through the uterus,** following labor, miscarriage, or abortion. Generally in these cases there are accompanying local changes, but in a few the poison appears to pass the unguarded portals of the organ, while the latter exhibits nothing abnormal.

(3) The cases in which the poison gains entrance into the body

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[1] *Surgical Pathology and Therapeutics*, p. 340.

without obvious wounds or raw surfaces are relatively more common. When the skin is quite natural, septic infection or intoxication cannot occur, but the slightest abrasion or cut, bed-sore, etc. may serve as a gate of admission. These slight lesions "may be almost completely healed by the time the severe symptoms of the disease are developed" (Strümpell).

(4) **Mucous membranes** often admit the virus, being less protective in nature than the skin. The numerous bacteria—benign and pathogenic—that are constantly present in the intestinal canal may also find in local lesions (as in typhoid fever, dysentery, etc.), or catarrhal inflammation even, points of lodgement and cause a systemic infection. To this category belongs that form of septic infection which follows gonorrhea. The so-called cases of "spontaneous septicemia" are also usually occasioned by absorption from the mucous surfaces.

Rheumatic or septic manifestations often follow attacks of ***tonsillitis***, and it is probable that the tonsils are more frequently points of entrance for the organism than has hitherto been supposed (Wade, Bannatyne).

(5) "**Sepsis Intestinalis.**"—This special form of poisoning is caused by canned meats, ice cream, sausages, and cheese. Vaughan, to whom we are indebted for the first description of "sepsis intestinalis," found in cheese a ptomain which he named *tyrotoxicon*, and which he regarded as the active agent in this group of poisoning cases. The symptoms are due, according to his statement, to poisoning by chemical substances, being instances of *sapremia;* but it may yet be found that the intestinal micro-organisms play a more or less prominent part in the process.

(6) Ogston[1] recognizes as one of the mildest forms of sapremia the sickness and nausea produced by a bad smell, which, he claims, is but a ptomain of putridity that may, under certain contingencies, produce serious symptoms. On the other hand, persons who are habitually exposed to bad odors (workers in sewers, in the dissecting-room, etc.) may acquire a considerable degree of immunity against poisoning of this sort. The fever in these cases corresponds in severity to the dose of the poison.

(7) **Septicemia** may be associated with or follow osteomyelitis.

Clinical History.—(1) **Symptoms of Sapremia.**—The fact that this form may occur without bacterial infection, either local or general, must be emphasized, but more frequently there will be either local infection or putrefactive changes, with the production of a grave general condition due to the absorption of the poisonous chemical products. In certain other acute infectious diseases (diphtheria, tetanus, typhoid fever, erysipelas, etc.) the general symptoms are similarly engendered. Perhaps the most typical examples of sapremia seen by the physician are those due to tyrotoxicon and to the unaccustomed inhalation of foul odors. At the beginning a chill may occur, but this is more generally wanting. In "*sepsis intestinalis*" marked *local symptoms* may initiate the attack, as nausea, vomiting, colicky pains, diarrhea, etc., and in all forms there is *fever*, the temperature often rising rapidly to 101° or 103° F. (38.3°–39.4° C.) and sometimes higher. *Prostration* and *anemia*, particularly the latter, may be prominent symptoms. ***Microscopic examination*** of the blood generally shows leukocytosis, and always a marked reduction in the number of red corpuscles.

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[1] Warren, *loc. cit.*, p. 342.

Sapremia following childbirth is a most typical sub-variety, and, apart from the special history, the symptoms are much the same as those detailed above. It is the form most amenable to treatment, the removal of the cause being followed by a rapid disappearance of all alarming symptoms.

(2) **Symptoms of True Septicemia.**—There is an *incubation*-period which is of variable duration, though usually averaging several days. The *onset* is more gradual than in the previous variety, although often marked by a *chill.* Accession of *fever* following surgical procedures, with headache, anorexia, prostration, sometimes vomiting and diarrhea, and especially dulness occasionally amounting to mild stupor, announce the affection: these symptoms should also excite suspicion in the absence of obvious causal factors. They become intensified, and now the attack may closely simulate certain other infectious diseases (typhoid fever, acute miliary tuberculosis, ulcerative endocarditis, etc.), the clinical picture as outlined presenting nothing characteristic. There are, however, more or less distinctive features, which will be considered seriatim.

(*a*) **The Fever.**—This is usually of the *continued* type, and tends to increase in degree, fatal cases often terminating in hyperpyrexia. At the beginning the temperature may rise quite rapidly, and in some cases it may even be subnormal. Deep morning remissions may be observed.

(*b*) **The Circulatory System.**—The pulse is frequent, and near the end becomes very weak. In subacute cases characteristic lesions (endocarditis in particular) may develop, but are difficult of recognition, since they do not, as a rule, give rise to audible murmurs or other physical signs. In other instances soft murmurs may be heard, but it is indeed hard to discriminate these from functional sounds. Moderate leukocytosis (principally of polymorphonuclear variety) is observed, and the presence of micrococci in the blood during life has been demonstrated.

(*c*) **Gastro-intestinal System.**—The spleen may become perceptibly enlarged, and gastro-enteritis is usually present, either in an acute form with vomiting and frequent serous discharges or more often merely with a diarrhea of moderate intensity (septic diarrhea).

(*d*) **Cutaneous Symptoms.**—Punctiform hemorrhages into the skin are of prime importance in the diagnosis. Occasionally more extensive ecchymoses appear, scarlatinal eruptions also showing themselves, but these are less characteristic. Among rare appearances herpes, roseola, edematous inflammations, and faint jaundice (affecting the skin and conjunctivæ) may be observed. The icterus is probably due to disintegration of the red blood-corpuscles in the liver.

(*e*) **Renal Symptoms.**—The lesions constitute the so-called "septic nephritis," the urine often containing a fair amount of albumin, epithelium, tube-casts, and red and white corpuscles.

Diagnosis.—(*a*) **Sapremia** can be distinguished by the history, the immediate appearance of the symptoms, their character, and by the prompt effect of the removal of the exciting cause. The diagnosis often requires a most careful search for the known etiologic factors, though even without the latter we can sometimes arrive at a correct conclusion by a careful process of exclusion.

(*b*) **True Septicemia.**—Here the existence of an incubation period, the contiued fever, mental apathy, faint jaundice, splenic enlargement, and the characteristics of septic nephritis, all combine to form a well-defined group of phenomena. A careful blood-examination should be made for micrococci, etc., and cultures should be undertaken in spontaneous septicemia and associated forms (*e. g.* septico-pyemia). The surgeon should look to the condition of the wound if one is present.

Course and Prognosis.—The course may be brief, virulent attacks sometimes terminating fatally within forty-eight hours, this being especially true of sapremia when the dose of the poison is large. The gravity of the case in the latter form is in direct proportion to the amount of virus that enters the system, the outlook being good when the cause is removable. On the other hand, in true septicemia this avails nothing, the progression tending steadily to the end. The mildest types may, in rare instances, terminate favorably, but the effects are not dependent upon the dose, and the minutest quantity may lead to specific results in their fullest intensity. It must not be forgotten that septicemia may pursue a chronic course in which the symptoms are milder, although the termination is very generally unfavorable.

Treatment.—Of first importance is the removal of the cause whenever practicable. The physician must support the patient's strength by a suitable dietary and by the judicious use of cardiac stimulants; the former should consist mainly of liquids (milk, egg-white, meat-juice, etc.), and the latter of alcoholics, together with strychnin and ammonia. Of medicines, internal antiseptics (mercuric chlorid, creasote, etc.) richly deserve a trial, though striking results have not been obtained from their employment. The fever calls for antipyretics. Cardiac depressants, as acetanilid and phenacetin, should not be resorted to, however, when great cardiac asthenia exists. To meet the renal conditions the free use of water, together with the least irritating of the diuretics, is to be advised and encouraged. A powerful agency in eliminating the microörganisms and their toxic products is found in normal salt solution, which may be administered by hypodermoclysis ("washing the blood"). Not less than from one to several pints of this fluid are to be used daily. In the inoperable cases, antistreptococcic serum should be employed. At first 1000 units of Marmorek's serum should be injected daily, later at longer intervals.

PYEMIA.

Definition.—A disease of the blood invariably associated with suppuration, and due to an absorption of pyogenic organisms.

Pathology.—The cadaver does not undergo putrefaction as early as in septicemia. Briefly considered, the pathologic lesions that fall within the physician's province arrange themselves under the following heads:

(1) **Thrombosis and Embolism.**—At first the veins leading to and from

the seat of the local changes from which pyemia arises contain thrombi which may soften into a puriform material. Thrombi are also found frequently in the lungs, a circulating embolus first finding lodgement in the pulmonary artery and its branches; they may be present in the liver, kidneys, spleen, cortical substance of the brain, and in other localities.

(2) **Abscesses.**—These so-called metastatic abscesses are set up by septic emboli or result from the thrombi (chiefly pulmonary and portal), and are found in the various internal organs, mainly, perhaps, in the lungs, liver, spleen, and kidneys. They are not large, but may coalesce and form cavities of the size of an apple. The kidneys are the chief organs of elimination in this disease, and hence it happens that numerous clumps of micrococci, producing miliary abscesses, are frequently seen in the regions of the Malpighian bodies. Infarction may be observed also. There are many other, though rarer, seats of abscesses, as the muscles, submucous and subcutaneous tissues, bones, the parotid gland, brain (cortical portion), ovaries, and testicles.

(3) **Lesions of the Skin and of Mucous and Serous Membranes.**—At the post-mortem examination hemorrhagic extravasations and pustules are often visible in the skin. The mucous membrane of the alimentary tract is rarely affected, differing in this point from septicemia, though occasionally ulcers may be noted, and most commonly in the stomach near the pyloric orifice (in puerperal cases) and in the large bowel. Probably they are always secondary to the submucous miliary abscesses. The serous membranes (pleura, pericardium, meninges of the brain, synovial membranes) may be the seat of purulent inflammation and of hemorrhagic extravasations.

(4) **Cardiac Lesions.**—Ulcerative endocarditis forms the chief morbid lesion. It begins in the form of small nodular vegetations upon the valves (most frequently the mitral), which disintegrate and leave ulcers behind (*vide* Ulcerative Endocarditis).

Etiology.—**Bacteriology.**—Experimental investigations have shown conclusively that the organisms usually responsible for this condition are the staphylococcus and the streptococcus. Whether the former or the latter be the agent of infection in the given case depends chiefly upon the condition of the tissues at the starting-point, especially with reference to the character of the local defensive processes; also, though to a lesser extent, the degree of virulence of the micrococci.

Other important pyogenic micro-organisms are the gonococcus, pneumococcus, bacillus pyocyaneus, bacterium coli communis, bacillus tetragenus, and many of the specific micro-organisms.

Paths of Infection of the Body.—(*a*) Almost always the entrance is by *the blood-vessels*, the special varieties of micrococci that cause pyemia, reaching the veins and producing thrombo-phlebitis. Less frequently they reach the arteries and produce thrombo-arteritis. From the former condition emboli may be disseminated throughout the system, while from the latter the emboli are arrested in the neighboring capillaries to which the tributaries of the vessel lead. Micrococci independently of emboli may be found wandering in the blood-stream.

(*b*) Another path of entrance is *the lymphatic system*, but here the cocci meet with greater forces opposing their attempts to spread than in the blood-vessels, and hence it is a much rarer mode of propagation.

(*c*) In *spontaneous pyemia*, in which there is no wound to act as an infection atrium, we must presuppose the existence of either a trivial lesion, as in "spontaneous septicemia," or an area of lessened resistance. The latter may be produced by inflammation, by a contusion, and in other ways, and all that seems necessary is a lowering of the tone of the general system (Warren). I am certain that ulcerative endocarditis is not frequently the starting-point, but is usually secondary to foci of inflammation elsewhere, as claimed by Osler. The appendix is often the primary or original focus in this category of cases, micrococci localizing themselves here in consequence of a preceding disturbance of the circulation or catarrhal inflammation. I recollect one case in which no original abscess was found at the post-mortem.

Predisposing Causes.—(*a*) *Epidemic Influence.*—It has been proved by abundant experience that certain seasons are characterized by epidemic outbreaks of the disease.

(*b*) Cases have sometimes been noticeably more frequent in the early months of the year (February and March) than in other seasons.

(*c*) *Age and Sex.*—Males are more frequently affected than females, and most cases occur about the middle period of life or at the time of greatest danger from traumatism.

Clinical History.—**Incubation.**—The disease sets in from a week to ten days after the reception of the wound or even earlier, and always develops secondarily to suppuration somewhere in the body.

A most conspicuous symptom, and usually the first, is the *chill:* it may, however, be preceded for a variable time by *fever* of a continued or intermittent type. The fever of pyemia is of the suppurative type. Profound *prostration* develops early; the *skin* presents an icteroid appearance; and gastro-intestinal symptoms may appear, but are not prominent. The signs of abscess of the lung, liver, and other organs may develop in some cases, while in others the whole clinical picture is colored by the ill-defined characters of ulcerative endocarditis.

(*a*) **The Chill.**—This may be mild, though oftener it is quite severe. It is repeated at somewhat irregular intervals, and rarely it may recur several times on the same day. Chills are most apt to occur during the daytime.

(*b*) **The Fever.**—A rapid rise of *temperature* accompanies the *chill.* The fever-curve is of the irregularly intermittent or profoundly remittent type, with intervening periods, showing slight or marked variations, and as decided deviations may occur within a short space of time, a two-hour record should be kept. The temperature rarely falls to the normal level; it may do so, however, and remain there for one or two days. To explain the peculiarities of the curve in this disease we need only recall the great variety of pathologic processes before noted. With the sharp fall of temperature *sweating* occurs, and leaves the patient more or less exhausted, though only temporarily so as a rule.

(*c*) **Respiratory System.**—Symptoms referable to the organs of respiration appear early. The pulmonary abscesses are usually latent, but may give rise to dyspnea, cough, and occasionally a purulent expectoration. *Pain* is present if they are superficially located, and under such circumstances the physical signs of cavity or of pleural effusion may be noted. The signs of *pneumonia* at one or both bases may also develop, the expectoration now becoming rusty.

(*d*) **Splenic and Hepatic Symptoms.**—The foci of suppuration in the liver are difficult of recognition unless they become large as the result of coalescence and are superficially located (see article Hepatic Abscess). Splenic infarction may also be safely diagnosed if there are pain and great tenderness (due to localized peritonitis) in the left hypochondrium, with progressive enlargement of the organ. In one case I detected distinctly crepitant sounds over the site of the spleen during life.

(*e*) **Cardio-vascular Symptoms.**—The *pulse* at first is accelerated, but moderately full and regular; later it becomes exceedingly rapid and feeble. Frequently cases in which ulcerative endocarditis develops are apparently of spontaneous origin. (*Vide* Endocarditis in the section on Diseases of the Heart.) Among the blood-appearances during life are *leukocytosis* and a rather marked reduction in the red corpuscles, with moderate poikilocytosis. Nucleated erythrocytes may be present. The blood-plaques are increased.

(*f*) **Cutaneous Symptoms.**—The most prominent is a mild yet decided grade of *jaundice*, that is hepatogenous (?) in nature. Sweating has already been alluded to as a symptom, both during and after the febrile paroxysms. The skin finally shrinks from emaciation. *Skin-eruptions* are common, particularly erythema, purpura, and pustules, and the general surface is often decidedly *hyperesthetic*.

(*g*) **Genito-urinary Symptoms.**—The urine is concentrated and urates are copiously deposited. There is *albuminuria*, which may be due to the pathologic changes or may be ascribable to the fever. The microscope discloses the presence of tube-casts, micrococci, pus- and (more rarely) blood-corpuscles. Albumose has been found in the urine.

(*h*) **Nervous Symptoms.**—The mind generally remains unclouded until an advanced stage is reached; then delirium sets in, and is followed by a terminal coma. Metastatic purulent meningitis, with its usual symptoms (hemiplegia, strabismus, ptosis, deafness, etc.), may appear.

(*i*) Symptoms may be presented by the **joints and bones.** Metastatic arthritis, usually suppurative, is a not unusual concomitant, and in some cases it is combined with similar involvement of the long bones. An acute osteomyelitis may be the only ascertainable source of the pyemia.

Septico-pyemia.—By this term is meant the combined presence of suppuration and a general intoxication (septicemia). The symptoms of pyemia (recurrent chills, copious sweats, metastatic abscesses, and early nervous symptoms) dominate the scene in the majority of the cases. Some of these instances pursue a comparatively mild, chronic course.

Differential Diagnosis.—The disease is often confounded with malarial intermittent fever, the distinctive features of which have been given under the differential diagnosis of the latter disease, but a diagnosis may always be made from the effect of quinin upon the fever. A few points of contrast, by means of which *septicemia* and *pyemia* may be differentiated, are tabulated below:

PYEMIA.	SEPTICEMIA.
Always associated with suppuration.	Suppuration may be absent, but there may be a sloughing wound.
Multiple chills.	A single chill.
Irregularly intermittent fever-curve.	Continued type of curve.
Profuse sweats accompanying febrile attacks.	Absent.
Rapid emaciation and profound prostration.	Less marked.
Nervous symptoms usually come on late.	Earlier.
Hyperesthesia.	Absent.
Slight jaundice.	Less marked (very faint).
Metastatic abscesses.	Absent.

Prognosis.—Pyemia may kill after an illness lasting but a few days. On the other hand, it may become more or less protracted, so that a chronic form has been distinguished. In this variety the symptoms are milder in character, and recovery may rarely ensue.

Treatment.—So far as the physician's province extends, the treatment is identical with that of septicemia. For the sweating the best agents are aromatic sulphuric acid and atropin; the latter may be given with agaricin (atropin, gr. $\frac{1}{120}$—0.0005; agaricin, gr. $\frac{1}{8}$ to $\frac{1}{4}$—0.008 to 0.016), at bedtime. Prompt surgical interference must be resorted to, not only with a view to asepsis of the primary wound, but also to evacuating the primary and all secondary foci of suppuration. The use of antistreptococcic serum has led to immediate improvement. The employment of normal saline solution by the method of hypodermoclysis aids in eliminating the peccant material.

ACUTE ARTICULAR RHEUMATISM.

(*Rheumatic Fever.*)

Definition.—An acute febrile disease, the exact nature of which is unknown, though it is probably infectious. The chief local manifestation is a multiple arthritis, and its chief complications are cardiac (endo- and pericarditis). Hueter first advanced the germ-theory to account for the disease, and, although the specific causal agent has not as yet been discovered, this view is the only one that offers a satisfactory explanation for the production of the lesions, the acute onset, the clinical course, and the complications of the disease. The frequent involvement of the joints in many diseases belonging to the acute infections may properly be regarded as supporting this theory. Strümpel points out the fact that in Leipsic, where articular rheumatism is one of the most frequent of acute diseases, it has been observed for years that at certain times there are only a few cases, while at others there is a striking increase in the number.

Pathology.—The disease does not show peculiar lesions, and, although the joints are the chief seats of invasion, still in many instances, and even in aggravated cases, the changes presented are slight or altogether wanting. Usually the synovial membranes of the affected joints are injected and swollen, and their surfaces may be more or less coated with fibrin. The effusion, which in a majority of instances has been found sterile, is mainly serous, but contains fibrin and often leukocytes, and occupies the joints. A similar exudate infiltrates the periarticular tissues. The tendinous sheaths may also be inflamed; the cartilages in protracted cases may become eroded; and rarely a purulent exudate may be seen.

Fatal cases, except when death is due to hyperpyrexia, usually show the changes peculiar to endocarditis, pericarditis, or myocarditis, and less frequently those of pneumonia or pleurisy. The fibrin-factors of the blood are augmented.

Etiology.—Bacteriology.—Guttmann, Collin, and Sahli have found the staphylococcus in the articular exudate of patients suffering from complicated or recurrent cases of acute articular rheumatism, and Sahli is inclined to include the disease in the group caused by this organism. Netter, however, has found the streptococcus, and Lang a peculiar bacillus. Singer has examined 92 cases bacteriologically and discovered staphylococci or streptococci in the majority of these cases, and also post-mortem. Pierre Achalme[1] has described an organism which he found in the blood (*Thiroloix Achalmii*) of cases of rheumatism with cerebral complications. It is a bacillus somewhat resembling that of anthrax; it is readily stained with anilin-dyes and by Gram's method, and is anaërobic. When inoculated into guinea-pigs, it causes inflammations of the serous membranes characteristic of rheumatism. Treboulet and Coyon[2] suggest that Achalme's bacillus is associated with only the severe forms of rheumatism, and they have found it in some other cases associated with a *diplococcus*, which latter was found in all other cases of rheumatism examined. The diplococcus was facultative, anaërobic and stained by Gram's method. While we are scarcely justified in ascribing the affection to any special micro-organism, "the bacillus of Achalme has apparently the strongest claims to be considered the cause."[3] A number of cases have been reported in which there is some evidence of direct contagion.

Predisposing Causes.—(1) An *infective lesion* (septic wound, attacks of angina, etc.) that has preceded for some time the appearance of the pain and articular manifestations may often be found, and this may be conceived to form a portal of entry for micro-organisms (Sacaze). The frequency with which an attack of tonsillitis precedes the development of acute articular rheumatism almost indicates a pathological relation between the two diseases (Cheadle, Wade, Gerhardt, Packard). (2) *Seasons.*—The months of February, March, and April furnish the largest percentage of cases, though the disease is also quite prevalent in the remaining cold months; on the other hand, the disease may sometimes be especially frequent in summer. Edlefsen and Newsholme have shown that the incidence of acute articular rheumatism is closely connected with the rainfall, the disease being most prevalent in dry seasons when the ground-water is low. (3) "Catching cold" was formerly classed among exciting causes, but while this affection often follows exposure to abrupt changes of temperature, it merely predisposes to the disease. (4) *Climate.*—The disease is most prevalent in temperate latitudes, being rare both in the cold and tropical zones. (5) *Occupation* is of primary importance, especially if it entail oft-repeated or prolonged exposure to the influence of wet and cold or to severe changes of temperature. Hence those who follow certain avocations are attacked with great relative frequency—*e. g.* coachmen, laborers, sailors, and servant-girls. (6) *Age.*—Primary attacks are most common from fifteen to thirty-five years of age. Out of 655 cases, 80 per cent. occurred between the twentieth and fortieth years (Whipham). Cases are also rather numerous between ten and fifteen years, and I have met with 4 under the former age. Sucklings rarely suffer. (7) *Sex.*—Acute articular rheumatism is somewhat more common in men than in women, and possibly owing to the fact that the former sex more often follows predisposing occupations. (8) *Hereditary influence* plays a causative rôle. (9) Conditions of ill health, particularly digestive and hepatic disturbances, seem to exert a slight yet decisive effect. (10) *Chronic endocarditis* renders its victims very prone to attacks of acute articular rheumatism, and some contend that the two diseases are etiologically one and the same. (11) *Choreic children* often develop rheumatism. Batton analyzed 115 cases; he found that within 3 years 11.3 per cent. of the children, without previous rheumatism, developed the disease, and after 5 years this total was increased to 20 per cent. (12) *Endemic and Epidemic Influence.*—In certain localities the disease is endemic, and epidemic incidence has been noted by McClymont, Newsholme, and others. House epidemics have also been observed.

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[1] *Ann. Pasteur Inst.*, Nov., 1897. [2] *Bull. de la Soc. méd. des Hôp.*, Dec. 24, 1897.

[3] D. Riesman, *Journ. Amer. Med. Assoc.*, Dec. 8, 1900.

An attack of acute articular rheumatism is not protective in character, but increases susceptibility. In this respect the disease resembles certain other infectious diseases (pneumonia, erysipelas, etc.).

Clinical History.—Of the *incubation* period nothing is known, though prodromata, both local and general, may be observed. There may be malaise, slight fever, angina, laryngitis, etc., and last from a few hours to a day or two. The *invasion* is usually abrupt, with fever and synovitis, affecting one or oftener several joints, and a chill or a series of chilly sensations may accompany or precede the rise of temperature. The involved joints are tender, often red and swollen, and exhibit the local signs of a rapidly developed inflammation. *Pain* is a most prominent symptom. The medium-sized or larger *joints* (knee, ankle, and wrist) are first involved, and especially those of the inferior extremities; next the shoulder-, elbow-, and hip-joints; and lastly the fingers, toes, and intervertebral articulations. Quite unusual articulations may become implicated (*vide infra*). One of the chief peculiarities of the disease is in the fact that the joints that are affected are not all the seat of anatomic changes simultaneously, but that the process migrates from one joint to another from day to day, and often crosses from one side of the body to the other. Sometimes this occurs at longer intervals. Hence the number of joints involved at one and the same time may be either few or many.

In cases of average severity the general features are subordinate to the local symptoms. The fever is usually moderate, the temperature not exceeding 103° F. (39.4° C.), and the temperature-curve is of the irregularly remittent type, corresponding in severity with the joint-symptoms. Defervescence is by *lysis.* The skin is bathed in a copious perspiration which is not dependent upon a previous fall of temperature. Nervous symptoms are rarely observed.

The *general course* of the disease exhibits wide variations, both as to duration and intensity of symptoms. It may not outlast several days, appearing with mild symptoms; on the other hand, cases sometimes persist for six to eight weeks. The latter instances, and even typical cases, are apt to show brief non-febrile periods, alternating with marked paroxysms, and similar cycles may be repeated. Cases in which the symptoms are distinct from the start may terminate in recovery within a shorter time than those in which the features are of mild character. As will be seen hereafter, the disease frequently manifests complications, especially cardiac.

Leading Symptoms and Complications in Detail.—(1) **Joints and Surrounding Structures.**—As I have stated, pain is much complained of, and is greatly augmented by motion and by pressure of any sort. It may be out of all proportion to the degree of the anatomic changes. The joints affected are generally swollen (most markedly in the knees), and the swelling is due partly to effusion into the joint and partly to inflammatory edema of the periarticular structures. The sheaths of the tendons, the bursæ, and often the adjacent muscles and fasciæ exhibit inflammatory changes; hence it is usual to see an extension of the swelling for a variable distance from the joint, the backs of the hands often showing this to a marked extent. The skin may present a pink or rose-colored blush over circumscribed areas or taking the form of streaks.

In even *mild cases* there are usually two, three, or more joints involved, though it often happens that one bears the brunt of the disease, little complaint being made of others less severely implicated. Hence it should be a golden rule to examine carefully all the joints at each visit. Involvement of a single articulation (*monarticular rheumatism*) does sometimes occur, but the diagnosis of these cases offers great difficulties. On the other hand, an existing polyarticular rheumatism may become centered in a single joint and there linger with great obstinacy.

In *severe cases* numerous joints may be invaded, with an involvement of the joints of the symphyses, of the jaw, of the ribs, and the sterno-clavicular articulations. Under these circumstances the patient assumes a dorsal decubitus, and seeks to relieve his excruciating pain by holding his limbs in a semiflexed position and absolutely motionless. If now an attempt be made to change his posture, he complains piteously of darting pains in the affected joints. The *fugacity* of rheumatic arthritis has already been alluded to.

The inflammation, however intense, may quickly subside in one joint, while at the same time an acute disturbance appears in another. Usually resolution is complete, no trace being left of former inflammation, though the disease may recur in the joints primarily involved. Suppurative arthritis may supervene, though rarely, and its occurrence points indisputably to mixed infection. This complication may lead to ankylosis—a sequela which does not belong to pure rheumatism.

(2) **The Cardio-vascular Symptoms.**—The *pulse* is quickened to 100 beats per minute or over, but is soft and full, and when cardiac or other complications arise it shows special characteristics which are described in appropriate sections of this work. In rare instances it is very rapid, feeble, and irregular, apart from the influence of the cardiac involve-

ment. The results of a careful blood-count show a high grade of *symptomatic anemia*, which may develop with marvellous suddenness. *Leukocytosis* is also present.

Great importance attaches to the *cardiac affections* that so frequently complicate this disease. They may arise in any case, even the mildest, or at any stage of the disease, and hence the conscientious physician cannot afford to neglect the matter of closely and regularly examining the heart. It must be recollected that no special symptoms announce the development of cardiac disease. At first we may note an increase in the febrile movement, more or less palpitation, sometimes dyspnea, and precordial pains, which often do not amount to more than a sense of soreness. There may also be attacks of angina pectoris of apparently purely nervous origin (Strümpell).

(*a*) The most frequent cardiac complication is *acute endocarditis*, which is present in 25 to 30 per cent. of the cases. We are, however, sadly in need of reliable statistics upon this point. It usually takes the form of simple (verrucose) endocarditis, and affects most frequently the mitral valves. But, though usually indicated by an apical systolic murmur, it is hard indeed to eliminate the functional murmurs that may also develop in the course of this disease. Unless combined with the symptoms detailed above, the presence of a blowing systolic murmur does not afford trustworthy evidence of the existence of acute endocarditis. I have witnessed two instances in which endocarditis preceded the arthritic manifestations. Church and Cheadle[1] state that "in a large majority of cases, if no endocardial murmur is present during the first ten days of an attack, the endocardium escapes." While it rarely endangers life and may leave no trace, in the majority of instances the acute endocarditis does not undergo complete resolution, but leads to sclerotic changes and terminates in incurable chronic valvular disease.

(*b*) Next in the order of frequency is *pericarditis*, which may or may not be combined with the former. In many cases the effusion consists of organizable lymph (often large in amount); less commonly it is serofibrinous and rarely becomes purulent or blood-stained. It is distinguished by its pathognomonic friction-sound, though also by other characteristic signs (*vide* Pericarditis).

(*c*) *Myocarditis* is often present to a slight extent in rheumatic endocarditis and pericarditis when these occur independently of each other, but more often and to a more marked degree when endo-pericarditis exists. Hence it is far less common than either endocarditis or pericarditis. The changes and symptoms occasioned will be discussed under Myocarditis. In this connection it should be pointed out that the condition weakens the cardiac walls and leads to dilatation of the ventricles (usually the left).

If we consider rheumatism an infectious malady, we can readily understand why the local manifestations should appear not only at the different articulations, but also in the cardiac structures, and, as we shall see, in other viscera.

(3) **The Skin.**—Rheumatism produces copious *perspiration*. The sweat emits a sour odor and gives at first an acid reaction, though later it may be neutral, and rarely alkaline. The temperature-curve in

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[1] *Allbutt's System of Medicine*, vol. iv., p. 15.

most cases is not materially influenced by the sweats. Occasionally the drops in temperature and the free sweats are concurrent, but the latter symptom is apt to persist despite the oscillations in the temperature. *Sudamina* appear, often in extensive crops. Among other *skin eruptions* less frequently observed are forms of erythema (especially *E. nodosum*) and urticaria, which latter may be associated with purpura (*urticaria hæmorrhagica*). The association of the latter condition with polyarthritis is known as *peliosis rheumatica*, though, according to some writers, this is not rheumatic in nature. Cutaneous ecchymoses, and even extensive hemorrhages into the skin and from the mucous membranes—a general hemorrhagic diathesis—may also be encountered.

Subcutaneous Rheumatic Nodules.—In 1881, Barlow and Warner called attention to the fact that during and after acute articular rheumatism, particularly in children and young adults, small subcutaneous nodosities attached to the tendons and fasciæ may in exceptional instances be observed. These small nodules are rather firm, movable, and usually painless. The skin over them is simply elevated, with no traces of inflammatory action. They are most frequently found at certain points of election (fingers, wrists, edge of the patella, malleoli, and over the back of the elbow), though also seen less frequently elsewhere; they may disappear, and after a brief interval reappear. On *microscopic examination* it is seen that round and spindle-shaped cells enter into their composition. Riess believes them to be of embolic origin. I met with one case of the sort which occurred in a male aged forty-two years, in which acute articular rheumatism was also complicated with endo-pericarditis and pneumonia. Most of the nodosities were of the size of a bitter almond. The case proved fatal. Cheadle considers that the eruption of large nodules signifies persistent and uncontrollable cardiac disease.

(4) **The Fever.**—The fact that the fever fluctuates materially in this affection has already been noted. It remains to be pointed out that if suppuration occur as a complication, the fever may be of the hectic variety; also that rarely hyperpyrexia is suddenly developed, and with it marked cerebral symptoms (restlessness, delirium, and sometimes convulsions, finally merging into stupor) are, as a rule, though not necessarily, associated. This serious condition commonly develops about the beginning of the second week. In my case cited above, it began on the sixth day. Delirium usually comes on either shortly before or after the acute development of the *hyperpyrexia*. The pulse becomes excessively rapid and feeble and physical prostration extreme. The temperature may rise rapidly with slight interruptions until it touches 108° or 109° F. (42.7° C.), and as the fever reaches its maximum death usually ensues. The temperature may continue to rise after death. The cause of "hyperpyretic rheumatism" is not definitely known. It has been claimed that the intemperate are most apt to be attacked, but this belief is not corroborated by many clinicians. In a case of my own, however, of acute articular rheumatism, in which pericarditis with hyperpyrexia occurred, the patient was an "alcoholic." It is reasonably certain that the symptoms are due to an intense infection, with concentration of the poison upon the nerve-, and especially upon the thermal, centers.

(5) **The Muscular and Nervous Symptoms.**—It has been stated that

the adjacent muscles and fasciæ may exhibit inflammatory changes. They may also show more or less swelling, and are often very tender to the touch, while in long-continued cases muscular atrophy ensues. The cause of this change is not clear, but the most likely view is that it results not so much from disuse of the muscles (the old theory), as from some trophic disturbance due either to the arthritis, or peripheral neuritis, or, to some extent at least, from extension of the rheumatic inflammation from the nearest articulation. Other theories have been advanced, but are scarcely tenable.

Mention has been made of the grave *nervous symptoms* that are attendant upon hyperpyrexia, but, independently of the latter condition, nervous phenomena may be present. There may be restlessness and sleeplessness (due to pain), but active delirium is exceptional in uncomplicated cases, and it is usually associated with a temperature of 104° F. (40° C.) or higher. In adynamic types, which are rare, low muttering delirium merging into stupor, and even coma, may be observed. Active mental symptoms are sometimes due to cerebral embolism secondary to acute endocarditis. When pericarditis is a complication, wild delirium, with or without hyperpyrexia, or the low muttering variety with stupor, is not unusual. The drunkard may develop *delirium tremens*. *Coma*, leading quickly to a fatal result, may develop without other previous or associated nervous symptoms, and DaCosta has reported cases in which a fatal coma was probably due to uremia. Rarely coma develops during the period of convalescence. *Convulsions* may be noted, generally preceding the coma, though rarely as an independent symptom. *Melancholia* may arise in the course of the disease, but more frequently at its close. *Meningitis* must be numbered among the rarest of complications.

Chorea is a not infrequent sequel of this disease in children, and more rarely is associated with it. Of 554 cases analyzed by Osler, in only 88 were chorea and rheumatism associated. These instances may or may not be accompanied by acute endocarditis.

(6) **Pulmonary Symptoms.**—*Pleurisy* occurs, and is generally excited by an extension of inflammation from the pericardium, and from the pleura the inflammatory process may be propagated through the diaphragm to the peritoneum. *Bronchitis* is sometimes present, but is rarely a part of the rheumatic morbid process; it is secondary, and in most instances is occasioned by the co-operation of the factors that are at work in every disease in which enforced recumbency and great prostration coexist. In like manner, *broncho-pneumonia* may be produced. *Lobar pneumonia* rarely occurs, and is confined to aggravated cases, but pulmonary congestion is occasionally seen, and may prove fatal. Pulmonary complications are also prone to develop secondarily to pericarditis, and especially to endo-pericarditis.

(7) **The Renal Symptoms.**—The *urine* is diminished in amount, is high-colored, and of high acidity and density. The standing specimen deposits urates. As in other infectious diseases, there is commonly present a slight febrile albuminuria, but acute nephritis is extremely rare. The chlorids are sometimes diminished, but rarely absent.

(8) **The spleen** is slightly enlarged in some cases. The *saliva* has sometimes an acid reaction, and, according to certain writers, the sulfocyanids are in excess.

Clinical Peculiarities of Acute Articular Rheumatism in Children.—The arthritic symptoms in children are in abeyance while endocarditis and pericarditis are predominant, and these cardiac conditions may appear before the joint-lesions are observed, but it is quite probable that endocarditis follows the joint-lesions twice as frequently in children as in adults. Parsons lays stress upon reduplication of the cardiac second sound, audible at the apex only, as an indication of the development of endocarditis. This sign is to be distinguished from reduplication heard at the base, sometimes as the result of Bright's disease and sometimes as the consequence of pulmonary obstruction. Acid sweats are slight in children. Rheumatic tonsillitis is quite common, and may precede, accompany, or follow attacks of rheumatism in children. Erythema is a frequent concomitant, and is often mistaken for scarlatina. The febrile movement lasts usually but a few days.

Diagnosis.—The acute development as a primary affection of polyarthritis with fever is a symptom-complex on which an assured diagnosis can be usually based.

Differential Diagnosis.—*Pyemia* must be carefully separated. In pyemia, however, the general condition is graver, fever precedes the local manifestations, and the fever-curve is irregularly intermitting. Rigors also occur in pyemia at varying intervals, accompanied by a steep elevation of temperature—symptoms that are absent in rheumatism. Again, suppurative processes in the various viscera and skin and slight jaundice appear in pyemia. Rheumatic symptoms fluctuate greatly, while the pyemic do not.

The multiple swelling of the joints which develops after childbirth is to be regarded as *septic* in nature. In these cases arthritis leads rapidly to suppuration, with more or less destruction of the joints. *Gout* will be distinguished from rheumatism in connection with the consideration of the former disease (*vide* p. 402).

Monarticular rheumatism is with difficulty differentiated from a group of affections which simulate it closely. (1) The so-called *gonorrheal rheumatism* often affects a single joint, especially the knee; but in this disease there is usually a definite history of recent infection, and the local features (pain, swelling, etc.), unlike true rheumatism, are far more pronounced than the general. The course of gonorrheal arthritis is longer in duration, and is generally connected only with a single joint from the start; while acute articular rheumatism almost always begins as a polyarthritis, with subsequent fixation in one articulation. Cardiac complications are rare in the former disease.

(2) *Acute osteomyelitis* is generally single, and is sometimes mistaken for rheumatism, from which it differs, however, in the localization of the lesions in a single joint from the start, the greater prominence of the local symptoms, and in the implication of the epiphyses and the shaft of the affected bone rather than the joint, and in the graver general symptoms from the time of onset.

(3) There is a liability to mistake the *acute arthritis* of infants for rheumatism. This attacks by preference the hip or knee, and is a purulent inflammation due to pyemia (Townsend), hence having no relation to the disease under consideration.

(4) *Scrofulous arthritis*, particularly in children, has been confounded

with rheumatic monarthritis. The former is less indurating, the swelling presented is less symmetric, and the course is far less acute than that of the latter.

(5) In the course of the *hemorrhagic diseases*, scurvy, purpura, and hemophilia, effusion into the joints, either hemorrhagic or serous in nature, occurs with great frequency and is associated with rheumatic pains. The differential diagnosis is to be formed from the tendency to hemorrhage, and in scurvy by the lesions of the gums. The absence of fever is usually decisive: unfortunately, it is frequently present in these joint-affections.

(6) *Glanders*, at the onset, may be mistaken for rheumatism.

Prognosis.—Recovery is the general rule. As in other infectious diseases, so in rheumatism, the chief immediate danger springs from the great intensity of the type of infection, as manifested in hyperpyrexia with grave nervous symptoms, the development of the general hemorrhagic diathesis, etc.—happily rare occurrences in this disease. Certain complications, such as pericarditis, endo-pericarditis, pneumonia, etc., may render rheumatism grave or even hopeless, and rarely the endocarditis that complicates the disease is of the ulcerative variety and leads to fatal pyemia. Pulmonary embolism may occur and cause death.

The influence of personal factors may impede recovery, such as intemperate habits, great obesity, the existence of previous organic disease of the heart or Bright's disease, etc.

Treatment.—(1) **Sanitary Environment, Diet, and Stimulants.**—The sick-room should be well ventilated, and its temperature maintained at 65° to 70° F. (18.3°–21.1° C.), but draughts should be avoided. The patient should be lightly dressed in flannels and covered with a sheet of the same material. The *diet* should be liquid and nourishing, milk being the best food-article. Farinaceous matter, skimmed milk, milk and Seltzer water, buttermilk, egg-white, may all be employed if whole milk cannot be taken in adequate amount. I begin the use of easily-digested forms of animal food soon after defervescence has occurred, but have immediate recourse to the earlier, liquid or soft diet upon the return of pain and fever. An ordinary dietary is to be gradually resumed. *Stimulants* may be employed if indications for their use are present, and the customary mode of administration may be followed.

(2) **Internal Therapeutics.**—There has been of late a surprising unanimity among clinicians in commending the use of the salicylates in the treatment of this disease—more so than at any previous time since their introduction. They are employed in most of the larger hospitals both in Europe and America. Differences, however, relating to the mode of administration and the particular salt to be selected still exist. Wood[1] favors ammonium salicylate, for the reasons that it is freely soluble, is rapidly absorbed, and when given in sufficient amount quickly produces the symptoms that mark salicylic action, while, in addition, it is less depressing than the other salts of salicylic acid. It is best given in milk and is usually well borne. My experience with this salt in acute articular rheumatism, though as yet somewhat limited, has been satisfactory. Until the present time sodium salicylate has met with more general favor than any other single salt of salicylic acid.

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[1] *University Medical Magazine*, Jan., 1895.

The pure acid is also used, though not to any great extent at the present day. As regards the mode of administration, the total daily amount taken is of higher importance than the size and frequency of the dose. The amount given in twenty-four hours should not exceed 2 drams (8.0), while often 1½ drams (6.0) of the sodium or ammonium salicylate is sufficient. My method is to give gr. x (0.648) every two hours during the first day, or until the pain and other local features have largely disappeared; then the remedy is given at longer intervals, but not omitted entirely. In this manner fresh exacerbations are most probably averted. If the latter occur, however, larger doses must be instituted, so as to cut them short. Some recommend that the medicine be stopped as soon as the pain has been controlled. Lassere recommends methyl salicylate, and in 40 cases found it effective in relieving the pain. Some prefer salol to either the pure acid or the salicylates: in my experience, however, the use of this drug has not been followed by good results in the severe acute forms of the disease. Doubtless the reason for this lies in the fact that salicylic acid can neither be introduced into the system in sufficient amount nor rapidly enough in the form of salol.

Kinnicut has recommended the employment of the oil of wintergreen, a salicylic compound which does not generally produce the unpleasant toxic symptoms so apt to be excited by the salicylates or salicylic acid. The dose is ♏x–xx (0.60–1.25), given in capsules or in milk, to be repeated every two hours. Salicin (gr. x—0.648, every hour, increased to gr. xv—0.972) is sometimes efficacious and invariably agrees. Salophen, in daily doses of 1 dram (gr. xv—0.972, every four hours), may be substituted for sodium salicylate if the latter produces gastric disturbances after a few days' treatment; it is almost specific in its effects. Salophen passes through the stomach unchanged, to split into salicylic acid and acetylparalidophenol in the intestines. Sodium salicylate enemata (ʒj—4.0—of the salicylate and ♏x—0.60—of the tincture of opium in each injection) may be of advantage in certain cases. The remedy is absorbed from the rectal mucosa, though more slowly than from the stomach.

The treatment with the salicylates or salicylic acid mitigates the fever, relieves the pain, and shortens the stay in bed by a few days, but does not curtail convalescence. The statistics of Williams go to show that the salicylate treatment also tends to protect against the development of cardiac complications, though it does not seem to influence the course of the complications once they are established. In my experience the alkaline treatment operates potently to obviate the occurrence of the heart-complications and shortens the period of convalescence, but exerts slight, if any, influence upon the fever-curve and pain. These facts led me long since to use the specific and alkaline treatment in combination, giving, in addition to salicylates or other salicylic compounds, as above indicated, an alkaline remedy, such as sodium bicarbonate, potassium citrate, etc., in sufficient doses to render, and then maintain, the urine of slightly alkaline reaction.

There are a few other remedies that should be referred to, and, although more or less serviceable, they are without specific influence. The foremost among these is antipyrin, but I have come to believe that safer and equally efficacious remedies have replaced this drug. Potassium iodid and the preparations of colchicum belong to this category, and should be tried. Their effects are most beneficial in cases that drag on after the acute stage is over. Lactophenin is used by Roth as a most useful remedy in acute rheumatism. Good results have been reported from the use of aspirin (gr. vij–xv—0.466–0.972, thrice daily) in both acute and chronic rheumatism.

(3) **Local Measures.**—These occupy a subordinate place in the management of acute articular rheumatism. Their number is legion, but only a few of the more valuable can be adduced here. In mild cases the affected joints should be wrapped in cotton batting or in flannel. If the pain is severe despite the use of the salicylates internally, fomentations as hot as can be borne or hot cloths lightly wrung out of Fuller's lotion (sodium carbonate, ʒvj—24.0; laudanum, ℥j—30.0; glycerin, ℥ij—60.0; and water, ℥ix—270.0) are beneficial. As salicylic acid is absorbed through the skin, it has been combined with other agents for local use in the following formula:

℞. Acid. salicyl.,
Lanolini, *āā.* ʒiij (11.65);
Ol. terebinthinæ, ʒiij (11.25);
Adipis, ʒiij (11.65).
M. et ft. ung.
Sig. Rub over the affected joints and follow by wrapping in cotton.

Methyl salicylate, by local application, is of service. It is put on the skin over the affected joints drop by drop, and the joint then enveloped in gutta-percha tissue and a flannel bandage applied to it. Cold compresses and the ice-bag to the joints have been strongly advised, particularly by German authors. The affected joints should be kept at perfect rest, and this is best accomplished by padded splints and a roller bandage. Blisters near to the joints involved and the light application of the Paquelin thermo-cautery are sometimes serviceable. Taylor[1] has successfully employed currents of hot air, applied by means of an instrument (electro-thermogen).

The treatment of the *complications* will be considered under their appropriate headings. Should, however, hyperpyrexia occur during the progress of the affection, it is to be relieved by cold affusions, since large doses of internal antipyretics are of themselves dangerous. It may also be stated that the cardiac complications—endocarditis, pericarditis, and endo-pericarditis—rarely require special remedies. If marked cardiac asthenia appears, as indicated by the feeble first sound, the salicylates may be replaced by salicin, which is less depressing in its effect upon the heart. Cardiac stimulants may be required. A copious pericardial effusion calls for paracentesis (*vide* Sero-fibrinous Pericarditis).

During convalescence the patient should not be allowed to get out of bed too early. My own rule has been to keep him in bed for a week after the temperature has returned to the normal and after the pain has disappeared, except it be during the hot season. These precautions are taken to avoid the occurrence of relapses. After the patient goes into the open air he should be told to avoid cold, and wet in particular.

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[1] *Lancet*, Nov. 26, 1898.

During this period iron is to be employed until the blood-examination fails to show anything abnormal. For the stiffness and swelling that sometimes persist, or disappear very slowly after the acute attack, massage and the application of hot water or warm baths seem to yield the best results.

SUBACUTE ARTICULAR RHEUMATISM.

THIS is, as a rule, a sequela of acute rheumatism, and may occur, though rarely, in persons who have not had a previous acute attack. Both the local and general features are of a mild type, but the course is apt to be prolonged into two, three, or more months. Usually the local symptoms are confined to one or two of the larger joints, with little swelling or redness, and the pain is slight except on movement. The temperature rarely exceeds 101° F. (38.3° C.), and at times may be practically normal. Though the course is prolonged, the joints usually return to their normal state; occasionally, however, the disease becomes chronic. As in the acute form, so in the subacute, anemia becomes well marked and cardiac complications are not uncommon, particularly when the disease occurs in children.

The **treatment** embraces, in addition to the usual antirheumatics, the use of iron, quinin, cod-liver oil, and, when practicable, a change to a warm climate. The affected joints demand hot applications and massage.

GONORRHEAL ARTHRITIS.

Definition.—A septic synovitis caused by the gonococcus, and hence having no connection with true rheumatism. It usually manifests itself toward the close of an attack of gonorrhea, but it may develop during the active stage of the disease or at any period during the course of gleet.

Pathology.—The signs of ordinary synovitis are generally found in the affected joints, though, not rarely, the inflammatory process is periarticular (*gonorrhœal tenosynovitis*). In these cases the inflammation may travel along the sheaths of the tendons for a considerable distance. Synovial effusion may occur, and rarely may be purulent, this being most frequent in gonorrheal inflammations affecting the wrist and hand. Gonococci have been found in the effusion, though this is not generally the case, and it is now thought by many writers (Finger, Councilman, and others) that the gonococcus may be the only infective agent concerned in the morbid process. Others contend that the metastatic inflammation of the joints is due to the presence of pyogenic cocci, since they have been found to be frequent companions of the gonococcus. In this and other forms of secondary inflammation it must not be forgotten, however, that gonorrheal arthritis may be due in great part to the absorption of ptomains from the urethra. The disease is present in

2 per cent. of all cases of gonorrhœa in males and rarely occurs in females (Gaither); it may follow any urethral discharge or may be associated with menstruation or leukorrhea.

Clinical Symptoms.—Two leading varieties, acute and chronic, are encountered. (1) *Acute Gonorrheal Arthritis.*—This may be very mild, amounting merely to slight fugitive pains and some stiffness of one or more joints, without noticeable swelling or redness. The typical, acute form, however, presents the symptoms of a severe fibrinous or sero-fibrinous inflammation of a single joint, developing quickly. The pain is often violent; there is swelling of the joint with extension along the course of the tendons, and the condition is obstinate. Unless pus be present (a rare event) the constitutional features do not correspond in severity with the local, there being little fever and slight impairment of the general health. There are many instances in which the complaint begins as a polyarthritis, with subsequent concentration upon one or two of the larger articulations, especially the knees or ankles. Fibrinous ankylosis usually remains as the resulting condition.

Acute endocarditis may be of gonorrheal origin, but undoubted instances are not common. In the inflammatory products of this condition Hering has found the gonococci, as has also Councilman, in the heart-muscles (gonorrheal myocarditis). Rarely, gonorrheal endocarditis assumes the ulcerative or malignant form. As the result of invasion of the blood by the gonococci, *suppurative arthritis* may develop and form a part of *gonorrheal septicemia.* I observed one case in which *pleurisy* was associated, and among the widespread complications *embolic*, *septic pneumonia*, and *iritis* deserve special mention.

(2) *Chronic Gonorrheal Arthritis.*—This occurs (*a*) as a serous effusion (*hydrarthrosis*), and (*b*) as a chronic inflammation of the articular and periarticular structures (synovial membranes, bursæ, periosteum, and tendons with their sheaths). The former is usually monarticular, settling with especial frequency in the knees, and may be wholly painless. The latter is more or less painful—causes dense swelling of the joint, and frequently of the structures for some little distance above and below the latter. Both forms lead to great restriction of motion.

The **diagnosis** cannot be determined apart from the history of urethral infection, or the detection of the gonococci in the blood or the joint-effusion. The acute form is distinguished from *acute articular rheumatism* by the more intense pain, the extent to which the periarticular tissues are involved, and the negative character of the general symptoms. The chronic variety must be discriminated from *chronic synovitis* due to other causes, and this often proves a difficult task.

Treatment.—I have never seen the slightest benefit from internal medication in gonorrheal arthritis, except possibly from the use of mercury. Maragliano advises quinin hypodermically and internally. J. C. Wilson[1] has obtained excellent results from massive doses (♏x–lx—0.60–3.75 t. d.) of the syrup of iodid of iron.

Local measures, however, are of paramount importance. Absolute rest to the part is indicated, and the limb should be placed upon a splint; then after making an appropriate anodyne application (ungt. ichthyol. or ungt. belladonnæ), it should be bandaged as firmly as possible. In

[1] Jacobi's *Feschrift*, 1900.

other instances complete immobilization in plaster-of-Paris dressing gives good results. Before doing this in acute cases the patient should be anesthetized, and after the procedure, if pain be great, a hypodermic injection of morphin may be given. In *chronic forms* the aim should be to remove the effusion (if present) and the swelling, and to restore the natural motility to the utmost. For the latter two indications massage and passive movements are best. Swelling may also be diminished by the use of the thermo-cautery at intervals, and blisters are highly serviceable in causing a disappearance of the effusion.

VARIOLA.

(*Small-pox.*)

Definition.—Variola is an acute contagious disease, characterized by its sudden onset and severe period of invasion, followed by a remission of the fever and an eruption of papules, which pass through the stages of vesicle, pustule, and scab. The stage of pustulation is accompanied by secondary fever. Variola runs a variable course, but on the whole has become milder far in character during recent years.

Historic Note.—Small-pox has existed from the earliest antiquity in India, Africa, China, and other Eastern countries. During the thirteenth century (1241) it entered England, in the early part of the fourteenth Ireland, and in the latter part of the fifteenth Germany. In 1507 it was imported to America, and first appeared in the West Indies; a little later (1520) the Spanish troops conveyed the disease to Mexico, where it destroyed not less than three and a half millions of people in its pestilential march. It was brought to the United States from Europe in 1649, and gained its first foothold in Boston, whence it progressed at intervals in a westerly direction to the western coast-line. At the present day there is a very limited opportunity to observe the affection except in its modified form (*varioloid*). About five years ago, during the Spanish-Cuban war, the disease was transferred from Cuba to the Southern States, afterward spreading to many of the Northern and Western States. In numerous localities the cases multiplied to such an extent as to approximate at least an epidemic prevalence of the disease, although of unprecedented mildness.

Pathology.—The eruption of small-pox consists in an inflammatory cellular infiltration of the *rete mucosum* and has four successive stages—(1) *Papular*, (2) *Vesicular*, (3) *Pustular*, and (4) *Scab*.

(1) *The Papule.*—At first there is a hyperemia of the papillæ of the skin appearing as small red spots. These soon become round, discrete patches that may be rolled like shot under the skin, and then become elevated, owing to the increase in the cells in the *rete mucosum*.

(2) The *vesicle* appears at the apex of the papule, and results from a circumscribed elevation of the superficial layer of the epidermis in consequence of the mechanical pressure exerted by the fluid exudate, which is excited by active peripheral inflammation. The vesicle is not unicellular, but is loculated (by fibrinous reticuli), and contains serum, leukocytes, fibrin-filaments, etc. If a section of a vesicle in the very

early stage be made through the deeper layers of the *rete mucosum*, an area of coagulation-necrosis is observed, which is due to the presence of micrococci (Weigert). The vesicle shows central umbilication, which corresponds with the necrotic area.

(3) The *pustule* is formed by the filling of the reticuli with leukocytes. Cellular infiltration and swelling of the true skin beneath the pustule occur, as a rule, as the result of diapedesis. Moreover, suppuration may involve the *cutis vera*, and as a consequence scarring results. In hemorrhagic small-pox the reticuli are occupied by an abundance of red corpuscles which have passed in from the adjacent blood-vessels, and may infiltrate the upper as well as the deeper layers of the epidermis surrounding the vesicles or pustules. The pustules may dry up, but commonly rupture, and in either case the result is (4) *scabbing*.

The eruption has run an atypical and even abortive course in the cases occurring in recent years. An early maturation of the papules has been observed; in many cases they became solid, conical elevations with a small vesicle at the summit. W. M. Welch[1] states that the lesions seemed to involve only the outer epidermis and the layer of cells immediately over the papillæ.

The mucosa of the mouth, pharynx, and, rarely, the esophagus and the rectum may be the seat of a variolous eruption, and the plaques of Peyer may be somewhat swollen. The eruption also appears in the larynx, the trachea, and bronchi, where ulcers rather than true pustules are seen, and the conjunctiva and nasal mucosa frequently show the specific lesions.

Hemorrhagic small-pox presents extravasations occurring in the serous and mucous membranes, the connective tissue, the parenchyma of the various viscera, and also, though much less frequently, in the nerve-sheaths, bone-marrow, blood-vessel walls, and the muscles. In this form the spleen is firm (Ponfick, Osler), and the liver is sometimes enlarged and the subject of fatty degeneration. Hemorrhagic infarction of the lung occurred in 5 out of 7 cases examined by Osler.

Secondary Lesions.—The catarrhal inflammation of the larynx may extend in depth till it touches the perichondrium of the cartilages (perichondritis), and a croupous exudate in the larynx may often coexist with edema. Lesions are present in the lungs, some of them frequently (general bronchitis, bronchopneumonia), and others rarely (hypostatic congestion, lobar pneumonia), and *pleuritis* may be observed. Cloudy swelling, diffuse inflammation, and sometimes fatty degeneration of the *liver* have been noted; the *spleen* is enlarged and pulpy as a rule. The *heart* may show myocardial alterations—chiefly parenchymatous and fatty—and rarely endocarditis and pericarditis occur. The *kidneys* show cloudy swelling and occasionally nephritis develops. Weigert found that at the commencement of the stage of suppuration the microscope revealed "small-pox cylindric masses" in the various viscera. The cylindric masses are in reality capillaries filled with micrococci, which are derived from the eruption. These small areas of coagulation-necrosis may be the seat of miliary abscess if septic material be also absorbed from the pustules. The blood presents no special microscopic appearances.

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[1] *Philada. Med. Journ.*, Nov. 18, 1899

Etiology.—**Bacteriology.**—J. Christian Bay[1] has isolated from small-pox lymph and vaccine points an organism (*dispora variolæ*) which he believes to be the *causa morbi* of small-pox and vaccinia. The long diameter of the bacterium measures 0.6μ–1μ, and the short diameter from 0.2μ to 0.3μ. The organisms contained two spores, one at either end, and when reared on artificial media developed in colonies, and were readily stained with anilin blue or violet. Copeman and Klein, working independently, have described a bacillus found in a human pustule and the lymph from cow-pox, which seems to give greater promise of being the special organism of the disease. "This bacillus is extremely small, 0.4μ to 0.8μ in length, and 0.2μ to 0.3μ in width, and is grown and stained only with great difficulty" (L. Leroy). Haushalter and Étienne[2] consider the hemorrhagic symptoms in small-pox due to secondary infection with the streptococcus, since they have found this organism in the blood of those dead of hemorrhagic variola. Widal and Sabrazes have also noted the streptococcus in autopsies upon small-pox cases.

Predisposing Causes.—The *receptivity* for variola is wellnigh universal, and among the few who have enjoyed immunity were three distinguished physicians—Diemerbroeck Boerhaave, and Morgagni. It may be said that one attack confers permanent immunity, but exceptionally a second or even a third may occur. *Vaccination*, also, if successful, affords future protection against variola, but to this rule exceptions are not infrequent.

Age.—All periods of life are liable to the disease, but the very young are affected in a relatively larger proportion than older persons. During the entire puerperal stage there is an increased liability to the disease. It rarely affects the fetus in utero, and most babes even, who are exposed to the virus at the time of birth, will not take the disease if immediately and successfully vaccinated.

Sex is without influence.

Race.—Among uncivilized peoples variola spreads with frightful rapidity, the negro and other very dark races being affected in larger numbers and more severely than whites. A dread of the infection predisposes to its occurrence.

The Contagion; where Found; Modes of Conveyance and of Infection.—One case of variola is *primâ facie* evidence of the existence of another, and that the poison from the latter was somehow transferred to the former. The specific poison exists in the blood and in the secretions and excretions (most probably), but mainly in the pustules and dry scabs and in exhalations from the lungs and skin. The contagion is conveyed principally from the sick to the healthy by the dust-like particles of the dried scabs.

Modes of Infection.—(*a*) *Inoculation* with either the blood or the contents of the eruption or the dissolved dry scabs is followed by variola. (*b*) Contact with, or proximity to, a patient suffering from small-pox is very apt to convey the poison, with resulting variola in the person thus exposed. To what distance the contagion can be conveyed through the air is not known, but it is probably considerable; and all authors are agreed that it is one of the most infective diseases with which we are acquainted. It is contagious from the earliest active stage to the end of convalescence, and possibly even during the stage of incubation.

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[1] *Medical News*, January 26, 1895, vol. lxvi. p. 94.

[2] Saunders' *Year-Book* for 1899.

(c) Transmission by fomites is common, the poison adhering to clothes, body- or bed-linen, etc., and evidence is not wanting to show that the poison is highly tenacious of pathogenic power. Its vitality is retained after death, and the room occupied by a patient, the bedding, and the articles of furniture all serve to convey the disease unless thorough disinfection be enforced. The *infection atrium* for the poison into the system is probably the *respiratory tract.*

Clinical History.—Incubation.—This stage varies with the mode of communication of the poison. If following inoculation, the symptoms appear in six or seven days; when originating from infection, usually in twelve days, though this stage may be either lengthened by a day or two or shortened to an equal extent. During a portion of this period complaint may be made of certain ill-defined symptoms, but these are usually absent, and the onset is sudden and accompanied by characteristic signs. These are—a *severe rigor, high fever, headache, and intense lumbar pains.* Instead of the usual severe rigor, repeated chills, extending over twelve to twenty-four hours, may occur. The symptoms of the onset have been milder in the recent outbreaks, although similar in character to the severer types of former epidemics. During the preliminary fever the *respirations* are accelerated, the *pulse* becoming decidedly more rapid, and there may be general bronchitis. The tongue is coated and slight pharyngitis may exist. There are anorexia, general vomiting, and constipation or rarely diarrhea. Restlessness, delirium, and stupor are the principal nervous symptoms observed. Infective albuminuria is common; of 1400 cases of variola, Arnaud noted albuminuria in 95. In the female menstruation is apt to occur.

The **physical signs** referable to the lungs are few, and consist of a few dry and, later, moist râles, heard on *auscultation.* *Palpation* detects splenic enlargement. From the second day the so-called *initial rashes* may appear: (*a*) the *diffuse scarlatinous eruption,* which in no way differs from ordinary *scarlâtina;* (*b*) the *measly eruption,* which may be diffuse and present a striking similarity to that of measles. Either associated with these or occurring independently there may be a hemorrhagic eruption (usually purpura), the petechiæ coming out by natural selection, mainly upon the hypogastric region or the inner surfaces of the thighs and in the axillæ (Simon). The initial rashes occur in a considerable proportion of cases (10–15 per cent.). The *stage of invasion* lasts three days as a rule. The temperature then declines rapidly, while at the same time the true *variolous eruption* appears upon the skin and mucous surfaces. It develops first upon the face, particularly upon the forehead and the hairy scalp, and spreads in a downward direction till it reaches the legs, where it last appears. The skin in the femoral triangle rarely shows the true variolous eruption. Each pock passes through the various stages noted in the pathologic description—viz. papule, vesicle, pustule, and scab; and when the stage of pustulation has been reached a secondary fever develops. During the following remission of fever the headache, lumbar pains, etc. subside. The fever of suppuration which then succeeds is accompanied once more by marked constitutional disturbances, particularly nervous derangements (wild delirium, etc.), and at this time complications are also apt to develop. On the eighth or ninth day of the eruption (the twelfth or thirteenth day of the disease) the pustules begin to dry up, forming yellow crusts; the redness and swelling of the skin subside; and two or three days later the scabs loosen and are thrown off. During this stage the fever again declines in company with the constitutional symptoms, and convalescence ensues. As previously stated, when suppuration involves the true skin scars are the inevitable result, and these remain to the end of life. The hair drops off sometimes, even to the extent of total alopecia, but is generally renewed.

The general *course* described above is that of an average case, but, as I have said, a remarkably mild variety of variola has prevailed in the last few years in America. That the disease has become progressively milder in character in recent times is unquestioned.

The cases met with in the recent epidemic appearances in numerous sections of this country have been unusually mild, and also manifested strange features; and to these the terms "modified" and "mitigated" small-pox may be appropriately applied. Although not all cases were equally mild, many of them were practically ambulatory, since they would not remain in bed after the eruption appeared, and this was also true of those unprotected by previous vaccination. There was little or no secondary fever, and desiccation was rapid. Says W. M. Welch,[1] whose experience is unparalleled: "I must say I have never seen cases present, uniformly, so mild a type as during the present year (1899), nor have I been able to find in the vast amount of literature published on the subject any account of a similarly mild epidemic in this or any other country.

Leading Symptoms and Complications.—(*a*) **Eruption.**—The eruption in the more typical cases appears at the end of the third or on the fourth day, coming out first upon the forehead, particularly along the border of the hairy scalp, and spreading in a downward direction in regular progression. It appears in the form of slightly elevated maculæ, which are at first of a pale-red color, and later assume a darker red hue, resembling small fleabites. These increase in size during the next forty-eight hours, at the end of which period they are developed into (1) *papules*. The change of character is accompanied by intense itching and burning of the skin-surface. To the feel they are papular, like shot under the skin. The eruption is always most abundant upon the face and scalp, while the hands and fingers are the next most favored seats. At the end of the third day (the sixth day of the disease) the conical apices of the papules contain liquid, forming thus (2) *vesicles*. The latter increase in size till the entire papule is converted, at the same time acquiring more and more decidedly a central umbilication. Puncturing a vesicle does not cause it to collapse, but allows only a small portion of its liquid contents to escape, owing to its reticulated character. As the vesicle increases in size its contents become opaque, and in three days more, or about the sixth of the eruption, the vesicles become (3) *pustules*. Umbilication now disappears, and the pustule looks full and well rounded, and is surrounded by a red border or "halo." If the pocks be close set, as on the face, wrists, and fingers, the intervening skin is inflamed and swollen and the itching and burning become almost intolerable. The pustules may coalesce along their edges, and thus the eruption becomes confluent. The eyes are closed as the result of swelling and tumefaction of the face, and the hands and feet assume a rounded, ball-like appearance. The face, as a whole, is markedly misshapen and is ultimately disfigured. When the pus is not liberated (a comparatively rare event), its desiccation begins on the ninth day (the twelfth day of the affection); if the pustule is ruptured earlier (as when confluence occurs), it begins at an earlier day. (4) The *scabs* now form, and remain until about the twelfth day of the eruption, and when pits or scars result they gradually fade until they remain as permanent whitish spots.

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[1] *Loc. cit.*

The eruption upon the mucous membrane develops simultaneously with that of the skin, and among favorite surfaces for its appearance are the mouth, tongue, soft palate, and pharynx (causing *dysphagia*), the nasal chambers (causing *coryza*), the larynx (causing *hoarseness*), the trachea and bronchi (causing *bronchitis*). This mucous efflorescence does not proceed to the development of pustules, but forms ordinary ulcers as a consequence of early maceration of the superficial layers of the mucosa, and these ulcers also may become confluent.

The *skin* presents certain complications that are always secondary and are deserving of mention (erysipelas, abscess, gangrene, bed-sores).

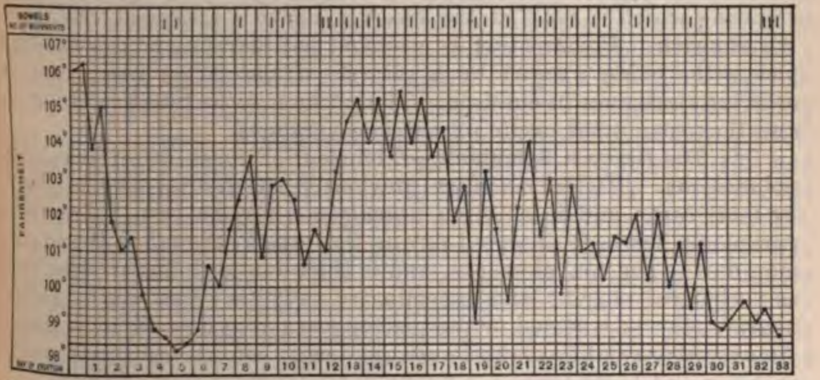

FIG. 19.—Temperature-chart of a case of variola, from a patient in the Municipal Hospital, Philadelphia. A. F——, aged three years; not vaccinated.

(*b*) **The Fever.**—The temperature at the onset rises rapidly, and may touch 103° or 104° F. (40° C.) on the first day, its range being high and of the continued type during the invasion period. Evening temperatures of 105° F. (40.5° C.) or higher may be observed, and in three days (or with the first appearance of the papules) the temperature remits, but does not intermit in true variola. It remains at a low elevation till the stage of suppuration is reached, when a fresh rise occurs. This secondary fever-curve is apt to show exaggerated points of elevation and deep remissions. The latter are generally the result of septic absorption (the fever of suppuration). Secondary fever, since the variolous infection has grown milder in type, is often slight or may be wanting (*vide supra*). This period lasts from one to three or four days in typical cases. When desiccation of the pustules begins defervescence also commences, and proceeds in a gradual manner by lysis. There may be a post-variolous rise, and if so its presence is to be attributed to some sequel or other.

(*c*) **The Circulatory System.**—The *pulse* is soft and much accelerated (100 to 130) and of good volume during the invasion stage. It is slower during the period of remission, only to be greatly increased in frequency during the second stage of fever. During the latter period it may, owing to cardiac failure, become very rapid, and finally irregular or even intermittent. Simple *endocarditis* rarely, and *pericarditis* somewhat more commonly, occur as complications.

(*d*) **Respiratory Tract.**—The *laryngitis* and *pharyngitis* which are due to the presence of pocks in the respiratory mucosa have already been mentioned. *Laryngeal perichondritis* with edema of the glottis, the latter perhaps being the result of a direct extension of the pock-ulcers to the perichondrium, may arise; it is ominous. Chief among the grave secondary complications is *bronchopneumonia* (inhalation pneumonia); and *lobar pneumonia* also occurs, though rarely. *Pleurisy* is not infrequent, particularly as an associated condition in bronchopneumonia.

(*e*) **The Digestive System.**—The variolous efflorescence in the buccal and pharyngeal mucosæ may be an agency in predisposing to a secondary inflammation in adjacent organs—*e. g.*, suppurative otitis media, suppurative parotitis, pseudo-diphtheria, etc. *Palpation* almost always shows an enlarged spleen, and not infrequently an enlarged liver. The vomiting which is usual at the onset is due to a catarrhal condition of the stomach. Constipation is common, but diarrhea is also sometimes met with, being excited by a catarrh of the large intestine, and is especially common in children. The pocks may be found in the rectum and they sometimes excite dysenteric symptoms.

(*f*) **The Nervous Symptoms.**—The chief of these have been already pointed out. *Violent delirium* (previously referred to) may be followed by fatal *coma*, and in children *convulsions* may be seen. Very rarely paraplegia has been observed during the attack, though it is more common during the convalescence, and is then due to different causes, such as peripheral neuritis and disseminated myelitis (Westphal). *Multiple neuritis* may be a sequel or the pharyngeal nerve may alone be affected. Among other conditions rarely arising during convalescence are insanity, epilepsy, and hemiplegia.

(*g*) **The joints** may be swollen and painful after small-pox, and in rare cases periostitis may be observed.

(*h*) **Renal Symptoms.**—Apart from the febrile albuminuria already referred to, renal complications are of great rarity. Hemorrhagic nephritis may occur, and is always of a serious character, though, fortunately, it is very rare.

(*i*) **The Special Senses.**—The pustules may form upon the conjunctivæ and eyelids, and several important conditions result from this variolous involvement of the eye—viz., conjunctivitis, keratitis, choroiditis, and panophthalmitis. Hebra met with ocular complications in 1 per cent. of 5000 cases of small-pox. Otitis media has already been mentioned.

Special Clinical Forms.—There are two unusual types of variola that are important in being severer than the moderate (discrete) form already described.

(*a*) **The Confluent Form.**—This is the result of an abnormally severe infection, and is less common than formerly. The *ushering-in symptoms* are very severe, and the eruption may appear as early as, or even before, the third day, when the temperature remits. The separate papules are vastly more abundant and close-set; and after the stage of pustule is reached the face and hands present an uninterrupted area of suppuration. The deformity of the countenance is correspondingly pronounced. Naturally, the *local symptoms* are intense and the fever and its concomitants are in exact proportion. The *nervous symptoms* often predominate. Salivation is frequent. The eruption may also entirely cover the mucous surfaces. The *lymphatics* of the neck may be greatly swollen—a circumstance that contributes to the facial disfigurement. The various complications previously adduced are of comparatively frequent occurrence, and following these a general pyemic process may develop. When death occurs it is usually preceded by the phenomena of the *typhoid state* (typhomania, tremors, subsultus tendinum, a rapid, feeble pulse, dry, brown tongue, and diarrhea). On the other hand, if recovery ensues, it is tardy.

(*b*) **Black Small-pox.**—In this form the blood is much changed, so that hemorrhages into the skin, mucous membranes, and various viscera occur. It is important to distinguish several sub-varieties, as follows: (1) A *benign* form, in which blood is infused into the pustules when patients are allowed to leave their bed too early in convalescence. Here the condition is due to the effect of gravitation, and hence is confined almost solely to the lower extremities. (2) Doubtless the ordinary variolous eruption may become *slightly hemorrhagic* without aggravating the constitutional condition. (3) A *dangerous hemorrhagic* tendency may be manifested. During any of the eruptive stages—papular, vesicular, or pustular—hemorrhages may occur into the eruption, and, moreover, free bleedings may take place from the various mucous surfaces. The initial symptoms are usually intense, the eruption abundant, and in consequence of the hemorrhages collapse often occurs. The most serious complications, pneumonia, diphtheria, and nephritis (followed by uremia), are also apt to develop and terminate life. This and the subsequent sub-variety are truly anomalous. (4) A not uncommon form of hemorrhagic variola is met with in which the *acute hemorrhagic diathesis* develops during the period of invasion. Its onset is characterized by the usual symptoms intensified, and as early as the second day ecchymotic patches appear upon the skin-surface and grow rapidly by peripheral extension, the mucous surfaces also showing more or less extensive ecchymoses. The regular variolous eruption rarely appears, though occasionally shot-like papules may be detected here and there. The temperature may be slightly elevated, but is rarely high. Death often occurs before the time for the appearance of the characteristic eruption.

There are also varieties of small-pox that pursue an abnormally mild course. Of these, (*c*) **varioloid** deserves first place. By this term is usually meant small-pox occurring in individuals who have been protected by a successful vaccination, but it may also be the result of natural insusceptibility. Hence variola and varioloid are one and the same affection. The *initial symptoms* of varioloid do not differ either in character or severity from those of true variola, but the general course of the attack is peculiarly prone to manifest irregularities. In the pre-eruptive stage an erythematous rash is very common, and its appearance is regarded by many as being of value in discriminating varioloid from variola.

When the *regular eruption* appears the fever falls to normal and remains there. The rash comes out by the end of the first or on the second day, the papules being scanty, and quite as liable to appear first upon the trunk as upon the face. They are identical with the papules of variola, as is true also of the vesicles; but pustulation rarely develops, for the reason that the resolution takes place, as a rule, before the latter stage is reached.

The *secondary fever* is either very slight or entirely wanting. The mucous surfaces are affected only slightly. Papules and vesicles may be found in close proximity; not so in variola. Desiccation begins between the fifth and seventh days of the eruption (the eighth and tenth of the disease), and hence, as compared with variola, the course is cut short and serious complications almost never occur. There has been noted the same marked tendency to extreme mildness of phenomena that characterize variola in the recent epidemics.

(*d*) An **abortive** form is occasionally observed. Many of the recent cases belonged to this variety. It is characterized by the great intensity of the invasion symptoms, but these promptly subside, and the patient enters at once upon a stage of speedy recovery.

An exceedingly mild type may arise during seasons of epidemic prevalence of the disease, either with or without a scanty and undeveloped eruption, when the diagnosis is made entirely from the etiologic circumstances.

Diagnosis.—With a clear history and the presence of the characteristic features a positive diagnosis is a simple problem. But at any period before the papules are fully developed it may be confounded with certain other acute infections, notably cerebro-spinal meningitis, typhus fever, scarlatina, and measles. After the variolous eruption makes its appearance the disease may be confounded with impetigo contagiosa, pustular syphiloderm, and varicella.

Differential Diagnosis.—In *typhus fever* the onset is very like that of small-pox. The former may, however, be distinguished by its peculiar etiologic factors, especially its origin by importation or its non-prevalence in the vicinity; the appearance of the eruption, first upon the trunk (chest and abdomen) in the form of maculæ, and later becoming petechial. Moreover, in typhus the temperature does not remit with the appearance of the eruption.

From hemorrhagic small-pox *typhus* is sometimes distinguished with great difficulty. In the former death often occurs before the eruptive stage is reached. In typhus shot-like papules are never detected upon the skin-surface of the hands and head, whereas they are sometimes found in hemorrhagic small-pox.

Hemorrhagic small-pox may be simulated by *cerebro-spinal meningitis.* If the history be not clear, then recourse to lumbar puncture will settle the doubt.

Scarlatina may early be distinguished from the erythematous (scarlatinous) rash which often precedes the appearance of the variolous eruption; this is, as a rule, neither so intense nor so uniformly distributed over the skin-surface of the body as in true scarlatina.

The *macular stage* of the eruption may be confounded with *measles.* The absence of the characteristic prodromes and symptoms of invasion belonging to the latter disease, the redness and swelling of the conjunctivæ, the photophobia and marked coryza, the stubborn cough, and increased fever after the eruption appears, make the separation easy, as a rule. After the maculæ develop into hard, shot-like, conical papules the scales are turned in favor of variola.

Impetigo contagiosa presents no initial stage; it begins as vesicopustules (not papules) which appear "on the normal skin and are superficial and enlarge by peripheral extension, often attaining the size of a 10-cent piece and having a flat appearance" (W. M. Welch). The patient may infect new areas by scratching. Scars do not result.

Syphilis distinguishes itself by a milder initial stage, by the indurated base of the pustule, by the appearance in crops of the skin-lesions, and by the polymorphous character of the latter. There is neither umbilication nor characteristic pitting after the scabs fall, but a coppery hue remains.

Nothing, however, could be more difficult than to differentiate certain mild cases of discrete small-pox (in the non-vaccinated) and varioloid from *varicella.* In the table below, however, may be found a few contrasted points of distinction, which, I trust, may prove helpful:

VARIOLA.	VARICELLA.
History.	
Absence of previous attack.	Same.
Previous or present case in the vicinity.	Traceable to previous or present case of varicella.
Not successfully vaccinated.	Negative.
Occurs at any age.	Almost always in childhood.
Characteristic pre-eruptive stage—rash on the third day.	Eruption not preceded by prodromes.
Eruption.	
Appears first upon the forehead, extending downward.	Appears first over parts covered by clothing. No regular progression over the body.
Vesicles uniform in size, umbilicated, and deeper seated.	Vesicles vary much in size, are rarely umbilicated, and feel soft and velvety.
Eruption contains serum, later pus.	Only serum.
Most abundant on face and fingers.	Most abundant upon back and lower extremities.
Various stages of eruption observed at points removed from each other.	Various stages side by side.
Pin-prick does not cause collapse of vesicles, being multilocular.	Does cause collapse, being unilocular.
Secondary fever usually present.	Absent.

Prognosis.—The prognosis depends upon (*a*) the degree of severity of the type, the severer forms (confluent and certain of the hemorrhagic) being grave. The hemorrhagic variety, in which large ecchymoses suddenly develop, is almost invariably fatal, and often before the cases have advanced to the eruptive stage. The aggregate number of pocks that appear and the gravity of the infection are, as a rule, proportionate.

(*b*) The prognosis is modified by *individual peculiarities* (age, race, intemperance). Thus it is much more fatal in the very young than in older subjects, much more fatal in dark- than light-complexioned races, more fatal in the intemperate than in the temperate, and so on.

(*c*) Complications increase the death-rate considerably. Of these, bronchopneumonia, lobar pneumonia, acute nephritis with uremia, septico-pyemic conditions, pseudo-diphtheritic angina, and pericarditis are most potent for evil. Among the foremost serious symptoms may be mentioned excessive vomiting, wild delirium, coma, a temperature of 106° F. (41.1° C.) or over, urgent diarrhea, and dysentery.

The death-rate has been computed to be between 15 and 30 per cent., varying, however, with each epidemic. During the recent widespread prevalence of the disease in the United States the mortality rate was unprecedentedly low.

Treatment.—The varied indications in the treatment of small-pox will be considered separately:

(1) **Prophylaxis.**—The rules that have been laid down elsewhere (*vide* Treatment of Typhoid Fever) for disinfection in infectious diseases must be rigidly enforced in this affection. Quarantine (*public and private*) must be secured if the deadly progress of small-pox is to be averted. Absolute isolation cannot be carried out successfully in private houses, and in view of this fact special, well-equipped hospitals should be provided for the reception of the disease. It is important also to remember that persons who have been afflicted with the disease cannot, with safety to others, resume their former places, either in the family or in society at large, before they are completely convalescent. The best means of prevention, however, is vaccination, and this subject will receive separate consideration (*vide* p. 231).

(2) **General Management.**—The room occupied by the patient should be large and freely ventilated (an essential matter, though strong drafts are to be avoided), and all carpets, curtains, and articles of furniture not absolutely needful should be removed.

The *diet* is an element of treatment that demands most careful attention, and should be varied according to the stage of the affection. During the initial stage it must be restricted to liquid nourishment (milk, animal broths, etc.), and in addition cooling drinks, including ice, lemonade, and other of the various fruit-juices (diluted). During the stage of remission of fever we may add soups, jellies, eggs, toast, etc., and with the onset of the stage of suppuration a supportive diet, reinforced by the judicious use of stimulants, is an essential part of the treatment. Light forms of nourishment must now be given in definite quantities at short intervals.

(3) **The fever and associated symptoms** during the invasion stage are best controlled by the cold or gradually cooled baths, which possess all the advantages in this disease that they command in typhoid fever. Cold sponge-baths, the ice-cap, or the cold pack may be resorted to if cold immersion baths are not accessible to the patient. The internal antipyretics must be given with a sparing hand, if at all, and only as antiseptic agents, on account of their depressing effects.

The therapy of this stage also embraces the treatment of certain symptoms. The vomiting may be incessant and exhausting, and chipped ice, champagne, dilute hydrocyanic acid, and cocain hydrochlorate should be tried in the order mentioned. If diarrhea be severe, it should be checked (though neither wholly nor suddenly) by the use of arsenite of copper, the acetate of lead (gr. ij—0.1296) and opium (extr., gr. ¼—0.0162), in combination, or by bismuth salicylate (gr. v—0.324) and β-naphtol (gr. iij—0.1944). The nervous symptoms are usually restrained by the cold affusions, but occasionally a wild delirium may necessitate a combination of sodium bromid (gr. x–xv—0.648–0.972) with the deodorized tincture of opium (♏v—0.333), given every two or three hours. Very often the wise administration of stimulants removes all necessity for the use of further means of overcoming the nervous symptoms. The catheter must be used if retention of urine should occur. For the intense pains that belong to this stage no other remedy can be compared with morphin sulphate (gr. $\frac{1}{8}$ to $\frac{1}{4}$—0.008 to 0.016), to be administered hypodermically, and repeated if necessary; this measure also ensuring good sleep.

(4) As previously stated, the **eruption** appears with the termination of the initial febrile period, and deserves the closest attention. The indications are twofold: (*a*) to limit the eruption as far as is possible, and (*b*) to modify its course, so that extensive suppuration and consequent disfigurement may be prevented. Ablutions with lukewarm water, to which may be added some antiseptic (carbolic acid and glycerin, or, better, a mercuric-chlorid solution—1 : 5000 or 1 : 10,000) will be found of great use. To prevent pitting many local applications have been used. Formerly, a common mode of treatment was to open the pustules as early as possible and touch them with silver nitrate—either in the solid stick or brushed over in a strong aqueous solution. The formula of Schwimmer, herewith given, gave excellent results in a case of my own:

℞. Acid. carbolici,	4.0–10.0;
Ol. olivæ,	40.0;
Cretæ præparat.,	60.0.

M. et ft. pastamolis.

It has been recommended to touch each pustule with carbolic acid, and then to apply this agent in equal parts with the oil of thyme (Sansom). It is important that only a certain proportion of the pustules be touched at once. The parts must be kept aseptic and clean, while irritation from scratching, etc., must be carefully avoided. Moore and Fingen have recommended the use of red curtains or shades to cut out certain chemical rays. J. Moir,[1] from an experience with 4000 cases, increases the elasticity of the skin by rubbing it with oil early in the disease; this tends to prevent pitting.

During convalescence, or as soon as the general condition of the patient will admit of it, warm baths, with the free use of carbolic soap, are to be given at intervals of two days until several baths have followed the separation of the crusts.

(5) **The Period of Remission of Fever.**—There are very rarely any symptomatic indications apart from those presented by the eruption. It is of first importance, however, to support the powers of the system by means of tonics, nutrients, and stimulants.

(6) **The Suppurative Stage.**—All measures tending to support the strength of the patient are needed—the mineral acids, with the elixir of calisaya, quinin, strychnin, etc. Stimulants are often required, and it may become necessary to give them unsparingly, the character of the pulse being the physician's principal guide as to dosage. Gradually

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[1] *Edinburgh Medical Journal*, June, 1898.

cooled baths of the usual duration or warm baths somewhat more prolonged give excellent results. In this stage certain symptoms may require special treatment. The ulcers in the mouth and throat are best relieved by the use of a saturated solution of chlorate of potassium in water as a gargle or in the form of an atomizer spray. Ice allowed to melt in the mouth is also valuable. Hemorrhages demand prompt interference, and full doses of ergot must be given subcutaneously. Internally, the tincture of the chlorid of iron, gallic acid, the mineral acids, or turpentine may be administered.

The complications, as before intimated, are not numerous, and are for the most part secondary in nature. By frequently changing the position of the patient when bronchitis is present, and by encouraging him to cough frequently, as well as by the timely use of stimulants and the proper care of the mouth, pulmonary complications can often be obviated. Should lobular pneumonia occur, the plan of treatment which is likely to meet with most success may be briefly put thus: Free stimulation with alcoholics, the assiduous use of cold sponges or gradually cooled baths, and nourishing foods. *Laryngeal perichondritis* with edema of the glottis may suddenly demand tracheotomy. To avoid the development of *bed-sores* an air-cushion or a water-bed should be provided, if needful. Care should also be exercised to prevent ocular complications, and their occurrence demands a vigorous form of supportive treatment. I have much confidence in the use of cold compresses, instilling into the eyes at the same time a solution of boric acid (gr. x to xv—0.648 to 0.972—to f℥j (30.0).

(7) **Special Modes of Treatment.**—These would be found to be numerous were we to enumerate all of them, but only those based on the principle of antisepsis are worthy of notice. According to one plan, which has many advocates, antiseptic agents are administered internally. The remedies that have been most frequently employed in this manner, and with perhaps the most promising results, are the sulphocarbolates, salol, sodium salicylate, carbolic acid, creasote, mercuric chlorid, and the sulphites. R. A. Woodson[1] adopted as the general plan of treatment in the Holguin epidemic, daily scrub-baths, 1–2000 mercuric chlorid, and open-air treatment.

Kinyoun, Landmann, and Béclère have used the serum from vaccinated subjects (human beings and the lower animals) or from variolous patients in advanced stages of the disease in the treatment of small-pox. The cases, however, are insufficient to warrant deductions.

Special Methods of External Medication.—Talamon[2] recommends a special plan of external medication in the form of a mercuric-chlorid spray for small-pox vesicles and pustules, his object being to keep the surface under the influence of an antiseptic. The mercuric-chlorid solution is prepared after the following formula:

℞	Mercuric chlorid,	gr. xv (1.0);
	Tartaric acid,	gr. xv (1.0);
	Alcohol (90 per cent.),	f℥jss (6.0);
	Ether to make	f℥jss (45.0).

Sig. To be applied as a spray three or four times daily for one minute.

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[1] Saunders [illegible] [2] [illegible] Diseases, February, 1891.

It is essential to exercise the precaution to protect the eyes, which may be covered by layers of cotton dipped into a saturated solution of boric acid. Talamon advises the commencement of his method on the first day of the eruption, the application to be preceded with a vigorous washing of the face with soap, which may be rinsed off with boric acid and then dried with absorbent cotton. After the spray has been used the face should be covered with a layer of a 50 per cent. glycerolate of mercuric chlorid in order to keep the skin continuously aseptic. After the fourth day the number of sprayings per diem is gradually lessened, so that by the seventh day they may be discontinued; but the application of the glycerolate should be continued.

Talamon added, in the confluent and other grave forms of the disease, general mercuric-chlorid baths, lasting for three-quarters of an hour to an hour. The buccal and pharyngeal eruption is to be treated by gargles and lotions of boric acid. Internally, the therapy is limited to sustaining the strength of the patient by means of alcohol (℥iij–iv; 90.0–120.0 daily), according to the gravity of the case.

VACCINATION.

Historic Note.—One of the first steps in preventive medicine was the practice of inoculation as a method of protection against the infection of small-pox. It had been practised in China and other Asiatic countries for centuries, and Lady Montague, the wife of an English ambassador to Turkey, early in the eighteenth century introduced it into England, after which time and until vaccination was known, it was very extensively practised there.

Pus taken directly from a small-pox pustule was introduced beneath the epidermis, and the person inoculated developed variola, though in a milder form than when arising from ordinary infection. The attack ran a more rapid course, having fewer pustules, rarely terminating fatally, and protected the individual from subsequent attacks.

The objections to this method were that it did not always produce a mild form of variola, a small percentage of cases having a fatal termination, and that, however mild the attack, other unprotected persons brought in contact with it were as liable to contract small-pox in as virulent a form as if contracted in the usual way.

In a paper published in 1798, Edward Jenner, a physician of Gloucestershire, England, and a pupil of John Hunter, first made known to the world the value of vaccination. Twenty years previous he had observed that persons employed in dairies, who were accidentally inoculated with cow-pox, were insusceptible to the contagion of small-pox, and, after experimenting all these years, he became satisfied that inoculation with the vaccine lymph was a preventive against small-pox. After the publication of his paper he was subjected to ridicule and abuse by the profession, but through his persistence he was finally allowed to practise his method of vaccination in the wards of a hospital, and in the course of a few years it became generally recognized and was practised in France and America, as well as in England. Later, the method fell into disrepute for a time, owing to the fact that certain persons who had been vaccinated subsequently contracted the disease, it not being known then that a revaccination was necessary from time to time. Strange to say, however, in the century that has passed since the first vaccination by Jenner there has practically been no change or improvement either in the method or the vaccine used.

Vaccinia, or cow-pox, is a mild eruptive disease that occasionally occurs among cattle, a similar disease being produced in them by inoculation with the small-pox virus from man. It is communicable by contact only, and is usually carried from one cow to another by the hands of the milkers; hence being usually found on the udder or teats of milch cows. Since Jenner's time many theories have been advanced as to the exact nature of this disease in cattle, and at the present day the subject is still in dispute. It is now, however, generally conceded that if cow-pox is a distinct disease, originating only with the cow, the eruptive disease produced in this animal either by inoculation of small-pox virus from man or of "grease" from the horse is, at least in all essential respects, a disease not to be distinguished from primary or idiopathic vaccinia. Guarnieri has described certain parasitic organisms, the *Cytorectes Guarnieri*, found in corneal lesions produced by the injection of vaccine lymph. This observation has been confirmed by Pfeiffer and others, but the pathogenic nature of these protozoa has not been determined.

The *vaccine virus* consists either of the liquid contained in the vesicle or of the scab resulting from the desiccation of the pustule. The former is furnished from vaccine farms, of which there are several in this country, is then dried on ivory points, and, if kept in a cool place, retains its virtue for a week or ten days, or, possibly, longer, but should be used as fresh as possible to ensure a successful result. It is also sometimes preserved in capillary glass tubes, sealed at both ends, or between glasses, and kept in this way it is less liable to infection through uncleanliness in handling. The scab from the cow is not used.

The Site.—The point usually chosen for vaccination is on the arm over the insertion of the deltoid muscle; but in girls, for cosmetic reasons, it is sometimes preferred on the leg, and the most common site is over the junction of the two heads of the gastrocnemius muscle.

Technique in Vaccination.—After the part selected has been rendered surgically clean, gently scrape the skin with an aseptic lancet or other instrument until serum begins to exude. If by too vigorous scraping blood should be drawn, it must be carefully dried with a piece of sterile cotton before the lymph is applied. Hutchins has recommended a method in vaccination of denuding the surface of the skin with a caustic in place of the lancet. A piece of cotton, as large as the spot to be denuded, is wet with liquor potassæ and laid on the skin for two or three minutes, after which the spot is wiped dry and the softened epidermis rubbed away with an ink-eraser, a piece of soft wool, or preferably a piece of sterile gauze cotton, when the vaccine is applied in the usual way. The advantages of this method are its painlessness and the absence of terrifying instruments and bleeding.

The charged end of a point, which has been previously dipped in tepid water, is now gently rubbed over the abraded spot and the limb left exposed to the air until the lymph has been dried upon it. It may then be protected by a piece of gauze strapped on it or by a shield.

Humanized lymph is still preferred by some, and when this is used the "arm-to-arm" vaccination is best. The lymph is taken from a characteristic vaccine vesicle (from the fifth to the seventh day of its development) of a healthy child and applied directly to the arm of another. The virus may be dried and preserved for use as in the case of bovine virus.

The *scab* resulting from a vaccine vesicle on a healthy child was formerly quite generally used, and it could be kept a long time without losing its virtue. It was sure in its action, and offered the advantage to the physician of being easily preserved; but it was more liable to become infected than the lymph when preserved in the usual way, and, since the vaccine farms are so conveniently located, lymph may be obtained from them at any time without delay.

The possible danger of conveying syphilis or other constitutional disease from one person to another by means of humanized lymph should lead to its abandonment in favor of the bovine lymph.

Period of Life for Vaccination.—It is usually advised to vaccinate infants within a few weeks or months after birth, but, unless small-pox is prevalent, it is best to wait until the latter part of the second or the beginning of its third year, as the child has then passed through its teething period and will be better able to resist the effects (slight though they may be) consequent upon vaccination.

Time for Revaccination.—To ensure the individual against infection he should be revaccinated at puberty and every few years afterward, or at any time when small-pox is epidemic or liable to become so.

Symptoms.—After vaccination no local or constitutional effects—except the slight irritation due to scarification—are noticed until the third day, when a *small red papule* appears. By the fifth or sixth day a *vesicle* appears. By the ninth day it is fully developed, and, like the vesicle of variola, is filled with colorless lymph, is umbilicated, multilocular, and has a distinctly inflamed areola of deep red color, accompanied by heat, itching, and tenderness. By the tenth day this may extend an inch or two from the vesicle. Quite frequently the axillary or inguinal glands (depending upon the location of vaccination) are swollen and tender, and in a tubercular child they may go on to suppuration. After the tenth day all these symptoms gradually decline; the *pustule* dries up, and then forms a brown scab which is usually detached in the third or fourth week, leaving a permanent cicatrix.

Complications.—Occasionally one or more additional vesicles are formed at a little distance from the point of inoculation, and, rarely, there is a general vesicular eruption, due to absorption of the lymph. An *erythematous rash* is not uncommon, and appears, if at all, about the sixth day. *Erysipelas* may occur as a complication, and, if it is prevalent in the house, vaccination should, as a rule, not be performed; if deemed necessary, great care should be taken to ensure cleanliness.

An ulcer may form which may be weeks in healing. *Eczema* and other skin-affections are usually aggravated during the course of vaccination, and it is possible for *syphilis* to be inoculated with the vaccine virus. Any of these complications call for the usual treatment.

VARICELLA.

(*Chicken-pox.*)

Definition.—An acute, contagious disease, characterized by a cutaneous eruption of papules, passing into vesicles and pustules; also by slight fever and mild constitutional symptoms. For a long time it was confounded with varioloid, but its distinct character has now been recognized for many years. Complications and sequelæ are infrequent.

Etiology.—It is well established that the contagium of varicella is found in the vesicles, as the disease has been communicated by actual inoculation with their contents. The specific poison has not been satisfactorily isolated, although it is suspected that certain protozoa are the direct cause, but, as in the case of vaccinia and small-pox, positive proof is wanting. Varicella may be transmitted by exposure to another case or possibly through the medium of a third person, the school and asylum being the most frequent points of its origin. It affects children of all ages, and usually one attack is protective. It closely resembles measles in its contagiousness.

Symptoms.—The *incubation* period is uniformly from fourteen to sixteen days. If there be a prodromal stage of the disease, certainly in the vast majority of cases it cannot be recognized, though a slight *fever* and general indisposition may be noticed for twenty-four hours before the appearance of the eruption. In many cases the *eruption* is the first symptom. This occurs in the form of small reddish puncta, from which rapidly develop rosy-colored maculations, and these become tensely distended, transparent, or slightly yellowish vesicles of the average size of a split pea. The eruption *appears first* upon the upper part of the body, the chest and back, neck, scalp, and face (on the latter quite sparingly), and always upon the hairy scalp. Frequently the *vesicles* form on the mucous surface of the lips, inside the cheeks, on the tongue, palate, conjunctivæ, and in the progenital regions of both sexes. At times the glands of the throat become slightly enlarged and painful, the vesicles are superficial, the child has the appearance of having received a shower of boiling water, and the firm papule which precedes the variolous rash is altogether wanting. The vesicles are at first transparent, and their contents plainly show through their translucent roof-wall which is composed only of the stratum corneum of the epidermis. *Umbilication* rapidly occurs at the apex, and the contents of the vesicles become lactescent, and gradually sero-purulent. The *areola* is most distinct when the vesicle is fully formed and fades as the latter dries. *Crusts* form, which drop off in from five to twenty days, depending upon the depth to which the skin has been involved. On the trunk, as a rule, no mark is left, but after the more severe attacks, when the true skin has been involved, scars remain, and frequently there is quite deep *pitting*. The marks are usually on the face when the skin has been unprotected. On the hands and feet the vesicles appear without having been preceded by a papule, and sometimes there is no areola, each vesicle resembling a drop of water upon a healthy skin. *Pustules* may develop in consequence of irritation or infection, as the result of scratching, or in feeble or poorly-nourished children and in unhealthy children deep ulceration may occur, lasting for weeks. In rare cases there may be necrotic inflammation about the site of the pox (*varicella gangrænosa*).

In *mild cases* only ten, twenty, or thirty spots may be found on the body, but in severe cases the skin may be almost covered in certain regions. The eruption, however, is never confluent. The *temperature* is highest on the second or third day, when the eruption is appearing. In mild, uncomplicated cases the thermometer registers 101° or 102° F. (38.8° C.) for two or three days at most, but in severe cases the temperature may be as high as 104° F. (40° C.). This is usually due to broken health prior to the acute illness. The temperature falls *gradually* as the rash fades, and presents a temperature-curve similar to that of measles.

There is usually neither coryza, vomiting, cough, nor diarrhea, and in their place is only the general indisposition which is associated with any febrile disease.

Complications.—*Erysipelas* occasionally acts as a serious complication in delicate children. It may develop about the pocks, particularly when they are deep and associated with some ulceration, and scratching with unclean fingers is its prime causal factor.

Adenitis, mild and isolated, and *suppuration* with abscesses in the deeper cellular tissue are occasionally seen.

Nephritis is infrequent, but may occur in unhygienic surroundings or in carelessly managed cases, just as it may follow scarlet fever or measles.

Varicella is also quite frequently complicated with other infectious diseases, and varicella, scarlet fever, and measles have been seen curiously blended in epidemic form. Varicella and measles, however, are most frequently associated.

The **diagnosis** of varicella offers no special difficulties. The eruption comes out slowly and in crops, so that papules, vesicles, and crusts may be seen upon the skin in close proximity. Again, it should be noted that the umbilication is due only to the fact that the drying up of the vesicle begins at the center, and that the pocks may appear on the mucous membrane. Varicella is distinguished from *urticaria* by the presence of fever, and from *eczema pustulosum* by the mild febrile symptoms of the latter, the discreteness of its pustular lesions, the absence of itching and of infiltration of the skin in patches, and by its tendency to symmetric development.

Variola and *varioloid* of infants are to be distinguished from varicella by the prodromal symptoms, and by the greater rise of temperature, though the distinction between mild varioloid and severe varicella in infancy and childhood will always tax to the utmost the skill of the keenest diagnostician. The sooner it is understood that intermediate forms are likely to occur, which cannot be positively assigned to one or the other category, the better it will be for both the profession and the laity.

The **prognosis** in private practice is always favorable. Only in the slums or in hospital cases complicated by erysipelas, adenitis, or nephritis may grave results be anticipated. The milder cases may, however, leave slight monuments of their existence in the form of one or more depressed cicatrices which may mar an otherwise beautiful face.

Treatment.—Isolation should be enforced in schools and in all institutions containing many young children. In private houses, unless the younger children are delicate, quarantine is unnecessary. The disease may be transmitted to others as long as the crusts are present, and hence isolation should be maintained until they have fallen off. In most cases constitutional symptoms of the disease are so mild as to require no treatment. It is best at the outset to place the child in bed for a few days, and sponge daily with warm carbolized water; the local itching may be allayed by sponging with a weak solution of carbolic acid or by the use of carbolized vaselin. When the crusts have formed, especially on the face, an ointment of zinc oxid containing ichthyol (2 per cent.) should be applied, and care should be exercised to keep the skin clean and to prevent scratching. In all cases the urine should invariably be examined several times during and following the attack.

SCARLET FEVER.

(*Scarlet Rash; Scarlatina.*)

Definition.—Scarlet fever, or scarlatina, is a self-limiting, acute, contagious disease, characterized by vomiting, fever (more or less typical), angina, and in twelve or twenty-four hours by a diffuse, punctiform, scarlet eruption, followed by membranous desquamation and, frequently, by nephritis. It is a disease of childhood, but may occur at any time of life.

Scarlatina is a widespread disease, though perhaps less universal than measles. It is endemic in all the large cities of the globe, and at intervals the cases multiply into more or less extensive epidemics. Smaller towns and rural districts are visited, and the epidemics are usually traceable to importation of scarlatinal poison, so that it may be stated that they never originate *de novo.*

Pathology.—There are no pathognomonic changes. When death occurs early the chief lesions are presented by the throat, while in addition engorgement of the viscera is noted, especially of the brain. The exanthem is rarely visible. In malignant types, however, in which the eruption is not seen during life, it makes its appearance rarely after death, and this aids in establishing the nature of the affection.

When death occurs at an advanced stage the lesions are those either of nephritis (with dropsy), or of septico-pyemia, or of inflammation of one or more of the serous surfaces (pleurisy, pericarditis, endocarditis, meningitis, etc.). Additional changes in the various viscera are, for the most part, identical with those met with in other acute infective diseases, and hence need not be described here. The blood, it should be pointed out, is dark, fluid, and coagulates feebly, owing to a decrease in its fibrin factors. The process of desquamation may be observed, together with more or less emaciation in protracted cases.

Among other lesions which are more or less peculiar to the disease are (*a*) The *eruption*, which is a dermatitis of very mild grade. J. F. Schamberg[1] points out that the discrete vesicles sometimes seen originate in the hair-follicles or in the deeper layers of the *rete*, and contain a turbid leukocytic fluid. (*b*) *Scarlatinal angina*, which in its mildest form usually presents more or less hyperemia and a slight swelling

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[1] *Proc. Phila. Path. Soc.*, Jan., 1901.

ing of the mucosa of the tonsils, soft palate, and pharynx. In the severer grades the inflammation is phlegmonous (scarlatina anginosa), and sometimes terminates in ulceration. There is great swelling (especially of the tonsils), and the formation of abscesses, due to secondary infection, is common. Extension of the purulent inflammation to the connective tissue of the neck produces marked induration, and more or less extensive abscesses may take place. Gangrene sometimes supervenes. (*c*) In certain epidemics a *membranous exudate* accompanies the *scarlatinal angina*, and this may or may not be truly diphtheritic. When it appears early it is non-diphtheritic, as a rule, and often due to the streptococcus; on the other hand, when it comes on late it often shows the presence of the Löffler bacillus. There is also a malignant form of membranous scarlatinal angina, occasioned by a secondary streptococcic infection (Hirschfeld). (*d*) *The Nephritis.*—The renal lesions are included in the description of "Acute Bright's Disease."

Etiology.—The **bacteriology** of the affection is imperfectly known. The streptococcus pyogenes has been found in nearly all the inflammatory complications of the disease, especially scarlatinal pneumonia and angina, and some pathologists (Babés, Bergé, Klein) have held it to be the cause. Marmorek, Raskin, and Mosny, however, believe that it is an example of mixed infection, the streptococcus being merely a secondary factor, and Marmorek has been confirmed in this view by the results of his experiments with antistreptococcus serum.

W. J. Class[1] first described an organism (*diplococcus scarlatinæ*), which in all probability is the specific causative factor of scarlet fever. His researches have been confirmed by those of Gradwohl,[2] Jaques,[3] Page,[4] and others. The habitat of the diplococcus is not known, but it has been found in the blood, throat, epidermal scales, and urine of scarlatinal cases. The size of the organism is variable, and it stains with standard watery dyes easily, uniformly, and regularly (Gradwohl).

Class[5] reports on his experiments to obtain an *antitoxin for diplococcus scarlatinæ*, in which he was successful, using swine as the largest animals susceptible; blood-serum, obtained from a female inoculated with the germ, protected guinea-pigs from cultures of the same germ, the control-animals dying in six or seven days.

The *general receptivity* for scarlet fever is not so great as in certain other exanthemata (*e. g.* small-pox, measles); hence in a household in which there are several children some are apt to escape the disease, even though all have been equally exposed.

The *virus* is probably contained in the excretions from the *throat*, *nose*, or *ear*, and in the epidermal scales thrown off from the surface of the body. It is also present in the blood of scarlatina patients.

Modes of Conveyance.—The majority of the cases are produced by *contagion*, and I have observed that a single contact of a healthy child with a scarlet-fever patient suffices to convey the disease. It is also communicated by *fomites*, and the poison of scarlatina contained in clothing retains its infective power for months. The patient himself is a center of infection until the end of the period of desquamation. Again, any objects (furniture, utensils, library books, toys) which the patient has touched or handled may serve to communicate the poison. The disease may also be transferred by persons who have been in the sick-room, while they themselves escape. Infected dairies have been known to disseminate the poison and give rise to epidemics. The infection may also be air-borne, though not for any great distance. Behle reports an outbreak of human scarlatina in swine, and it is fairly well proven that kine are susceptible to the disease, though in a modified form, and are potent to transmit it to man.

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[1] *Monthly Bulletin of the Chicago Dept. of Health*, March, 1899.

[2] *Philada. Med. Journ.*, March 24, 1900.

[3] *Bulletin N. W. Univ. Medical School*, March 31, 1900.

[4] *Journ. Boston Med. Sci.*, June 20, 1899.

[5] *Philada. Med. Journ.*, June 23, 1900.

Mode of Infection.—Most probably the poison is *inhaled* into the throat, where infection usually occurs; but it may gain entrance to the body through the *alimentary tract.* Infection may also take place *through the blood*, as is shown by the fact that children have been born in all stages of the disease. Artificial inoculation with the blood of scarlatina patients has resulted in more or less typical forms of the complaint. *Open lesions* predispose, but whether they are essential to infection is not known. Seitz believes there is strong evidence of a *family predisposition* to the disease, as 371 out of 800 cases occurred in 152 families.

Predisposing Causes.—(1) *Age.*—The period of chief liability is from the second to the tenth year, after which it diminishes. It is rare under the age of one year, and especially so under six months. (2) *Recent wounds*—accidental or surgical—increase the susceptibility to the peculiar poison. (3) *Women in childbed*, for the same reason as (2); but care must be exercised, lest this class be confounded with septic affections. (4) *Season.*—The autum and winter months furnish the most cases.

Immunity.—Single attacks during the life of a person form a rule to which there are rather frequent exceptions.

Clinical History.—**The incubation period** is extremely brief, lasting usually from two to three or four days. It may rarely, however, be longer—five to eight days—or more rarely still shorter—less than twenty-four hours.

The **invasion** of scarlet fever is generally quite *sudden* and, as a rule, active. The child feels uncomfortable, looks stupid, complains of *sore throat* and decided nausea, and in the great majority of the cases *vomits.* The tongue is furred. If he be very young, *nervous symptoms* are prominent, and he may exhibit convulsions. The *pulse*, which is a strong diagnostic factor, is rapid and hard, reaching 140 to 160 at the very onset. The *temperature* rises quickly to 104° or 105° F. (40.5° C.), and remains high.

Eruption.—Within the first twenty-four or thirty-six hours the characteristic rash appears, and is, as a rule, first seen on the neck; there is no certainty about this, however, as it may first come out on the abdomen or back of the hands or on the thighs, and not be seen on any other part of the body. Frequently it is found on the dependent portions of the trunk. At first it is slight, but perfectly characteristic, and usually takes two days to mature. In mild cases it disappears within thirty-six to forty-eight hours, and at no time is more than a very fine rash, but when typical it cannot be mistaken, especially if accompanied by the premonitory symptoms. When seen from a short distance at the end of the first wenty-four hours of its appearance the whole body (except the face) is of a uniform bright scarlet color. If we examine more closely, we find that the eruption consists of a multitude of red points (*puncta*) that correspond to the hair-follicles. These points are surrounded by zones of erythematous redness, which, joining with one another, give a generally diffuse red appearance to the whole skin. Frequently, however, the rash consists of points representing the hair-follicles without the erythema, and in rough skins the rash may be more punctiform—that is, more strictly a condition of "goose skin." Sudamina are quite frequent. Pressure by the finger causes a pallor which at once disappears when the finger is removed. The patient's lips and chin are pale and in striking contrast with the vividly scarlet cheeks. In some cases the rash is patchy, especially on the limbs, and in these cases it may suggest measles, the patches consisting of clusters of fine papules or points with much surrounding erythema, while normal skin is present between the patches. In severe cases the rash may be hemorrhagic in character, minute extravasations of blood taking place in the skin; this may occur even in mild attacks, and not be seen until after death, but more frequently it is seen in malignant cases. Purpuric patches are frequently found after death when even in life they did not appear. There is itching, which may be either moderate or intense throughout the eruptive stage.

The rash is succeeded by a *desquamation* that will be extensive or slight according to the intensity of the fever. In *mild cases* the tonsils, palate, uvula, and pharynx are deeply congested, and the mucosa of the cheeks, palate, and tonsils may show the eruption. In *severer forms* the tonsils are red and inflamed, and covered with tenacious secretions, while minute yellow points corresponding to the tonsillar crypts are usually prominent. (*Vide* Malignant Scarlatina.) The nasal chambers are swollen, producing a free discharge, and the deeper cervical glands at the angle of the jaw are frequently enlarged. The *tongue* is coated with a thick, dense white fur (dead epithelium), and frequently shows a dry, glazed central band. In a few days the dead epithelium is cast off, clearing the tongue, when we have a red, clean, glazed tongue with greatly enlarged fungiform papillæ, giving us the *strawberry tongue* of classical history. The eyes are frequently swollen and the conjunctivæ injected.

Sleeplessness and mild delirium often mark a typical case, suggesting a congested state of the meninges, but it is neither usual for the child to be violent nor for the delirium to continue long.

The *pulse* is usually a strong diagnostic feature, and is always hard, quick, and wiry, varying from 140 to 160; it is out of proportion to the temperature and the general condition of the child. *Leukocytosis* is commonly noted. The *temperature* in average cases reaches 104° or 105° F. (40.5° C.), and in severe forms it may touch 106° F. (41.1° C.), the nocturnal remissions being slight and defervescence gradual (*vide* Fig. 20). The *urine* is scanty, thick, and contains urates, with a small quantity of albumin.

Within one week, if no complications have occurred, the attack will have reached its height and the symptoms have begun to decline. The rash gradually fades, temperature falls, the tongue is less red, the throat less injected, and the child seems more natural. If at the end of one week the fever continues, it suggests the many possible complications, the most frequent of which is a throat or tonsillar ulceration, inflammation of the cervical glands, otitis, or, most probably, acute nephritis. It must be well understood that no two cases of scarlet fever are alike.

Clinical Types.—**Mild Scarlet Fever.**—In very many cases of scarlet fever all the premonitory symptoms are absent, and the rash is the only indication of the trouble. There is neither vomiting nor fever to be recognized, and no tonsillar trouble of any importance, while the rash is neither uniform nor well marked. In these cases we must be very careful not to confound the eruption with urticaria or some of the many medicinal rashes. The most difficult cases of all to diagnose are those in which sore throat is present without a rash, inasmuch as there is nothing characteristic about a scarlatinal tonsillitis.

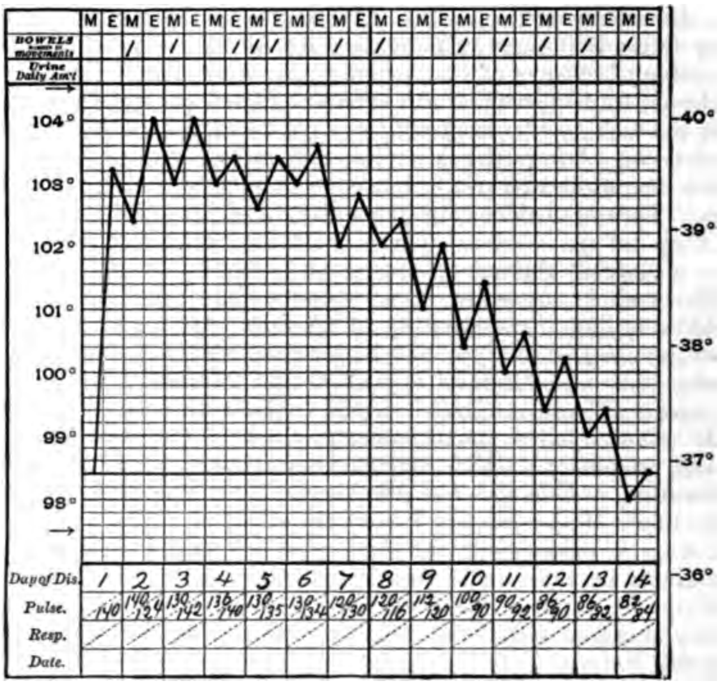

FIG. 20.—Temperature-curve of a case of scarlatina with favorable course—William C——, aged seven years.

During house epidemics when several children are affected it frequently happens that a child has sore throat and the "strawberry tongue" without a development of the rash. This may also occur in adults, and is the so-called *scarlatina sine eruptione*. These very slight cases of the fever are the most to be dreaded, as they may be followed by the most severe attacks of nephritis.

Malignant Scarlet Fever.—Death occurs usually by the end of the first week in severe cases, Drs. Ashby and Wright reporting a death within the first twenty-four hours (*atactic form*). In malignant cases, such as usually occur among the unhygienic and delicate, the tonsils may be covered by a membranous exudate, and the system quickly receive an overwhelming dose of the poison; death then results from septic causes (*anginose form*). In cases in which death occurs early a child soon becomes cyanotic, restless, or more frequently somnolent. In all these cases the temperature remains high—105° to 106° F. (41.1° C.), and very frequently 107° F. (41.6° C.). Diarrhea is frequently a troublesome factor in severe cases; coryza is very abundant; there is much glandular swelling and cellulitis, the neck becoming enormously enlarged and hard, the skin dull and livid in color; the extremities grow cold; the heart gradually becomes irregular, losing a beat, and finally fails.

If life is sustained through such an ordeal, the tonsils slough and the lungs may eventually become the seat of a septic pneumonia. In many desperate cases when life is prolonged to the end of the second or even third week general septicemia is most likely to occur. In this condition the tonsils ulcerate, sloughing patches appear on the fauces, the glands about the neck become enlarged and doughy, and the nasal mucous membrane gives out a purulent secretion in abundance. The temperature may remit, but continues high; the urine is albuminous; pus wells from both ears; and thus the child is gradually consumed and perishes. In all these cases pleuro-pneumonia will be found, together with hemorrhagic kidneys, and most probably small abscesses will be found in the latter at the post-mortem.

A third variety (*hemorrhagic*) shows at first cutaneous petechiæ, which grow rapidly into large ecchymotic patches. Hemorrhages also take place from the mucous surfaces, epistaxis and hematuria being very common. Death, as a rule, follows in two or three days.

Desquamation.—By the end of the first week the rash commences to disappear, the skin is (or soon becomes) mottled, dry, and rough, and gradually the scarf skin begins to separate. This process usually begins about the neck and trunk, and frequently large flakes are detached, the whole cuticle of the hand or foot sometimes coming off in one mass like a glove. The degree and character of the desquamation bear some relation to the severity of the eruption. In some cases the hair and nails have been cast off. In many cases desquamation is prolonged to the eighth week; it is usually longest on the hands and feet.

Complications.—**Otitis.**—The inflammation may extend from the throat along the Eustachian tubes to the middle ear, and pus be formed in the tympanic cavity, making its exit by perforating the membrane. This complication may occur either during the fever or at some time during convalescence. Suppuration in the middle ear is one of the common causes of a continued high temperature after the disappearance of the rash. *Pain* in the ear may not attract our attention to this unfortunate complication; most frequently, however, the child will place its hand on the ear and shake its head, as if to get rid of some source of irritation.

Pyemia.—*Pyemia* and *abscess* of the lungs may follow, and thrombosis of the lateral sinus may occur. The *tonsils* may be the seat of deep ulceration, and the soft palate may slough and show cicatrization of the soft parts of the throat in cases which may yet recover. The *cervical glands* may become enlarged and suppurate, either during the fever or while the child is convalescent. In debilitated or strumous children this complication may be very troublesome, with the formation of deep ragged ulcers, slow to heal, and in rare cases exposing the larger blood-vessels. *Broncho-* or *lobar pneumonia* may occur, and is most usual during the second week, being due to extension downward of the lesion from the throat. Pneumonia followed by *empyema* may also occur during convalescence.

Joint-affections.—(*a*) *Scarlatinal synovitis*, which occurs in 7 per cent. of cases, nearly always appearing from the fourth to the tenth day; in 72 per cent. of cases affecting the wrists (Marsden). Less commonly the small joints of the fingers, the elbows, the ankles, the knees, and soles of the feet may be affected. The trouble is fugitive, and seldom returns to the same joint, and is caused by the scarlatinal poison. (*b*) *Septic arthritis*, met with in severe or fatal cases, is often associated with grave throat symptoms. In these cases the knees may be most severely affected, remaining swollen for weeks, and in unusual cases suppuration may take place and be followed by pyemia. (*c*) *Rheumatic synovitis*, which usually develops during convalescence. Two cases have occurred in my practice, complicated with simple acute endocarditis. Rarely tuberculous invasion of the joints occurs as a *sequel*. Here the scarlatina merely affords a predisposition to tuberculosis.

Nephritis.—No other complication of scarlet fever can equal *nephritis* in importance or interest, this condition always giving rise to anxiety in otherwise mild and hopeful cases. During the height of the fever, as is the case in all exanthemata, there is a trace of albumin in the urine that has no special significance, and it is possible for the kidneys to escape without greater damage than occurs in other acute febrile affections. Independently of this febrile albuminuria, there are two forms of nephritis which it is important to bear in mind, though they have been frequently confounded:

(*a*) *Septic Nephritis.*—In severe forms of scarlet fever, when the throat symptoms include sloughing tonsils, involvement of the soft palate, and general adenitis, the urine quickly becomes loaded with albumin, but shows scarcely any blood and but few casts. No renal symptoms will be recognized, and if present they may be masked by the general condition of septicemia. There may be neither dropsy nor uremic phenomena, but the patient usually dies by the end of the second week, when a typical pyemic kidney is found containing minute abscesses. This condition of the kidney is only one part of the general pyemia, and merely illustrates the fact that this organ suffers during the course of the general inflammation.

(*b*) *Post-scarlatinal nephritis* is the form most likely to occur about the third or fourth week, and is the one generally known as scarlatinal nephritis. The kidneys are undoubtedly involved in an active sympathetic inflammation, and at the end of the fever, more than at the beginning, are engaged in carrying off waste products of the fever itself. From the nature of the disease they are in an irritable condition and prone to take on inflammatory changes, just as the bronchial tubes and the lungs are left in a very susceptible condition following measles and whooping-cough. In this way the uriniferous tubules become choked up by the desquamation that is going on inside. The *number of cases* that suffer from post-scarlatinal nephritis varies according to social conditions, the nature of the epidemic, the season of the year, the nature of the treatment received during the disease, and especially the care received throughout convalescence. Ashby and Wright fix the rate of those who suffer at 6 per cent. of hospital cases, but this is, undoubtedly, too high, since hospital cases receive better care during convalescence than private cases. The usual *time* for this form of nephritis to occur is from the end of the second up to the fourth week, but it usually begins very insidiously. Traces of albumin may be found for a few days before the blood and larger quantities of albumin occur, but it is often impossible to date the commencement of an attack. Usually after the fever has subsided the patient for a few days feels well, but very suddenly grows restless, is feverish at night, is thirsty, has a quick, hard pulse, and passes a small quantity of dark-colored urine. If care has been exercised, it will be found that the urine has been gradually diminishing for several days, and a slight puffiness about the face frequently announces the beginning of the trouble. Later the face becomes pale and puffy, while there may be edema of the feet and scrotum, and some vomiting. Under favorable treatment improvement may take place, large quantities of urine may be passed, and the child resume convalescence. The nephritic symptoms may, however, deepen until *uremia* appears, the pulse becoming slow, the temperature subnormal, and the tongue dry and brown. Vomiting is now a frequent occurrence; diarrhea is not unusual; nose-bleed and hemorrhages from the various mucous surfaces, and muscular twitchings may be noted, and most likely the end may be reached in a general convulsion. In all cases of nephritis great care must be taken to recognize heart-changes, and fatal results are more frequent from *cardiac failure* than from the uremic convulsions. The constant effect of nephritis is to raise the blood-tension, and this continued tension in the blood-vessels is followed by dilatation of the heart. Another not unusual result is *endocarditis* or *pericarditis*, with possible embolism.

Sudden death frequently occurs during the course of nephritis. The child may be doing well, possibly sitting up in bed and playing with its toys, when an attack of dyspnea occurs; the face becomes livid, the pulse disappears, and death quickly takes place. Death in such cases is usually said to be due to edema of the lungs: the *dilated heart*, however, has been overlooked, and, while edema of the lungs is present, it is only secondary to the cardiac failure.

It is not unusual for a false membrane to form upon the *larynx*. This is not infrequently due to the streptococcus pyogenes, but the Klebs-Löffler bacillus is oftener found (Ranke found it in more than half of 92 cases). A bacterial examination should always be made early in the disease, and if the *diphtherial* nature of the infection has been determined the serum-treatment should be employed at once.

Diagnosis.—A typical form of scarlet fever offers few difficulties in diagnosis. The period of incubation is short in comparison with that of any of the other exanthemata, particularly variola, measles, and varicella. The vomiting, which is almost a constant factor in the early stage, associated with high fever, would also exclude the other eruptive diseases. The pulse in itself is most strongly diagnostic, being quick, hard, and wiry, striking the finger at the rate of 140 to 180 per minute;

no other disease has a similar pulse. The early sore throat and the intense hyperemia of the whole mucous membrane, associated with severe constitutional symptoms, make it easy to differentiate from measles, varicella, and variola. The punctate erythematous lesions that appear during the stage of efflorescence of scarlet fever are not found in any of the other eruptive diseases. (*Vide* table on page 250.)

The **differential diagnosis** embraces the discrimination of those rashes that follow the use of certain drugs (quinin, belladonna, potassium bromid and iodid, chloral, etc.). The characteristic invasion-symptoms (vomiting, angina, etc.) of scarlatina are absent; also the high fever and frequent, hard pulse of the latter disease. *Drug-rashes* are seldom so vivid or diffuse as the eruption of scarlatina.

SCARLATINA.	ACUTE EXFOLIATING DERMATITIS.
Onset is sudden, with vomiting, angina, fever, and frequent, hard pulse.	Sudden, with fever only.
Eruption appears first on neck, face, and chest, soon becoming diffuse.	Appears first on trunk.
Duration, three or four days.	Duration, five or six days.
Desquamation begins after eruption has faded, often one week later.	Desquamation begins earlier, often before eruption has faded, and involves the hair and nails.
Ear and throat complications common.	Absent.
Nephritis is a common sequel.	Not so.
Relapses exceptional.	Relapses common.

The **prognosis** in regular, uncomplicated scarlet fever is in almost every case favorable, and, unless the treatment is unusually indifferent, the patient will recover. Severe types, however, and especially malignant scarlatina, are very fatal. Complications arise that will most seriously endanger life.

The **treatment** of scarlet fever is that of the symptoms, together with an attempt at arresting the complications.

Prophylaxis.—The patient should be strictly *quarantined* in an upper room for at least eight weeks or until desquamation has been completed. A competent nurse should be put in charge, and, whether a member of the family or otherwise, she should wear a washable dress, and should not mingle with the family, except her clothing be changed or thoroughly disinfected. The room is to be stripped of all superfluous hangings and furniture. *Inunctions* are required as soon as desquamation commences, with a view to preventing the diffusion of the dried epidermal scales; and the best preparation for this purpose consists of cosmolin, menthol, and carbolic acid, ten grains each of the latter to one ounce of cosmolin, after the plan of J. Lewis Smith. Carbolized water, 1 : 40 (thoroughly shaken), may be used to sponge the surface and may be agreeably followed by cocoa-butter.

The *disinfection* of the physician himself, I am sorry to state, is frequently neglected. He should generate chlorin gas by the following simple method, and allow it to permeate his clothes thoroughly before going into other families: A dram of powdered potassium is placed in a saucer, and a small quantity of hydrochloric acid added. The dish is then placed on the floor, and the physician stands over the vapor chlorid as it arises until it penetrates all his clothing. This, with the free use of the whisk and thorough hand-washing, renders him non-contagious and safe in entering any home or sick-room. Perhaps a less disagreeable method is to have in the patient's house a linen duster or surgeon's apron that has been dipped in a bichlorid solution and allowed to dry. This is slipped over the clothing before entering the sick-room, and is removed after leaving.

In the room, if the case be a severe one involving the throat, I keep the gas or an alcohol lamp burning under a small dish of water, so that steam is constantly generated. To the boiling water I frequently add carbolic acid or oil of eucalyptus; this saturates the room very pleasantly, and at the same time, I believe, limits the extent of the contagion.

General Management.—The sick-room should be large and well ventilated, and should be kept at a uniform temperature (68° to 70° F.—21.1° C.). A light flannel night-dress should be worn by the child, and the bed-clothing should be light as well. The *diet* should consist of milk, broths, egg-white, and fruit-juices, and after the temperature has declined soft diet may be allowed. A few days later the return to ordinary solid foods may be complete.

The evidences of heart-enfeeblement often arise and call for the judicious use of stimulants. It is to be pointed out that this class of agents is remarkably well borne in this affection, and hence may be freely administered. To a child of four years I give one dram (4.0) of brandy or whiskey every second hour, and often increase the dose as required. The preparations of ammonium, particularly the carbonate and the aromatic spirits, have also been warmly recommended as stimulants in this affection. They should be administered in milk as the vehicle to prevent gastric irritation.

Special Treatment.—*Bathing* is recommended in scarlet fever by the best writers, yet often in such an indefinite and uncertain manner as to give the busy practitioner neither any encouragement to resort to it nor any guidance in the matter of technique and mode of procedure. In the classical work of Thomas Watson, now over fifty years old, he hints in his treatment of scarlet fever "that, if the heat on the surface be very great and distressing, he should not recommend the cold effusion, but cold or tepid sponging would be very refreshing and beneficial." This sentiment finds its echo in most works on practical medicine at the present day. Unfortunately, the majority of medical practitioners do not give their instructions for the thorough sponging of their scarlet-fever cases, chiefly through fear of objections from the family.

The physician must quietly but firmly insist upon the patient being most thoroughly sponged three or four times daily, according to the severity of the individual case, using carbolized water (1:60), mercuric chlorid (1 : 8000), salt water, or alcohol and water, at a temperature of 70°-100° F. (21.1°-37.7° C.). Systematic bathing in this manner and inunctions as above described protect the body from certain disastrous complications and sequelæ. The ice-cap may be combined with cool spongings. In extreme cases, with marked nervous symptoms and high temperature, the cold pack, with cold affusions applied to the head and nape of the neck, may be cautiously employed, and a description of the method of giving a cold pack may be found under the treatment of Typhoid Fever. The gradually cooled bath may be substituted if open objection is made by the parents to the cold pack.

In regard to the use of *internal antipyretics*, I prefer phenacetin for older children, combined with quinin in capsules. Acetanilid is better for younger children, and I generally give one-third as many grains as there are years in the child's life. When medicine can be exhibited in the form of capsules, I always prefer to combine it with quinin or strychnin to overcome the tendency to depression. Phenacetin and acetanilid act successfully in controlling the nervous element, relieving headache and fever, promoting diaphoresis, and inducing refreshing sleep. Acetanilid is much more prompt in its action than phenacetin, but its effects are not so lasting. I therefore choose it for young children, and exhibit it in small doses in the form of a powder, and if the bowels are torpid I combine with the acetanilid small doses of calomel and soda. These agents are rarely required, and are not comparable in their good effects to hydrotherapy.

Internal Antiseptics.—Those remedies that are purely antiseptic, administered internally, have not given proof of their utility as yet. The sulphocarbolates of zinc and of sodium, on account of their breaking up in the system and liberating carbolic acid, cannot be used in a sufficiently large dose to meet with success. The syrup of phenic acid is used by many physicians, but their success does not as yet seem to warrant its being classed as an efficient remedy. Marmorek has used his *antistreptococcic serum* extensively, and, although it does not act as a specific, he claims that it prevents the serious complications and invariably renders the attack mild.

The care of the *nose and throat*, and eventually of the *ears*, will require all the skill of the medical attendant, and by commencing early in the case to give careful and constant attention to these parts we may prevent much trouble and danger later on. The attendant should use a small atomizer filled with warm water containing a solution of sodium bicarbonate (gr. xv–℥j—0.975–32.0). If decided inflammation should occur, a solution of hydrogen peroxid and cold water or glycerin (1 : 5) may be used, and then be followed by an oily preparation, such as liquid albolene containing menthol (a 5 per cent. solution).

If the patient cannot tolerate an atomizer, an application of the antiseptic oil directly to the posterior nasal spaces, by means of an aluminum applicator, may be made. Faithful attention to the removal of the secretion from the nose and throat will prevent accumulation, and thus prevent regurgitation up the Eustachian tube with its associated ear-troubles. In this way diphtheria can be prevented from gaining its full lodgement, and, if it gains ground at all, little trouble is experienced with this dreaded disease. For the appropriate treatment of this complication the reader is referred to the treatment of Diphtheria. If pain in the ear should indicate the extension of the trouble up the Eustachian tube, we must redouble our efforts, even though the desquamation within the Eustachian tube itself may be quite beyond the reach of our detergent wash.

The external auditory canal may also become blocked by desquamating epithelium, and this must be removed by gentle sponging. If the tension of the ear-drum becomes very great, it must be punctured. The crude method of dropping laudanum and sweet oil in the ear is to be condemned, as it serves as a nidus for a collection of dust and dirt, independent of the rapid accumulation of dead epidermis.

Scarlatinal synovitis I have encountered in but a small proportion of cases, and then it was of a transient character, leaving no damaged heart-valves behind. I am inclined to attribute this fortunate result to the faithful use of daily bathing and inunctions, long continued and at least until after completion of desquamation.

The most constant complication of scarlet fever is *nephritis*. The specific poison of scarlet fever is peculiarly obnoxious to the kidneys, and is largely eliminated through them; and upon this fact hinges the scientific part of the treatment of this disease. The more active we render the skin the less likely will there be danger to the kidney. If the urine is examined throughout the whole course of the disease, we will find in the earlier stage that it grows less in quantity and becomes more laden with the waste of the body, at times being nearly suppressed by mechanical blocking of the uriniferous tubules. If now the skin is not invited to act to its fullest extent, we will soon find our patient reduced to a comatose state. Free bathing has the happy effect of vicariously eliminating the poison, and in this way it removes the undue pressure placed upon the kidneys. (For the treatment of nephritis the reader is referred to the discussion of acute Bright's disease under Diseases of the Kidneys.)

MEASLES.

Definition.—An acute contagious disease, characterized by an initial coryza, general catarrhal symptoms, fever in the earlier stage, followed by a peculiar papular eruption on the face and body.

Pathology.—In uncomplicated measles we have no pathologic lesions. The only post-mortem changes found, as a rule, are those of catarrhal pneumonia and acute nephritis. All the internal organs are gorged with blood, and minute hemorrhages are found on their surfaces, while occasionally croupous pneumonia may be found involving a lobe or small portions of a lobe of the lung.

Etiology.—Measles occurs in epidemics, yet we have frequent sporadic cases in the larger cities. There is an epidemic prevalence in large centers of population every eighteen months or two years, but the different epidemics vary in their extent and fatality. It generally happens that when once the disease enters a home, street, or small court, scarcely any one escapes who has not been protected by a previous attack, those who suffer being for this reason, in nearly all cases, young children. The adults and older children may enjoy immunity in consequence of a prior attack, although this does not always follow. The susceptibility to measles in children, however, is very great. Biedert[1] found that only 14 per cent. of unprotected children escaped. In the Faroe Islands, under similar conditions, only 1 per cent.

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[1] *Jahrbuch. für Kinderheilkunde*, vol. xxiv. p. 94.

escaped (Madsen, Pannum). There is the same experience in schools and hospitals: when a case incubating the disease is admitted, the whole unprotected junior population is attacked. The *epidemics* occur mostly in the fall and winter, yet the season seems to have but little influence.

Bacteriology.—Micrococci, especially *streptococci*, have often been found in the secretions of the respiratory tract, but they have not been proved to be specific.

Canon and Pielicki[1] found in the blood of 14 cases, as well as often in the sputum and nasal and conjunctival secretions, a special bacillus of irregular size, which colored irregularly with methylene-blue. It was decolorized by Gram's method, did not grow on solid media, but did occasionally on bouillon.

Czajkamski[2] described motile bacilli, 2.5 to 5 micromillimeters in length, which did not color by Gram's method. They could not be cultivated on gelatin or agar, but grew on glycerin-agar, bouillon, and blood-serum, and killed mice by producing septicemia. Neither of these observations has been advanced to any firmer position in the etiology of the disease.

Immunity.—One attack of measles does not seem always to exhaust the soil, as in the other exanthemata: one, two, and in several cases families of children, including the parents, have had four attacks in successive years.

Clinical History.—The period of **incubation** is from seven to fourteen days, and in inoculated cases from seven to ten days.

Catarrhal Stage.—The early symptoms are those of a *cold* with some *fever*. The child has marked coryza, watery eyes, sneezes, and has a dry, croupy cough. Frequently the symptoms are those of a *catarrhal laryngitis* and *bronchitis*, the fauces and tonsils being hyperemic, with abundant secretion, and, in addition, an examination of the eyelids reveals a conjunctivitis. The patient may be acutely ill, the *temperature* rising several degrees in the evening, and falling slightly in the morning; the fever continues high until the rash is fully developed. The *rash*, consisting of one or more distinct *papules*, may be seen on the hard palate fully twenty-four hours before it appears on the face.

The eruptive stage is very characteristic, and usually makes its appearance at the end of the *fourth* day. The neck, face, forehead, and trunk receive the eruption in the order of mention. The whole physiognomy of the child is so characteristically altered that a well-marked case may be diagnosticated at a glance. The face is flushed; the eyes are red and watery; a short, dry cough, frequently metallic in ring, is present; and the nose and cheeks are covered with crops of dusky-red papules surrounded by a zone of erythema which sharply contrasts with the normal skin between the patches. The rash on the face is both *discrete* and *confluent*, or may be arranged at times in small crescents, and in the course of a day or two the whole trunk is invaded, but in a slighter degree. By the fifth, and seldom the sixth day, the eruption has reached its height, and commences to fade, first on the face and neck, then on the body and limbs, followed by a *fine desquamation.*

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[1] *Berliner klinische Wochenschrift*, 1892, S. 377.

[2] *Centralblatt für Bacteriologie*, vol. xviii. Nos. 17 and 18.

By the seventh or eighth day the rash is nearly gone, leaving a blue, mottled stain over the body. The temperature, which has reached 103° F. (39.4° C.) or even 105° F. (40.5° C.), falls when the rash is fully established—*i. e.* on the fifth or sixth day—while the headache, the severe bronchial cough, and the general features subside with the fever. If the temperature continues high after the rash is out, we may look for some complication, such as severe bronchitis, pneumonia, or acute nephritis (*vide* Fig. 21).

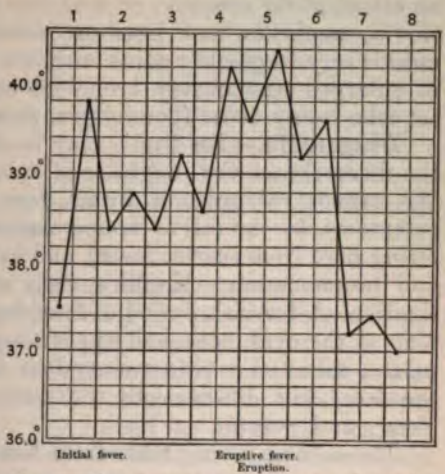

FIG. 21.—Temperature-curve of a case of measles.

An eruption first described by H. Koplik also occurs on the buccal and labial mucous membrane; it appears "as long as twenty-four hours, forty-eight hours, and even three to five days before the appearance of the skin exanthem." It is present before the signs of conjunctivitis appear, and when little or no fever is present. It was found in fifty-two consecutive patients in Koplik's clinic. This eruption consists of small, irregular spots of a bright red color, and in the center of each red spot is the interesting sign which Koplik has described, a minute bluish-white speck. To see the latter requires a strong, glaring daylight, and they must be looked for by everting the mucous membrane of the lips and that of the cheeks. The spots may be few, and again they may be quite numerous. By recognizing this sign, measles patients may be early quarantined and institutional epidemics promptly checked.

Complications.—In some epidemics the character of the disease is very severe, being marked by high fever (105°–106° F.—41.1° C.), a dry, brown tongue, delirium and convulsions, and feeble heart-action, due to the intense hyperemia of all internal organs—lungs, brain, kidneys, etc. I have observed cases in which the eruption was petechial.

The main complications are presented by the lungs. The accompanying bronchitis manifests a strong tendency to extend to the bronchioles, with resulting *broncho-pneumonia.* The extent and seriousness of this complication are largely dependent upon the degree of the previous debility. *Lobar pneumonia* is rarely met with.

Catarrhal or *membranous laryngitis* is frequent in the pre-eruptive stage or as a sequela. Quite rarely edema of the glottis occurs. *Ophthalmia* may occur in anemic and strumous children if strict eye-toilet is not enforced. *Glandular involvement* may take place in the cervical glands. *Otitis* is frequent during desquamation, suppuration taking place in the middle ear and the membrane being perforated. This may be avoided, however, by cleansing the post-nasal spaces frequently during desquamation. *Cancrum oris* and *noma pudendi* may also appear as complications of the disease. *Diarrhea* is frequent at the end of the eruptive period and as a sequela.

The health of the child often remains impaired for a long time after an attack of the measles: it is at this period that whooping-cough, diphtheria, nephritis, and, later on, *acute tuberculosis*, may arise. Tuberculosis very frequently gains entrance into the system from the existence of enlarged and cheesy bronchial and mediastinal glands. Nervous sequelæ rarely occur (hemiplegia, paraplegia).

Diagnosis.—Epidemics may be characterized by irregular forms of the disease, and the diagnosis of sporadic cases is often very difficult. We cannot recognize it by its dermal lesions, but by the prodromal symptoms, by the fall of temperature after the eruption is well out (differing here from scarlet fever), and by the character of the pulse, tongue, and desquamation. Koplik's early sign is usually present and is distinctive. A feverish period of four days, associated with catarrhal symptoms of the eyes, nose, and upper air-passages, a few papules on the hard palate, followed within twenty-four hours by a papular efflorescence on the face, will differentiate the disease from *variola*, *varicella*, *scarlet fever*, and *rubella*.

The accompanying table from Rotch will aid the discrimination:

	MEASLES.	VARIOLA.	VARICELLA.	SCARLET FEVER.	RUBELLA.
Incubation . . .	10 days.	12 days.	17 days.	4 days.	21 days.
Prodromata . .	3 days.	3 days.	A few hours.	2 days.	A few hours.
Efflorescence . .	Papules.	Macules. Papules. Vesicles. Pustules.	Vesicles.	Erythema.	Papules.
Desquamation .	Purpuraceous.	Large crusts.	Small crusts.	Lamellar.	
Complications and sequelæ . . .	Eye and lung.	Larynx. Lungs.		Kidney, ear, and heart.	

The mortality differs according to the surroundings of the patient. In healthy children under favorable environment the mortality is practically *nil*, while in tuberculous and wasted children it is very large, this being especially due to complications and sequelæ. Infants may be born with the rash on them.[1] The disease is quite fatal when it follows other acute infections (*e. g.*, scarlatina).

Treatment.—Measles is a self-limited disease, and we are unable to shorten its duration, nor is there any means of producing immunity from the attack. The treatment is necessarily symptomatic; hence our efforts should be directed to protecting the various organs that are most likely to become involved by complications, remembering at the same time that the nose, ears, eyes, and throat are involved during the feverish stage, and that the skin is in a very susceptible condition.

The patient should be placed in a large dark, well-ventilated room, with a uniform temperature between 68° and 70° F. (21.1° C.). He should remain in bed until the temperature has been normal for one week, and until the efflorescence has nearly faded and the desquamation is almost complete. The diet during the period of fever should be milk, bread, and light soups. Near the end of desquamation, if all symptoms are favorable, a more generous dietary may be allowed.

The bronchial cough, which may be very troublesome during the rst few days, can be readily relieved by some simple expectorant mixure, as—

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[1] *Hom. Med. Chronicle*, May, 1890; *Brit. Med. Journal*, vol. i. p. 612, 1890.

℞. Potassii citrat., ℥ss (16.0);
Succi limonis, ℥j (32.0);
Tr. opii camph., ʒij (8.0);
Syr. ipecac., ʒij (8.0);
Syr. tolu., q. s. ad ℥ij (64.0).—M.
Sig. ʒss–ʒj every two or three hours, according to the age and condition of the patient.

This will serve as a fever mixture as well as an expectorant.

For the coryza I have found that atomizing the nares with some oily vehicle (oleum petrolatum album, etc.) is advantageous.

The skin is in a state of great irritation, and from the very commencement of the disease until the end of desquamation a daily warm bath (95° to 100° F.—35° to 37.7° C.) should be given the patient. The body should be carefully dried and cocoa-butter then thoroughly rubbed over the entire surface. The child should live in an equable temperature for at least three weeks, and longer if desquamation has not then ceased. For months he should be protected from sudden atmospheric changes in order to avoid general respiratory troubles. If he be predisposed to tuberculosis, cod-liver oil and creasote should be prescribed for a period of two months or more.

RUBELLA.

(*Rötheln; Rubeola Notha; German Measles; French Measles.*)

Definition.—An acute contagious disease. It has no prodromal stage, and is characterized by slight fever, coryza, and an efflorescence upon the skin.

Etiology.—Rubella was not distinguished from measles and scarlet fever until about the middle of the eighteenth century. Since then considerable controversy has arisen at different times as to its nature, the theory being at one time strongly advanced that it was a combination of these two diseases, as many of the milder cases have symptoms common to both. That there is a difference, however, in the character and course of these diseases has been proved beyond doubt to careful observers by the facts that rubella occurs independently of either measles or scarlet fever; that contagion from this disease produces a similar disease; that one attack affords immunity to subsequent seizures (although those who have had other eruptive diseases are as liable as those that have not to contract this disease during an epidemic); and that its onset and clinical course are characteristic.

Rubella is contagious, and may occur epidemically or sporadically. It is like measles and the other exanthemata in its being of undoubted microbic origin, although, as is the case with them, the specific organism has not been isolated. When that is accomplished the diagnosis will be more easy and certain.

In hospitals or where persons are crowded and living under unhygienic circumstances the disease is very contagious and the epidemic will be quite general; but in family practice it is but slightly so, and the epidemics are limited, often being confined to a single household and attacking perhaps but one or two of the family. As stated by Edwards, it is spread by the cutaneous exhalations, breath, fomites, and clothing, and is probably contagious from the period of incubation until far into convalescence.

Clinical History.—The **incubation** stage lasts from ten to twelve days, though this period may vary and the disease appear three or four days after exposure. On the other hand, cases have been reported in which it was as long as three weeks. As a rule, the period of incubation is longer perhaps than in measles. The **stage of invasion** covers from one to three days, but in mild cases the *rash* is very often the first indication we have that the child has developed an infectious disease.

For a period of a few days before the rash appears there will be noticed chilliness, pains in different parts of the body, a dull, heavy feeling, perhaps feverishness, accompanied by sore throat, enlarged tonsils, coryza, and suffusion of the eyes, constriction over the chest, and a dry cough and bronchitis. Enlargement and induration of the cervical and other lymphatic glands, together with the sore throat, are common symptoms.

Just before, or with the appearance of, the rash there is a rise in *temperature* to 99° or 100° F. (37.7° C.), or in severe cases as high as 103° F. (39.4° C.) or more. Again, the invasion symptoms may be absent or so mild as to escape notice, and the first sign of infection be the appearance of a rash which first shows itself on the face and extends downward over the body. In some cases the eruption does not follow the regular course, and is confined to one part of the body, and cases have been reported in which it only appeared on the roof of the mouth or on the tonsils. In other cases every part of the body, including the palms of the hands and the soles of the feet, may be covered.

The eruption consists of *papules*, is multiform, confluent, and of a pale or rosy-red color. The patches do not assume any regular shape or form, and the skin between them may become hyperemic and cause itching. The rash reaches its height on different parts of the body in succession, fading in one part while appearing in another. Its duration is from two to five days, and possibly longer in some cases.

A slight *desquamation* usually occurs, and a slight pigmentation of brownish color after the rash fades is frequently noticed, disappearing after a few days. The temperature-curve is variable, but as a rule it remains between 100° F. (37.7° C.) and 102° F. (38.8° C.) while the eruption is present. As mentioned above, sore throat is nearly always present, with enlarged tonsils, a dry cough, and bronchitis. The glandular enlargement will also continue with the rash, and in severe cases the axillary and inguinal glands may become involved. The *pulse* varies with the temperature and respiration. Vomiting has been noticed as occurring during the eruption in severe cases.

After a period varying from three days to a week, with the disappearance of the rash, convalescence begins and the child rapidly regains its former health, and the whole course of the disease may be so mild that the patient cannot be persuaded to remain in bed.

Complications.—The most common are affections of the *respiratory tract* (pneumonia or severe bronchitis), and in some cases we have a *gastro-intestinal catarrh* of a troublesome character. *Diphtheria* or other contagious diseases may occur. A relapse is not uncommon, and may be as severe as the initial attack.

Diagnosis.—Rubella may be distinguished from *measles* by its less severe onset and course, by the lighter color and more diffuse character of its rash, and by the irregular shape which the patches assume. The presence or absence of an epidemic is an important factor in the diagnosis, and in cases occurring when there is no epidemic the diagnosis between this disease and measles of a mild type is difficult if not altogether impossible.

From a well-marked case of *scarlatina* the diagnosis offers no difficulty. The absence of its initial vomiting, the strawberry tongue, the character of the rash (which in scarlet fever is erythematous), and the shorter duration and milder course of rubella, all help to render the diagnosis easy.

RUBELLA.	ERYTHEMA.	URTICARIA.
Occurs first on the face.	On the hands and feet.	In wheals on arms and legs.
Marked coryza present.	No coryza.	No coryza.
At first no itching.	Burning pain.	Intense itching.
Contagious.	Not contagious.	Not contagious.
Microbic origin.	Reflex origin.	Gastric origin.

The **prognosis** in uncomplicated cases is invariably good, but when the surroundings are unhygienic, or in cases in which the child has been delicate previously, it is more serious. Complications, especially pneumonia or diphtheria, may prove fatal, and in some cases the mortality reported has been as high as 9 per cent.

Treatment.—The treatment is simple and principally symptomatic. A mild cough-mixture, such as is recommended in measles for the bronchitis, nutritious but easily digested food, and medicine to regulate the bowels when necessary, fulfil all the indications for internal medication. As in measles, cool sponging should be resorted to before and during the rash; and, when the fever is high, a cool tub-bath, where practicable, will be found to reduce the temperature, quiet the patient, and hasten the appearance of the eruption. During convalesence, if the child does not rapidly regain his appetite and strength, tonics, such as tincture of nux vomica and syrup of hydriodic acid, are indicated.

The complications are to be treated as they arise, but the sponging should not be discontinued until the temperature reaches its normal level.

WHOOPING-COUGH.

(*Pertussis; Tussis Convulsiva; Keuchhusten.*)

Definition.—Whooping-cough is a highly contagious disease which is characterized by a catarrhal inflammation of the respiratory tract, associated with a peculiar spasmodic cough, ending in a whooping inspiration.

Pathology.—There is no lesion that can be considered characteristic of whooping-cough, and there is no distinct causal lesion around which all the symptoms and complicating lesions are grouped. In the beginning there is catarrh of the naso-pharynx, and this may be the only lesion coincident with the development of the characteristic cough. In advancing cases this naso-pharyngeal catarrh becomes generalized by extension to the lachrymal ducts, the conjunctivæ, the Eustachian tube and the middle ear, to the glottis, trachea, large and small bronchi, and the air-vesicles. The more decided pulmonary lesions—emphysema, pulmonary collapse, pulmonary congestion and edema, and broncho-pneumonia—are advanced pathologic conditions accompanying the later stages or more intense forms of the disease (W. W. Johnston).

The *post-mortem* table does not give us much information as to the pathology except as to the sequences of the disease. In the early stages swelling and redness of the respiratory and digestive tracts will be found, together with a large quantity of viscid mucus.

Etiology.—The disease occurs in *epidemics*, yet occasionally may appear *sporadically*. Pertussis seems to have a tendency to occur in epidemics every two years, although in large cities the disease is generally endemic. There is no doubt that it should be classed with the specific diseases, yet for a long time, like mumps, it hovered between the specific and the catarrhal diseases for a home. Pertussis is directly contagious, though scarcely so in houses and school-rooms unless it be for those of a specially susceptible nature. It is possible, however, for the disease to be propagated in schools, though not to the same extent as measles and scarlet fever. It seems that a more decided and prolonged personal contact must be made, as with members of a family, to ensure transmission. One attack practically protects the child, yet exceptions to this rule may be found. The influence of the *seasons* does not seem to have any effect, though perhaps fall and spring are the more frequent periods; the station in life, whether hygienic or unhygienic, does not modify the disease. *Bad ventilation*, however, may propagate the disorder, and cause additional cases by favoring the increase of germs in the immediate surroundings. The *previous condition of health*, especially of the respiratory mucous membrane, seems to possess some predisposing influence, weak, delicate children with an irritable digestive tube associated with a catarrhal state of the respiratory passages, more readily contracting whooping-cough than those in robust health.

There seems to be an intimate association between whooping-cough and *measles*, and it is a well-recognized fact that an epidemic of measles will be followed by whooping-cough in the same sufferers. This is possibly due to the sensitive condition of the mucous membrane left by the measles, which is so favorable to the lodgement of the germs of pertusis; and the association of the two diseases must be more than accidental. There exists a certain individual susceptibility to whooping-cough, as well as to other infectious diseases, and yet many children never contract them, though frequently exposed.

Age exercises some influence on the development of whooping-cough, most cases occurring before the tenth year; after this time the frequency of the disease rapidly diminishes. West states that one-half of all cases develop under three years, but he must have based his knowledge upon an experience in hospitals and children's homes, as the experience of others does not sustain his statement. The disease occurs in adults but rarely, this being due partly to the fact that so many have suffered from it while young, and partly because of a lessening of the susceptibility with advancing years. It occurs frequently before the first year, and when it does it is the most fatal of all the diseases of childhood (Goodhart).

The *sexes* are about equally divided as regards susceptibility; many writers, however, seem to think that girls are most liable. Ofttimes one close exposure in a susceptible child is sufficient to ensure an attack. The germs seem to be located at first in the secretions of the respiratory tract, and are thus disseminated through the air, the disease being most highly contagious, therefore, during the paroxysms of coughing. Goodhart reports a case in which a third party was the medium in conveying the disease from one child to another, thus suggesting a possibility of the contagion being ponderable.

The highway of the contagion of whooping-cough into the system is evidently through the respiratory tract, though this fact has not yet been definitely settled. Published cases of pertussis in the new-born would even seem to make its transmission possible through the fetal circulation, yet the reports are neither numerous nor satisfactory, and cannot be depended upon.

Nature and Bacteriology.—The true nature of whooping-cough has been thoroughly discussed, but is not, as yet, fully settled. Many writers claim it to be a simple bronchitis due to "cold" associated with a certain nervous habit or mimicry. The cough is started by the bronchial irritant, and soon tends to become a habit, thus returning again and again, until it dies out in the oblivion engendered by more healthy and regulated discharges of nervous energy (Goodhart). This theory fails to account for the nervous element and the decided paroxysmal character of the cough. It has been held that the disease is a lesion of either the pneumogastric, phrenic, sympathetic, or recurrent laryngeal nerves, or perhaps even of the medulla. If this ground is valid, it is simply a neurosis. Eustace Smith says it is caused by the pressure of the enlarged tracheal and bronchial glands upon the terminal filaments of the pneumogastric nerve. Whatever the direct cause, the highly contagious character of whooping-cough, its appearance in epidemics, its incubating period, and the possible immunity from subsequent attacks seem to prove beyond argument that it should be classed among purely infectious diseases.

Bacteriology.—Linnæus (to quote Dr. J. P. C. Griffith, in the *American Text-book of Diseases of Children*) attributes pertussis to the presence in the nose of larvæ of insects. Letzerich found a micrococcus in the sputum which he believed to be the specific germ, and claimed to have been able to produce the disease in animals by introducing the secretion into the trachea. Diechler-Kurlow claimed that there was always present in the sputum an organism of the nature of a protozoon which possessed ameboid motion. The researches of Afanassieff, in 1887, however, have attracted the most attention. This observer isolated a short bacillus, which he named the *bacillus tussis convulsivæ*, and of which he was able to obtain pure cultures upon various media. Animals inoculated upon the respiratory mucous membrane with these cultures exhibited some of the symptoms of the disease and developed catarrhal conditions of the respiratory tract, with a tendency to bronchopneumonia. These observations have been confirmed by others, and a toxin has also been reported as present in the urine of patients suffering from pertussis which is identical with that produced by Afanassieff's bacillus. Kuoloff believes that the parasite of whooping-cough is a specific micro-organism, a protozoon, and has found uniformly in the fresh sputa of patients ameboid organisms with spheric spores characterized by concentric laminations.[1] Czaplewski and Hensel describe a short bacillus with distinctly-staining rounded ends, and commonly occurring in pairs. It is found in sputum, both free and in pus-cells, increasing as the disease advances. It can be obtained in pure culture; and grows on any ordinary medium except potato. It resembles Koplik's bacillus. The latter is facultative-anaërobic, and is not stained by Gram's method except in pure culture. The organism is not found in the sputum during the prodromal stage.[2] Even though it be admitted as most probable that some micro-organism is the cause of the malady, it is by no means clear how the symptoms are produced or where the principal seat of the infection arises. The careful investigations of Myer-Huni and of von Heroff indicate that the catarrhal inflammation is most pronounced in the mucous membrane of the nose, larynx, and trachea down to the bifurcation, but especially so on the posterior wall of the larynx in the interarytenoid region, the so-called "cough region." Undoubtedly we have in whooping-cough an infectious catarrhal process which affects the mucous membrane controlled by the superior laryngeal nerve, and the value in many cases of purely local treatment indicates that the abode of the germs is in this region, whence the poisonous products of their growth are absorbed.

The nature of the "whoop" has been frequently discussed to show the nervous origin of the disease, yet the infantile larynx is capable of responding to purely neutral stimuli owing to the flexible nature of the young cartilage. If we carry a young sleeping child from a warm room out in the cool air, the same characteristic whoop may be produced, showing that this reasoning cannot be depended upon.

Clinical History.—The period of **incubation** varies from four to fourteen days according to the extent of catarrhal trouble in the child existing at the time. Goodhart gives several authenticated cases in which the incubation ended on the eighth day. In the beginning the symptoms are those of a slight bronchial cough, which has a tendency to be more pronounced during the night. After a few days the cough assumes an inuenzal character, and at the same time it gradually grows metallic in ing and shows a laryngeal type. There is some fever present. This **atarrhal** or **feverish** stage lasts for a week or more, when it is followed y the **paroxysmal stage,** and these stages are divisions of the symptoms worthy of recognition, as the treatment in the first is not applicable to the second. Many authorities speak of a third stage as one of **decline,** which does not sharply occur, but includes the sequence of the disease. The **catarrhal stage** lasts about one week or ten days, during which the child is ill at ease, is feverish, and has a hoarse, dry cough. The symptoms may either be entirely laryngeal at first or bronchial, with a loss of appetite and broken rest at night. Auscultation at this time will reveal a few moist or dry râles in the larger bronchial tubes, but there is very little secretion. The cough seems to be out of proportion to the physical signs. As the catarrhal stage proceeds the cough commences to indicate its character by becoming more noisy, increasing especially at night. The physiognomy of the child commences to change, the face is swollen, the eyes suffused and watery, the under lids swollen and pink in color. This is one of the most decisive indications of the trouble, and may be recognized by a careful observer a few days before the "whoop" begins which stamps the disease and ushers in the second stage. The commencement of the **paroxysmal stage** is quite different from the easy and more constant coughing of the first stage. If the child is in bed, the onset of a paroxysm is usually quite sudden, but if he is up and playing, there is a period of restlessness, a premonition of the coming storm similar to the aura in epilepsy, and the child may even have time to run to his mother or nurse before the paroxysm comes on. Usually the paroxysms are induced by a quick inspiration, as during drinking, eating, or crying. The first (expiratory) part is short, and followed by a short whoop; this is very quickly followed by a long series of short expiratory efforts and a second and longer whoop, when the paroxysm may cease. In some cases a third and a fourth may quickly follow, until the child is quite exhausted. The paroxysms, whether short or long, generally terminate with vomiting or eructation of a quantity of stringy mucus. Food is ejected, and in most cases a little blood is mixed with the vomited mucus.

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[1] *Medical News*, Nov. 9, 1896.

[2] Saunders' *Year-Book* for 1899, p. 696.

At this stage of the disease, if at all severe, the *countenance* of the child is characteristic, and so much so that a mistake is no longer possible: the features are swollen, puffy, and dusky in color; the eyes are injected, the lids swollen and pink; the skin livid, due to a minute ecchymosis of the smaller capillaries. In many cases there will be extravasation of blood beneath the conjunctiva, due to the violence of the congestive cough. If the chest be examined at this stage, it will tell but little, provided we have no broncho-pneumonia, though a few moist râles may be found scattered through the larger tubes.

The spasmodic stage of whooping-cough has no set *duration* and varies frequently in intensity. In severe cases it may consist of twenty to forty paroxysms during the twenty-four hours. Some spasmodic coughs are not accompanied by a whoop, and the absence of this sign may be noted in very young children, as well as in those that are very ill with broncho-pneumonia. Some children vomit after a coughing spell without the whoop.

It is frequently observed that long after the spasmodic spell has come to an end the paroxysms return again and again, perhaps years afterward, with almost characteristic features, evidently acting under the stimulus of some perfectly neutral catarrh.

Complications.—In severe cases the complications are likely to be numerous.

Epistaxis often occurs in children; *hemoptysis* when vomiting is frequent; *ulceration* of the frenum linguæ in violent coughing; *convulsions* in vigorous children; and *broncho-pneumonia*, *pleurisy*, *pericarditis*, *laryngitis*, and *hernia* in severe, prolonged coughing. Convulsions and broncho-pneumonia are alarming; in young children a *profound stupor* takes the place of the convulsions, and the latter then become of graver significance.

Sequelæ.—*Acute nephritis* frequently occurs, and is as severe as that found in scarlet fever, although the condition has not received the recognition it should from the authorities. In a series of over 200 cases I have found the kidneys affected in 20 per cent. *Emaciation* is a very important sequence of pertussis. All the viscera are liable to fatty degeneration, and nutritional changes open the door to cheesy, glandular alterations, and eventually to a secondary tuberculosis. Atelectasis, by curtailing lung-space, frequently brings about a general collapse, and this condition very frequently explains the flattened chest found in young adults. Emaciation may also be due to *mucous disease*, a chronic gastro-intestinal catarrh of long standing.

Prognosis.—Associated with its complications, pertussis is a very fatal disease, especially in children under two years of age. Dolan regards it as third in rank among the fatal diseases of England, where the death-rate per million is five thousand annually. The deaths occur chiefly among children of the poor and in bottle-fed infants.

Goodhart regards whooping-cough as the most fatal of all the diseases in children under one year of age. He places the mortality as high as 12 per cent., and thinks that this is not too high; his statement, however, is hardly warranted, as he includes the deaths from the many sequelæ which we cannot estimate. Ashby and Wright place the mortality at 7.6 per cent.

Diagnosis.—Young infants usually do not "whoop," but cough spasmodically. Children with *pleurisy* or *pneumonia* do not whoop, yet we diagnose whooping-cough by the preceding catarrhal fever. From *influenza* in its early stages it is most difficult to differentiate the affection. The pink under eyelid has to me been the most certain sign. When the whoop appears and during the existence of an epidemic, however, the diagnosis may be rendered certain.

The diagnostic points prior to the whooping stage, enunciated by Eustace Smith (viz. "If a child be made to bend back the head, so that his face becomes almost horizontal, and the eyes look straight upward at the ceiling above, a venous hum, varying in intensity according to the size and position of the diseased glands, is heard with the stethoscope placed upon the upper bone of the sternum. As the chin is now slowly depressed the hum becomes less loudly audible, and ceases shortly before the head reaches its ordinary position") has not been very satisfactory. It is true that we do recognize the hum caused by he enlarged bronchial gland, but it occurs long after other symptoms re manifest, and thus its importance is much lessened.

I have for several years been able to place considerable value on the peculiar puffiness of the mucous membrane of the eyes and the swollen or edematous condition of the whole face and almost dusky color. This condition may exist for days before the catarrhal symptoms have extended throughout the respiratory mucous membrane. The cough at this stage may not be at all suggestive: it may be, in fact, purely bronchial.

This symptom of fulness about the eyes, which is quite as constant as in *measles*, would in fact suggest that disease, and must be differentiated from it. As we are able to diagnose measles by its appearance first on the hard palate, so I contend we may diagnose whooping-cough in its earliest stage by the characteristic swollen condition of the eyes and face. I insist upon this factor as of the greatest importance, as its recognition will enable us to institute specific treatment early, when the disease is yet local and may be brought more speedily under control.

Treatment.—The gravity of pertussis is scarcely appreciated either by the general physician or the public, and there is more criminal neglect in connection with whooping-cough than with any other disease. Medicinal treatment is exceedingly unsatisfactory, although the therapeutic measures are boundless: just as in phthisis and diphtheria, they cover the whole domain of the Pharmacopeia, and we have as yet found no specific. The remedies most in use are the antispasmodics and the germicides.

Whooping-cough has a striking parallel in diphtheria, in that it has in its early stages a local manifestation in its strong tendency to fasten itself upon the throat. How long this period exists we know to a certainty no more than we know just how long diphtheria is purely a local throat-poison; yet there is undoubtedly a period in whooping-cough, as there is in diphtheria, long or short, in which the virus—if it could be recognized—could be destroyed and the disease terminated. To abort cases thus within two weeks is not unusual, and this explains the number of reported cures made by germicidal remedies.

I have notes of 2 recent cases in which the characteristic whoop commenced at once with the general catarrhal symptoms, and was cut short by a hydrogen-peroxid gargle. I think these 2 cases illustrate very clearly the fact that the germs of the disease will locate on the mucous membrane of the respiratory passages and bring about a nerve-discharge which ends in the characteristic whoop. In my treatment of this disease I find the greatest necessity of recognizing the nature of the trouble early in the catarrhal stage. If I can satisfy myself that I am dealing with a case of early pertussis, my methods of procedure are much different from what they would be if the case were well advanced. We must remember that the two stages are not sharply defined, and that many cases entirely lack the catarrhal stage, just as there are many cases that do not whoop.

Pertussis, as we all know, is a notoriously unsatisfactory disease to manage, and if we put our whole confidence on a single remedy, we are likely to meet with keen disappointment. The drugs I have found most efficient in the catarrhal stage have been hydrogen peroxid in sterilizing the naso-pharynx, and asafetida, occasionally used for the paroxysms. Belladonna is to a high degree beneficial in young children. I push this drug until I get the full toxic effects, when I am generally rewarded with a diminution of the suggestive characteristics of the cough.

To be more explicit, I will detail the methods of procedure in a family in which I have instituted my plan of thorough treatment: A child of four years attending kindergarten was brought to me with a suspicious cough. The history was given of an exposure of over two weeks prior. The child had coughed for a few days, more at night than in the daytime; was feverish during the evenings; showed slightly swollen eyelids, thus suggesting the nature of the impending trouble. I ordered hydrogen peroxid and pure glycerin in equal parts, which were well diluted and thoroughly sprayed through the naso-pharynx every four hours. The diet was light and digestible; out-door life was encouraged, except on windy days. All excitement was avoided, so as to avoid the precipitation of any additional paroxysms. At night the child was placed in a large, well-ventilated room, and over its cot was erected a mosquito netting, so as to prevent any unusual draught—a procedure which I have found highly beneficial, as it materially lessens the number of the nocturnal paroxysms. When the cough was fully established and was accompanied by eructations of stringy mucus, I commenced the exhibition of the mixture of asafetida ½ dram (2.0) every two hours. The record of the paroxysmal stage was as follows: The first week averaged six coughing spells per day; the second week averaged ten per day; the third week, four paroxysms; and the fourth and fifth weeks averaged about two paroxysms during the twenty-four hours. When the younger brother, but eight weeks old, commenced to show evidences of the disease, I first used hydrogen peroxid as in the older brother, and immediately followed it with asafetida. This case continued scarcely four weeks when all symptoms subsided.

Mistura asafœtida, however, is at times disappointing, even in younger children. My second choice is the tincture of belladonna, exhibited in doses of one drop for every month of the child's life, the doses being rapidly increased until toxic effects are reached. Then I gradually increase the amount as tolerance of the drug seems to be established. In very young children I have obtained good results from the use of a freshly-prepared belladonna plaster placed between the scapulæ, and the physiologic action of the drug seems thus to be more constantly maintained. The plaster may be changed at the end of one week. In a number of very troublesome cases in young children I have gained a decided advantage by an application of a 2 per cent. cocain solution directly to the naso-pharynx. This treatment, however, does not preclude the use of hydrogen peroxid, which should be continued throughout the catarrhal stage.

Bromoform was resorted to in fully 20 per cent. of my cases, an was a keen disappointment; it seemed merely to stupefy the patien and did not apparently shorten the progress of the disease. The coal tar products, pushed to the toxic limit, modified the disease but slightly Belladonna and antipyrin in combination gave better results than eithe alone. Quinin, chloral, creasote, carbolic acid, I found to be of littl actical use, owing largely to difficulty in administration. This out-ne of the drug-treatment in whooping-cough has reference solely to e catarrhal and paroxysmal stages of the disease. As important ad-uncts to the management of the disorder careful hygiene must be en-orced, and a diet of the simplest character and a uniformly quiet life maintained. Throughout the whole course of the disease out-door life, as far as possible, should be encouraged, and if convenient a sojourn at the sea-shore will shorten the progress of the trouble and limit to a great extent the number of sequelæ.

PAROTITIS.

(*Mumps; Parotiditis; Epidemic Parotitis.*)

Definition.—An acute contagious disease, characterized by an inflammation and swelling of the parotid gland, and occasionally by an involvement of the salivary glands, the testicles, and in the female the mammæ.

Pathology.—Opportunities for post-mortem examinations are rare, leaving in some doubt the pathologic course of the disease; but it probably begins as a catarrhal inflammation of the ducts, involving the periglandular connective tissue. The inflammation is seldom severe enough or of such a nature as to produce suppuration.

Etiology.—Mumps is undoubtedly a constitutional or blood-disease with local manifestations. "It is a question," Goodhart says, "with mumps whether this disease shall be placed with the specific diseases or with those affecting the parts or organs with which the symptoms more particularly concern themselves."

The disease is no doubt of microbic origin, but the specific organism has not yet been isolated, and, while there has been some reason to believe that it is a bacillus, this has not been proved and is still doubtful. It is highly *contagious*, and at times, usually during the spring and autumn, becomes epidemic. It is communicated principally by the *breath* and *exhalations*, the greatest source of contagion being the salivary secretions. It may, however, be carried by a third person or by fomites, and is most liable to be communicated during the beginning of the attack, although the contagiousness continues until after the subsidence of the febrile symptoms. It occurs mostly among *children* and *young adults*, infants and old persons being rarely affected, while males are more liable than females. One attack usually gives immunity from a second attack in the same gland.

Clinical History.—The average period of *incubation* is fourteen days, but it may develop as early as ten or as late as twenty days after exposure. The *invasion* is marked by languor and a temperature from 101° to 103° F. (38.3°–39.4° C.), with possible headache and vomiting; the patient complains of pain at the angle of the jaw, and this is greatly increased if an acid (such as vinegar) is swallowed. With these symptoms is noticed a *pyriform swelling* of the parotid glands, the one on the left side usually appearing first, and the other one soon following. Occasionally cases are seen in which but one gland is involved, or the swelling may begin in both at the same time. This increases gradually until some time between the third and sixth days, involving the other salivary glands and causing marked disfigurement; the swelling fills the depression beneath the ear and extends to the cheek and neck, the most prominent part being just below and pressing outward the lobe of the ear. The *salivary secretions* are generally much increased, though there may be the opposite condition of marked dryness of the mouth. When the swelling has reached its height, pressure on the adjacent tissues causes a disagreeable sensation of tension, and chewing, swallowing, and even speaking, are at times painful and difficult. The skin over the affected part may be of a pale or of a dull-red color. Ringing in the ears and a dulling of the hearing is common. The nervous system may be affected, causing headache and delirium, or a low typhoid state may be present. The *duration* is about one week (six to ten days), after which time the swelling subsides, and by the tenth or twelfth day entirely disappears.

Diagnosis.—The diagnosis is easy, the nature and position of the swelling and the course of the disease being characteristic, while the fact that the tonsils are seldom involved prevents a diagnosis of acute tonsillitis.

Occasionally, however, in the course of *septic infection* or after operations, or owing to the extension of inflammation along the duct from the mouth, the parotid gland becomes the seat of an acute inflammation at first hardly distinguishable from mumps. The existence of a possible source of infection, and the fact that the gland under these circumstances usually undergoes suppuration, should lead to the recognition of the true nature of the case.

Complications and Sequelæ.—Mumps, as a rule, runs a mild course without any serious symptoms, but occasionally complications arise that tax the skill of the physician to the uttermost. The most common of these are *orchitis* in the male, which may be followed by atrophy of the testicle; and *mastitis*, *ovaritis*, or *vulvo-vaginitis* in the female, especially after puberty. These complications appear after the subsidence of the swelling of the glands of the neck, only occasionally developing while the glands are still affected, though cases have been reported in which the disease first manifested itself by involvement of the sexual organs. This complication lengthens the course of the attack and increases the constitutional symptoms, but the rule is complete recovery. *Otitis media* sometimes occurs, and a lesion in the auditory nerve, with more or less deafness (which, unfortunately, may be permanent), has been observed. *Meningitis*, with active brain-symptoms, *facial paralysis*, *convulsions*, *albuminuria*, and *arthritis*, have all been noted in certain cases.

Treatment.—The patient should be kept in a well-ventilated room of even temperature, and in bed if the fever is at all severe, and should be isolated from those who have not had the disease. Either hot or cold applications to the swelling will often give relief, and support to the swollen gland by means of cotton and a bandage is very comforting. Saline laxatives may be given, and aconite or some simple fever-mixture at the beginning of the attack is usually indicated. These simple measures are all that are required in an ordinary case, while complications or unusual conditions must be treated as they arise.

TUBERCULOSIS.

Definition.—A chronic (less frequently acute) infectious disease, caused by the bacillus tuberculosis. This organism produces specific lesions, taking the form either of separate nodular masses or diffuse growths, infiltrating the tissues, while aggregations of these elementary tubercles give rise to large tubercular masses. Tubercles undergo caseation and sclerosis, followed in turn by ulceration (in consequence of secondary pyogenic infection), or, more rarely, calcification.

Historic Note.—Prior to the discovery, in the early part of the nineteenth century, by Bayle and Laennec, of the tuberculous new growth as a distinctive body, this disease had been studied chiefly from a clinical point of view. At this early period the disease was believed to consist chiefly of a suppurative process, and in its observation the physician was unaided by auscultation. Later, the tubercle was recognized as a small rounded nodule without any special histologic characteristics. Villemin in 1865 performed his epoch-making experiments, and the tubercle was no longer distinguished by its anatomic characters alone. Though the theory of the infectious nature of tuberculosis had been previously advanced by Buehl and others, it was first clearly demonstrated by Villemin's beautiful inoculation-experiments upon rabbits and guinea-pigs with particles of tubercular and cheesy substances, producing the characteristic lesions of tuberculosis. It then remained for Koch to discover (in 1881) the specific cause of the most important of all human ills—the tubercle bacillus. So soon as the specificity of the disease was definitely established it became clear that the associated inflammatory processes, that were formerly believed to be primary lesions, were secondary. The important rôle, however, played by the latter, particularly in the production of the general features of the disease, will be shown hereafter.

Geographic Distribution.—Tuberculosis prevails in almost every quarter of the globe, but is more prevalent in certain latitudes than in others. Thus, in general terms, it may be said to prevail more extensively in warm than in cold countries, though it lessens in frequency as we approach either pole. Local conditions, however, exercise a more decisive influence in engendering predisposition than mere geographic position. It is of quite frequent occurrence in all densely populated municipalities, and more especially in the overcrowded sections of the latter; this fact explains why the inhabitants of cities of the North are but little less spared than those of the cities of the South. On the other hand, residents of mountainous countries, owing to the purity of the atmosphere and the elevation, are rarely among its victims. The influence of *race* in predisposing to tuberculosis should also be mentioned here, the South Sea Islanders, the Indians, and the colored race being peculiarly liable.

General Pathology of Tubercular Lesions.—**Distribution of the Lesions in the Body.**—Tuberculous new growths elect, most frequently, the lung, and when the disease occurs in the adult this organ is almost invariably implicated. Next in frequency follow the larynx, intestines, peritoneum, urogenital organs, and the brain. The other chief viscera of the body (spleen, liver, heart, etc., particularly the latter) are less commonly the seat of tuberculosis. In children the lesions exhibit a different distribution, the favorite seats being the lymph-glands, intestines, bones, and joints. In them the distribution corresponds pretty closely, if we except the bronchial and mesenteric glands, to that of surgical tuberculosis.

The Elementary (Nodular) Tubercle.—This may be developed in any tissue to which the tubercle bacillus has found its way, and the presence of the bacillus is its sole distinguishing feature, since the self-same bodies are generated by other micro-organisms—*e. g.* certain of the worms (eggs of the distoma), actinomyces, aspergillus glaucus, and even as a result of irritation by certain foreign bodies (podophyllum). Various forms of pseudo-tuberculosis have been described, but all are due to bacteria that differ from the bacillus tuberculosis. Mallassez and Vignal described a form produced by a micrococcus occurring in a zoöglea, and this observation was later confirmed by Nocard, Eberth, and others. Charrin and Rogers have described still another form, in which they found bacilli about 1μ long, actively motile, and growing freely upon ordinary media, but not growing upon glycerin and agar, and not liquefying gelatin.

The various stages in the development of a tubercle are—

(*a*) *Proliferation* of the fixed-tissue elements (connective tissue, endothelium of the capillaries, etc.) of the part infected, due to the local, specific irritant action of the bacilli. These anatomic products are transformed into epithelioid and giant cells. The epithelioid cells assume various shapes, chiefly rounded and polygonal; they have vesicular nuclei, and soon show tubercle-bacilli in their interiors. A certain proportion of the epithelioid cells, as the result of increase in their size and a repeated division of their nuclei, or by union of contiguous cells, become *giant cells*. The latter occupy the center of the tubercle, and also contain bacilli, the number of giant cells and of the bacilli being largely reciprocal. Thus, the giant cells are numerous in tubercular lymph-glands, joints, etc., in which the bacilli are relatively few; on the other hand, they are scanty in miliary tubercles, in which the bacilli are numerous—two facts supporting the view that giant cells display phagocytic action. Hektoen asserts that the giant cell is a living defensive agent.

(*b*) About the site of infection a *diapedesis of leukocytes* occurs in the nature of a defensive inflammatory process. At first the leukocytes are of the polynuclear variety and are quickly destroyed; but later mononuclear leukocytes (lymphocytes) appear. These latter resist the action of the bacilli, and I think their true function is a phagocytic one. The various forms of cells described are connected and surrounded by a reticular stroma "formed by the fibrillation and rarefaction of the connective-tissue matrix" (Baumgarten).

The fully-developed tubercles are small, nodular bodies whose diameters range from ½ to 2 or 3 mm. At first they are almost transparent, but soon lose this quality in consequence of the further changes described below. They are avascular bodies, and invariably undergo degenerative changes: (*a*) *caseation* and (*b*) *sclerosis*.

(*a*) *Caseation.*—This implies "coagulation-necrosis"—a destructive process proceeding from the center toward the periphery of the tubercle, nd the result of the local action of the bacilli or their toxins. The ells are thus transformed into a uniformly yellowish-gray structureless matter. When the foci are numerous and closely set, fusion may occur, with the production of larger or smaller homogeneous masses (cheesy pneumonia). The latter may soften, resulting in the formation of cavities: this is due, usually, to secondary pyogenic infection, causing ulceration. Less frequently the cheesy masses undergo calcification or become encapsulated. Such masses may remain indefinitely and are practically harmless.

(*b*) *Sclerosis.*—Preceding and during the time that cell-destruction is going on in the center of the tubercles the protective forces of nature are asserting themselves, though too often without avail. In the first place, hyaline transformation, with conversion of the cellular elements into fibrous tissue occurs. Frequently, now, the center of the tubercle is caseous and contains bacilli, while the peripheral parts are quite hard. Here the bacilli are incarcerated (*latent tuberculosis*). The fibroid change may pervade the entire tubercle. Again, the fibroid element in the tissues immediately surrounding the tubercle may be greatly increased and form new connective tissue, and this process be followed by secondary contraction, converting the tubercle into a firm fibrous nodule. The fibroid change in its completest development is observed in tuberculosis of serous membranes.

In every case of tuberculosis there is a battle for supremacy between the destructive forces on the one hand and the resisting, conservative forces on the other hand. As mentioned above, limitation of the tuberculous process takes place by fibrous encapsulation. In the majority of instances, however, the bacilli fall upon a receptive, favorable soil, when nature's benign curative means fail and extension occurs by the appearance of secondary tubercles in adjacent tissues. The dissemination and transportation of the bacilli are effected principally through the lymph-channels and blood-vessels, although to some extent also by the phagocytic leukocytes. Again, infection may occur by actual contact of the affected organ with neighboring parts, the disease spreading by continuity. Lastly, lesions may be propagated by the movement of organs; thus localized peritoneal tuberculosis may rarely become generalized in consequence of the peristaltic movements.

Again, fusion of minute centers of infection or of miliary tubercles results in the formation of larger nodules or areas, which lead by a process of local extension to *diffuse tuberculous infiltration* (gray infiltration of Laennec). An entire lobe may become similarly involved (tuberculous pneumonia), and "there may also be a diffuse infiltration and caseation without any special foci, a widespread tuberculous pneumonia induced by the bacilli" (Osler).

The term "gray infiltration" is misleading from a pathologic point of view, since the morbid changes differ in no essential manner from those described as occurring in the miliary or nodular tubercle. Moreover, the latter also presents a grayish appearance. The apparent difference between a miliary tubercle and diffuse tubercular infiltration lies in the fact that the latter displays a clearer tendency to spread by direct extension.

Associated Inflammatory Processes.—The tubercle bacilli excite associated inflammatory processes in the organs affected, and if the tuberculous lesions run a slow course a limiting wall of true fibroid induration circumscribes the area involved. By means of this induration the natural protective forces, either temporarily or permanently, check the progress of the local lesions, and the change is strictly analogous to the sclerosis that takes place in the peripheral parts of the elementary tubercle or immediately surrounding the latter, as in tuberculosis of serous membranes. On the other hand, when the tuberculous infiltration is less tardily developed the collateral reactive inflammation may show changes similar to those of catarrhal or croupous pneumonia (*vide supra*). It is a noteworthy fact that the constitutional features in tuberculosis are not so much dependent upon the primary as upon a secondary infection, chiefly with the streptococci. The latter are responsible for the serious septic element in certain varieties of tuberculosis (especially pulmonary), and some contend that the tubercle bacilli can excite suppuration directly. The pus, however, in this instance does not contain the streptococci, and is sterile. Mixed infection is, I believe, the rule (*vide* Pathology of Pulmonary Tuberculosis).

Etiology.—The Specific Cause and its Physical Characteristics.—In 1881, Koch discovered the tubercle bacillus, which is the sole cause of the disease. The bacillus is rod-shaped, straight or somewhat bent, and slender, its length equalling about one-third or one-half of the diameter of a red blood-corpuscle (Fig. 22). Its extremities are slightly rounded, it is non-motile, and on the interior of the bacilli small colorless spots can be observed on microscopic examination; these clear spaces in the bacilli represent plasmolysis, and have nothing to do with spore-formation. Spores do not occur.
When stained the bacilli have a somewhat beaded appearance, this being probably due to slight bulgings caused by the presence of spores. The tubercle bacillus is one of the few varieties of bacteria that retain the anilin dye after washings with acids.

Biology.—The bacilli can be grown on culture-media, but not without difficulty, since they demand an even temperature between 98° and 100° F. (37.7° C.), or that of the human body. The best soil is blood-serum previously coagulated by heating. Over the latter may be gently rubbed tuberculous tissue, which is then allowed to remain on the surface. The growth of the bacilli requires about two weeks, when colonies appear as dry, grayish-white or grayish-brown, thin scales or masses on the surface of the culture-medium. From such cultures others may be grown on glycerin-agar or on the potato.

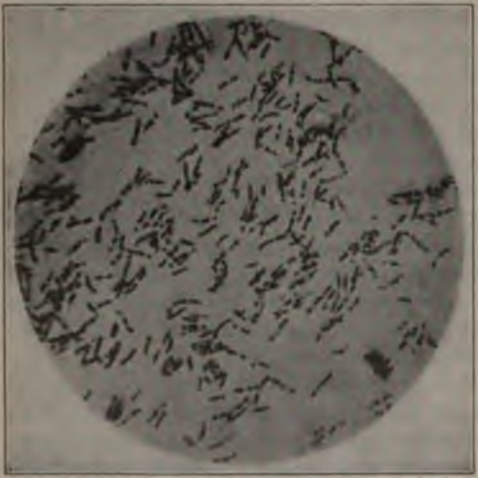

FIG. 22.—Tubercle bacillus in sputum (Fränkel and Pfeiffer).

Inoculations into the guinea-pig and other animals are succeeded in two or three weeks by the appearance of elementary tubercles—first, locally, and then in other organs of the body.

Chemical Products.—The growth of the bacilli is probably attended by the formation of secretory products. Thus an albuminoid substance has been separated, and this when injected into the body of an animal produces slight fever. The albuminoid separated from cultures of tubercle bacilli is a nuclear proteid, and not a specific toxin. There have also been isolated a ptomain and, some contend, an extract which displays pyogenic properties (Koch's tuberculin). The constitutional features of the disease may be ascribed, in part, to the circulation of these poisons in the blood, but principally to the pus-producing organism. Ferran holds that the *bacillus spermogenes* and the *t. bacillus* are identical.

Bovine Tuberculosis.—The disease is common among cattle (10 to 20 per cent.), and Koch first pointed out certain differences between the bovine bacillus and the bacilli of human and animal tuberculosis. Smith's[1] studies show that the bovine bacillus possesses the greater virulence. It is known that the human bacillus infects cattle with difficulty, while "the bovine bacillus infects animals, and probably also man, with great readiness" (MacFarland).[2]

Sources of the Bacilli.—The chief source of the bacilli is the sputum of tuberculous patients. It has been shown that in the advanced stage of pulmonary tuberculosis several billions of bacilli are expectorated daily (Nuttall), and the desiccated sputum is wafted into the atmosphere in the form of dust-like particles containing innumerable bacilli. When the facts that tuberculosis is almost universally prevalent, and that each patient throws off countless millions of bacilli are remembered, it is clear that abundant opportunity is everywhere presented for infection, or, in other words, that secondary sources of infection are numerous and varied.

Distribution of the Bacilli.—The tubercle bacillus is exceedingly tenacious of life, this being its chief distinguishing characteristic. Hence it is found in a viable condition, both (*a*) inside and (*b*) outside of the body.

(*a*) *Inside of the Body.*—As before stated, the number of bacilli found in tuberculous growths varies within wide extremes. In general terms, it may be said that the more rapidly the process advances the greater the number of parasites present. It must not be forgotten, however, that the activity of the tuberculous processes is intimately connected with the degree of resistance offered by the tissues. A chronic tuberculous focus may establish a fistulous connection with a vein or a lymph-vessel, and thus scatter the bacilli to the remotest parts of the body; and in such instances (as the direct effect of the original number of bacilli present) a chronic is quickly converted into an acute form of tuberculosis. The bacilli may also be found in the bodies of non-tuberculous persons. Strauss[3] demonstrated virulent bacilli within the nasal cavities of healthy persons whose positions necessitated their association with, and frequent presence in rooms occupied by, tuberculous patients.

[1] *Trans. of the Assoc. of Amer. Phys.*, 1896, xi., p. 78, and 1898, xiii., p. 417.

[2] *Text-Book upon the Pathogenic Bacteria*, p. 331.

[3] *Münchener medicinische Wochenschrift.*

(*b*) *The Bacilli Outside of the Body.*—Tubercle bacilli can maintain their existence almost indefinitely outside the body. On the other hand, they probably do not develop or multiply under the usual external influences, though, as I have said, their vitality is extraordinary. Their destruction cannot be effected by freezing nor by desiccation, and they survive for months in water. Their power to resist chemical agents (nitric acid, etc.) is also very great, but they may be destroyed by boiling for four or five minutes or by exposure to the direct solar rays. Tubercle bacilli are undoubtedly present in all inhabited places, and it is obvious that they may be conveyed for long distances by means of water, milk, and in many other ways.

The *sputum* dries and flies into the atmosphere in the form of dust, which not only floats in this medium, but also settles upon articles of furniture, the floor, the walls of living-rooms, hospital wards, draperies, clothing, bed-linen, etc.; and from these resting-places it may be conveyed back into the atmosphere. It has been shown, experimentally, that the dust obtained from the walls or from the air of rooms and hospital wards occupied by tuberculous patients is frequently, though not invariably, infected. It is the *in-door atmosphere*, laden with bacilli, that is especially liable to excite tuberculosis when breathed more or less constantly. In places only rarely frequented by consumptives the dust is usually free from virulent bacilli.

Modes of Infection.—(1) **Inhalation of the Bacilli.**—In the vast majority of instances the bacilli are inhaled with the inspired air, but the exhaled breath of tuberculous patients is not infectious. It is the dried sputa floating in the atmosphere that are pathogenic. Klebs and Flügge claim that infection may result from moist particles thrown off in coughing. Occasionally the bacilli attack first the upper respiratory passages (larynx, nose). Usually, however, primary infection takes place in the *smaller bronchi*, or less frequently in the *lungs*; and that these are the points of election is shown by the fact that healed tuberculous lesions in the bronchi and lungs of persons who died of other diseases are frequently met with at post-mortem examinations. Under similar circumstances the *bronchial glands* may be found to present tuberculous lesions. Thus, in 8 out of 30 cases in which both old and recent tuberculous lesions were absent H. P. Loomis found the bronchial glands infective to rabbits.

It is obvious that the bacilli which cause fresh cases come indirectly from other tuberculous subjects, and it has long been supposed that tuberculosis is a contagious affection; unlike small-pox, scarlatina, and other acute contagious diseases, however, tuberculosis is not transmitted by a single contact with a person ill of the disease. On the other hand, Flick and others have shown that persons who come into contact with, or who live in close proximity to, affected persons frequently fall victims to the same affection. ("The latter is as truly contagious as the former, differing only in degree.") We can safely say, therefore, that, though less liable to be transferred by contact than certain other affections, yet on account of the fact that tuberculosis usually pursues a chronic course there is every opportunity for prolonged or repeated contact with resulting infection.

Flick's elaborate topographic study of phthisis in the Fifth Ward of the city of Philadelphia, extending over a period of twenty-five years, shows conclusively that consumption obeys the laws of infectious and contagious diseases. His researches furnish incontestable proof that the tuberculous virus is limited to centers, and that the latter owe their existence to previous cases in the same house or locality; that a house which has had a case of consumption will probably have others within a few years, and may have a very large number of cases in rapid succession; and that approximate houses are considerably exposed to the contagion.

The *contagious theory* of tuberculosis gains support from the fact that husbands have been frequently observed to contract the disease from their wives, and the latter, since they are more constantly confined in the house, to become infected yet more frequently from the former. Weber has observed the case of a tuberculous husband who lost four wives in succession, another who lost three, and four others who lost two each. In like manner, the statistical studies of Cornet, Niven, Baer, and others show that the disease spreads through factories, prisons, cloisters, and even among the physicians, nurses, and attendants in hospitals for the reception of tuberculous patients, producing a mortality-rate from this disease ranging from 45 to 75 per cent. Seventy-three per cent. of nurses up to the age of fifty die of tuberculosis (Whittaker). It is obvious that those who are engaged in making the beds, dusting and sweeping the rooms of patients, are most exposed; and on the other hand, better hygienic living among these classes of individuals, and improved hygienic arrangements in prisons, institutions, and hospitals, have been found to reduce, decidedly, the death-rate from this dread affection. This result is to be accounted for by the following facts: (*a*) There is thus established a greater tissue-resistance to the bacillus tuberculosis on the part of the persons exposed; and (*b*) The germs are thus to a greater extent disseminated. Obviously, then, in institutions in which the proper sanitary precautions are used there may be few if any instances; and from the records of the latter, facts opposed to the contagious theory of the disease can readily be furnished.

(2) **Infection by Swallowing.**—(*a*) That the milk of tuberculous animals contains the bacillus, and that the use of contaminated milk may infect the human subject, are well-established facts.[1] Gerlach and Klebs long since observed the occurrence of the disease in animals fed with milk from cows affected with the so-called "pearl disease." It is not even necessary that the animal infected should have tuberculous mammitis (Ernst), though some are of contrary opinion (Flick, Sidney Martin, and others). The exact frequency of this mode of infection is not known, but there is considerable clinical evidence to support it. Infected animals, especially cows and pigs, that suckle their young very frequently transmit the disease to the latter, the infection usually resulting in intestinal and mesenteric tuberculosis. Hence it is obvious that the bacillus of tuberculosis is, in this instance, swallowed and finds lodgement in the *primæ viæ*. Bang has even shown that butter made from the milk of tuberculous cows may be infectious. Human tuberculosis is entirely analogous, and hence the tuberculous mother is likely to transmit the disease

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[1] See the elaborate statistical studies of Dr. George Cornet: "Die Tuberkulose in den Strafanstalten," *Zeitschrift für Hygiene*, Bd. x., 1891.

to her suckling offspring. This explains, adequately, why abdominal tuberculosis is frequent in children.

(*b*) *The meat of a tuberculous animal* (*e. g.*, cow, pig or fowl) may rarely be infectious, but the bulk of experimental evidence would seem to show that, unless the parts consumed are the seat of tuberculous deposit, infection does not follow. D. H. Bergey,[1] in discussing the public-health relations to tuberculosis, holds that the lower mortality from this disease shown by the Jewish race is ascribable to their careful meat-inspection. Again, the possibility of contamination during the course of preparation for the market, as well as during its transportation, must also be recollected. The experiments of Aufrecht, Chauveau, Klebs, Parrot, Trappeiner, and others show that tuberculosis may be communicated by incorporating with the food the expectoration from tuberculous patients.

(3) **Infection by Inoculation.**—Tuberculosis may be transferred by direct inoculation, as shown originally by Villemin's beautiful experiments upon the eyes of guinea-pigs. Infection may take place, though this is rare, through slight cutaneous lesions (cuts, fissures, excoriations, etc.), but only as the result of accidental inoculation of tuberculous matter. In this manner there is produced a local tuberculosis of the skin, as a rule. Rarely, the contagion is conveyed by the lymphatics to the glands in the vicinity. Persons who follow certain occupations are more or less liable to this mode of infection—*e. g.*, butchers, handlers of hides, dissectors of dead bodies, and, rarely, surgeons. Rare instances of transmission by inoculation occur in divers ways (the bite of a consumptive, a cut from the broken spit-glass of the latter, or even from his pocket-knife, as I have seen in one instance).

The handkerchiefs, body- and bed-linen of the patient may infect by inoculation those who handle or wash them frequently, if they chance to have a fissure or excoriation upon the hand. No doubt *lupus* also arises in the same way. Czerny has reported 2 cases of infection by transplantation of skin; Collings and Murray, 3 cases by tattooing (?). The contact of the lips of tuberculous operators with surgical wounds (as in sucking the latter) may transmit tuberculosis, as in the performance of the rite of circumcision.

Ravenel[2] reports 3 cases of accidental inoculation of the skin in man with the bovine tubercle bacillus, and it is probable that the pathogenic power possessed by it is greater than that of the tubercle bacillus from human sources.

(4) **Direct Hereditary Transmission.**—In exceptional cases the bacillus is found in the fetus *in utero.* In such instances the disease may remain latent, to break forth during childhood or later in life; and though the fetus itself may display no evidence of tuberculosis, the fetal viscera may yet be infective to guinea-pigs (Birch-Hirschfeld). Lehmann[3] has reported an undoubted instance of intra-uterine infection. The tuberculous mother died of tuberculous meningitis three days after the birth of her child, and the child lived twenty-four hours. In its spleen, lungs, and liver were found nodules resembling tubercles

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[1] Saunders' *Year-Book* for 1899.

[2] *Proc. Philada. Path. Soc.*, October, 1900.

[3] *Berlin. klin. Woch.*, July 9, 1895.

and containing tubercle bacilli in large numbers. Galtier has inoculated a pregnant animal with the disease, and found that the offspring was, in consequence, tuberculous at birth. The views of Baumgarten upon this question should be accorded careful consideration. This author believes that the contagion may be transmitted and become pathogenic at a variable period after birth—first, because the affection is very frequent in young children, even during the first months or weeks of life; and, secondly, because certain structures, not apt to be accidentally infected, are commonly the seat of tuberculous lesions in children—the bones and joints. After birth the bacillus may at any time either lose its vitality or take on a luxuriant growth. It is not known, however, in what percentage of these cases the lungs, intestines, peritoneum, and lymph-glands are free from tuberculous lesions. Moreover, Küss disputes the theory of the latency of the tubercle bacilli, and further contends that these latent foci do not exist before the age of three months; that they are rare before the first year, when they mature progressively.

In this place two facts deserve to be emphasized: First, that a child born of tuberculous parents is more receptive than one born of healthy stock; and second, that it is more liable to accidental infection.

The instances of direct transmission that have been traced occurred through tuberculous mothers. The observations of Csokor[1] upon hereditary tuberculosis in cattle also corroborate this dictum; but as the result of carefully conducted experiments by Vignal[2] it is reasonably certain that invasion by heredity is very rare.

(5) Dock and Chadbourne state that mixed modes of infection occur.

(6) Baldwin invites forcible attention to the danger of infection from the unclean hands of tuberculous patients.

Predisposing Causes.—(1) **Race and Nationality.**—The effect of nationality upon the receptivity to tuberculosis can be studied advantageously in America on account of the cosmopolitan character of the population. The tuberculous tendency on the part of Indians of this continent, even in the most favorable climates, is universally acknowledged, and the fact that the negro race is highly receptive to tuberculosis is also well known. Osler[3] gives the following corroborative statistics: "Of the 427 cases of pulmonary tuberculosis at the Johns Hopkins Hospital for the two years ending June 1, 1891, there were 41 cases in the colored—*i. e.* about 1 : 10. The ratio of colored to white of all patients in the wards has been 1 : 7." It is more than twice as common in the African as in the white, and still more prevalent with the Indian (W. L. Rodman). Sears[4] found that in 200 cases of tuberculosis nearly 50 per cent. belonged to the first and second generations of Irish immigrants.

(2) **Hereditary Predisposition.**—The percentage of cases in which heredity can be traced has been variously estimated at from 10 to 40. As before intimated (*vide Direct Hereditary Transmission*), a child

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[1] *Deutsche medizinal Zeitung*, Berlin, Jan. 29, 1892.

[2] *La Semaine médicale*, Paris, Aug. 1, 1892.

[3] *Text-Book of Medicine*, p. 204.

[4] *Boston Medical and Surgical Journal*, April 4, 1895.

reared by tuberculous parents runs great danger of being infected accidentally; and again, a person living in an infected house (with or without the presence of a tuberculous patient) is very liable to become infected, whether his antecedents give a tuberculous history or not. It follows that a correct estimate of the number of cases of phthisis in which hereditary influence plays an etiologic part cannot be obtained. Too much importance has heretofore been attached to the influence of inherited constitutional peculiarities to the exclusion of other potent factors, especially an infective environment. Moreover, a similar degree of predisposition may be acquired as the result of certain debilitating influences (childbirth, defective food-supply, close living- or working-rooms). An inherited tendency to tuberculosis is more unfailingly transmitted through the mother than the father. Multiple appearance is commoner in families with tuberculous parents (Dock and Chadbourne). Children begotten of parents who are drunkards, or who suffer from certain chronic incurable diseases (syphilis, cancer, etc.) at the time of the birth of their children, are liable to inherit a condition of the system that greatly increases morbidity, unless the tendency is overcome by a proper environment, together with systematic physical training during the first years of life. Moreover, persons who have the so-called tuberculous diathesis are frequent sufferers from catarrhal affections, especially of the respiratory organs. The latter condition forms a marked predisposing factor; yet, on the other hand, tuberculosis is met with in persons of robust figure.

The older authors of medical text-books describe two types of conformation—the *tuberculous* and the *scrofulous*. The latter has a heavy figure, thick lips and hands, large, thick bones, and an opaque skin; the former, a light figure, bright eyes, thin skin, oval face, and long, thin bones. The phthisical type of the chest will be referred to in connection with the physical signs of pulmonary tuberculosis. Here emphasis should be given to Cohnheim's view, which is for the greater part correct, to the effect "that the so-called phthisical habit is not an indication of a tendency to, but actually of the existence of, tuberculosis." Whilst the recognition of a pre-tubercular condition has its practical bearing, it must be recollected also that the term implies merely a "delicacy of constitution, incomplete growth, and imperfect development" (Fagge).

(3) **Previous Infectious Diseases.**—That there is no tendency to the transition of other diseases into tuberculosis, as was formerly supposed, cannot now be questioned in view of the undoubted specific nature of the latter disease. Tuberculosis is, however, embraced among the sequelæ of many acute infectious and chronic diseases—influenza, measles, whooping-cough, typhoid fever, cirrhosis of the lungs, and diabetes mellitus (the latter disease involving a predisposition to the former)—for the reason that they render the tissue-soil, especially that of the respiratory tract, more favorable to tubercular infection. Dock and Chadbourne have analyzed 100 cases of adult tuberculosis (bacillary phthisis); it developed rapidly after influenza in 16, and followed pneumonia in 9. It seems proper to mention here the fact that certain other diseases display an antagonistic effect (chronic valvular disease, pulmonary emphysema, etc.). Our knowledge of this subject, however, is as yet quite incomplete.

(4) **Age.**—This affects predisposition decidedly, though tuberculosis may occur at any or all times; and the relation between age and the distribution of the lesions has been previously indicated. Certain forms of tuberculosis are especially frequent in young children (meningeal, mesenteric, and lymphatic).

Pulmonary tuberculosis is most common between *twenty* and *thirty*. It is more rare during early childhood and in the aged, but may appear at any period of life, and the cases that occur in young children are likely to be rapid in their progress.

(5) **Sex.**—Predisposition has but slight relation to sex. Females are, however, somewhat more liable than males, and pregnancy in particular is a disposing factor. Again, when tuberculous females become pregnant the progress of the affection is accelerated, and even more so by the period of lactation. Regarding tuberculosis as being pre-eminently a house-disease, females are more exposed to contagion than males, because they are more closely confined in-doors.

(6) **Climate and Soil.**—Humidity of the soil and abundant atmospheric moisture increase the prevalence of tuberculosis. It is especially common in regions where sudden variations of temperature, or protracted cold with dampness, prevail. This increase is most probably associated with a heightened vulnerability, due to an increased tendency to catarrhal affections of all kinds (Osler). It has been shown that proper drainage of marshy districts has diminished, to some extent, the frequency of this disease (Buchanan), and, on the other hand, mountainous districts are often remarkable for freedom from the disease.

Local Causes.—(1) **Occupation.**—Persons whose employment exposes them to different forms of irritating inhalations are particularly liable. In such, however, there is usually first developed a fibroid induration, and the latter in turn is followed by pulmonary tuberculosis. The continual inhalation of an atmosphere laden with noxious particles, such as is met with in ill-ventilated and overcrowded working or living apartments, renders the tissues more vulnerable.

(2) **Bronchial Catarrh.**—An acute catarrh of the small bronchi prepares the soil for tuberculous infection. Frequently, however, this is the first step in tuberculosis, since the latter disease almost invariably begins as a local catarrhal process, involving the smaller apical bronchi. Here may be pointed out that gastro-intestinal catarrh (of somewhat protracted duration—H. M. King) increases the receptivity for tuberculosis.

(3) **Tubercular Pneumonia.**—In like manner, pulmonary tuberculosis may follow an unresolved pneumonia, but such cases are, as a rule, instances of tuberculous pneumonia primarily.

(4) **Hemoptysis.**—According to some authors, hemoptysis is potent in producing pulmonary tuberculosis. It is, however, certain that in most instances in which it appears to precede phthisis, and have a causal connection with it, it is in reality a symptom of existing pulmonary tuberculosis.

(5) **Pleurisy** may be, though rarely, the starting-point of phthisis. Its predisposing effect may be attributable to compression of the lung, thus interfering with the respiratory excursions, or to the bronchitis which is frequently associated. Pleurisy sometimes initiates fibroid in-

duration, which may then terminate in a tuberculous affection; but the fact is to be emphasized that a very large proportion of the cases of apparently primary pleurisy are tuberculous in nature.

(6) **Intrathoracic Tumor.**—Tuberculosis is often associated with intrathoracic tumors, and especially with aneurysm. Fehde[1] has reported 3 interesting cases of the kind.

(7) **Congenital or acquired contraction of the orifice of the pulmonary artery** predisposes markedly to tuberculosis.

(8) **Trauma.**—Injuries to the chest-wall, with or without laceration of the lung, are frequently followed by pulmonary tuberculosis. The explanation of this association is to be found in the fact that trauma increases largely the susceptibility of the parts injured by diminishing phagocytic activity—the natural power of resistance. It is a familiar observation in surgical practice that after injuries to, or operations on, joints, tuberculosis frequently ensues. Again, operations upon tuberculous lesions are succeeded by general tuberculosis—often acute—in about 8 per cent. of the cases.

TUBERCULOSIS OF THE LYMPH-GLANDS.

(*Scrofula.*)

Scrofula implies tuberculous infection, and scrofulous material inoculated upon susceptible lower animals, especially guinea-pigs and rabbits, invariably causes tuberculosis. The virus is, however, less virulent than that derived from other sources, and this explains the slow progress and often latent character of tuberculosis of the glandular system. A major predisposing factor is *age*, this form of tuberculosis preponderating in children. Hecker, from an examination of the records of the Munich Pathological Institute, found that in 147 cases of tuberculosis among children the lymphatics were affected in 92 per cent.; and it is generally conceded that in young adults tuberculous adenitis is not uncommon, and that it is rarely met with during and after the middle period of life. The lesions generally remain limited to the glands first infected—*i. e.* the cervical, mesenteric, etc., as the case may be—and this for the reason that the natural powers of resistance in the tissues are often able to oppose the march of the destructive forces. Another predisposing condition is an acute or chronic catarrh of the mucous membranes.

The cases are all divisible into two groups: (1) Local tuberculous adenitis, and (2) general tuberculous adenitis.

(1) **Local Tuberculous Adenitis.**—(*a*) **Cervical.**—This is the most frequent form, and is especially common among children. Of 2035 persons examined by Valland, enlarged cervical glands were found between the ages of seven and nine in 96 per cent.; between ten and twelve in 96.1 per cent.; between thirteen and fifteen in 84 per cent.; between sixteen and eighteen in 69.7 per cent.; and between nineteen and twenty-four in 68.3 per cent. Tubercle bacilli were found in the cervical lymph-glands in about 68 per cent. of adults. Negroes are found to be more prone to the affection than whites.

Etiology.—I have stated before that tubercle bacilli are sometimes und on the nasal mucous membrane of healthy persons. The pres- nce of an acute or chronic catarrh of the nasopharynx may now ower the resistance of the tissue-cells, so that the bacilli may gain ccess to the lymph-current, and through the latter to the neighboring glands, setting up tubercular adenitis. Though often the seat of tubercular invasion, the cervical lymph-glands do not furnish a highly favorable soil for the growth and development of the bacilli, and hence the tendency toward latency.

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[1] "Lungentuberculose mit Brusthöhlengeschwulste," *Inaug. Diss.*, Leipzig, 1894.

The *tonsils*, owing to their free communication with the atmosphere, in which there is a wide diffusion of tubercle bacilli, may be primarily infected. But here also, as in the case of other glandular structures, there is a tendency for the affection to become encapsulated, for the reason that the tissue-soil after a prolonged contest generally gains the ascendency over the invading bacilli. The latter may, however, under certain favorable conditions, break down the barriers opposed by nature and effect a lodgement elsewhere, or even become widely diffused through the economy. Thus Kinckmann in 64 autopsies found 25 cases of tuberculosis, in 12 of which the tonsils were affected.

A third mode of infection of the cervical lymph-glands is through the medium of slight injuries and abrasions of the skin or certain forms of skin-eruptions (eczema, etc.). These serve as doors of entrance for the bacilli, which find their way into the neighboring lymph-glands through the lymph-channels. Compared with infection from within, this mode is most probably much less frequent.

Symptoms.—The main feature is a *visible enlargement* of the affected cervical glands, chiefly the submaxillary. At first the glands are too small to be even palpated; later, they can be felt as small, firm tumors underneath the skin. By and by they appear as visible protuberances, ranging in size from that of an English walnut to that of a hen's egg or even larger. The *skin* over the enlarged gland is freely movable, as a rule; less frequently it becomes adherent—an indication of suppuration. When an abscess forms and is allowed to open spontaneously, there remains a chronic discharging sinus. Suppuration is attended with *fever*, *anemia*, and *emaciation*. In well-marked cases the separate tumors coalesce, forming large and irregular masses. The affection is usually bilateral, though almost invariably it is more marked on one side than on the other.

Not infrequently, in addition to the enlargement of the submaxillary, post-cervical, and supraclavicular glands, there is also involvement of the axillary, as was the case in a fatal instance in my own practice. The patient was a male child, eight years of age, who developed pulmonary tuberculosis. It may reasonably be assumed that the bronchial glands also become implicated, and may excite lung tuberculosis.

The *diagnosis* is based upon the history and the associated evidences (keratitis, conjunctivitis, eczema of the scalp or face, nasopharyngeal or bronchial catarrh, etc.), coupled with the glandular enlargement. Bacilli have occasionally been found in the purulent discharge from abscesses. Otis applies the tuberculin-test, and obtains positive reactions in 62 to 69 per cent.

The *course* of this affection is exceedingly slow, often extending over a number of years. Many cases, however, recover after timely surgical intervention. On the other hand, neglected cases are a menace to the life of a patient, since they may be followed by diffusion of the bacilli, with the development of a fatal form of disease.

(*b*) **Bronchial.**—Tuberculosis of the bronchial glands may be primary, or secondary to infection of the lungs, and it is commonly preceded by or associated with bronchial catarrh, which is its chief predisposing cause. The primary form is met with frequently in young children, the mediastinal lymph-glands being affected uniformly in 127 cases at the New York Foundling Hospital (Northrup).

The bronchial and tracheal glands are the receptacles for all foreign substances, including the tubercle bacilli that are not dealt with by the broncho-pulmonary phagocytes. After infection with tubercle bacilli the lymph-glands become swollen, tumefied, and are the seat of caseous change; later they may undergo calcification or proceed to abscess-formation. The latter may rupture either into the lungs, into the trachea or the bronchi, or into a pulmonary blood-vessel.

Symptoms.—If a fistulous communication be established with the air-passages, *cough* and *expectoration* of purulent material, blood, and caseous matter containing bacilli will be noted.

Secondary infection of the lung may occur in this manner. When rupture takes place into a vessel systemic infection promptly follows. Tubercular adenitis involving mediastinal lymph-glands may also lead to infection of the pericardium and then proceed to tuberculous pericarditis.

(*c*) **Mesenteric** (*Tabes Mesenterica*).—This may be primary or secondary, the latter being very common and a secondary infection to intestinal tuberculosis.

The former is rare, however, and the intestinal catarrh with which it is associated is doubtless tuberculous in the vast majority of cases. The mode of infection has already been pointed out. The lesions presented are similar to those met with in tuberculous bronchial glands.

The *symptoms* are not always distinctive, and may be entirely negative during the life of the patient; hence the condition is often incidentally discovered during the post-mortem examination. The *local symptoms* when marked are due in the main to an associated peritonitis. The abdomen is painful and more or less swollen. *Peritoneal effusion* is present, and sometimes sufficient in amount to be detected by the customary physical signs. Large and small *nodules* may sometimes be felt. *Diarrhea* is a marked and obstinate feature and is usually due to tuberculous intestinal ulcers. *Fever* of an intermittent type is almost constantly present, causing emaciation, and the objective changes (pallor of skin, mucous membranes) due to anemia become pronounced. This form of tuberculosis may persist as a local condition, but there is danger of extension to other organs (pleura, lungs). On the other hand, in the adult pulmonary tuberculosis may be followed by involvement of the mesenteric glands without involvement of the intestines, and in such instances there occurs an extension by contiguity along the course of the lymphatics that pass through the diaphragm, and finally, in adults, primary tuberculous new growths may be met with in the mesenteric glands.

Diagnosis.—A probable diagnosis can usually be made if careful attention be paid conjointly to the symptoms, physical signs, and course f the affection. The detection in a child of a tumor which may be oderately hard, doughy, or even fluctuating will aid materially in the iagnosis, and will also afford evidence of tuberculous disease in other rgans.

(2) **General Tuberculous Adenitis.**—This term implies tuberculous disease of the lymph-glands throughout the body, with little if any involvement of other organs; this is a rare condition. The affection may begin as a local tuberculous lymphadenitis, nearly all of the rest of the glands of the body becoming secondarily implicated. The primary seat of the trouble is perhaps most frequently the cervical lymph-glands, though in one instance observed by myself the mesenteric glands first became affected, the case terminating in pleuro-pulmonary tuberculosis.

Symptoms and Diagnosis.—There is *protracted fever*, the temperature being of the remittent or intermittent type. *Wasting* and *debility* are progressive until the patient presents a decidedly puny aspect, while the lymph-glands that are accessible to inspection and palpation are more or less enlarged and manifest a marked tendency to *suppuration*. The affection is usually *chronic*, though very exceptionally it may exhibit an acute course. One of the chief dangers overhanging the sufferer in this affection is that, owing to liberation of the bacilli, the meninges or the lungs may become tuberculous; these cases may also terminate unfavorably from asthenia. Cases in which the glands are but little enlarged, while the *general features* are marked, are puzzling. On the other hand, when the superficial lymph-glands are greatly enlarged the affection may bear a striking resemblance to Hodgkin's disease.

ACUTE TUBERCULOSIS.

This form of tuberculosis is characterized anatomically by the rapid development of miliary tubercles in many and widely-separated parts of the body. In some instances the new growths are pretty evenly distributed through all the organs of the body, manifesting the clinical symptoms of an *acute general infection*. In other instances there is a tendency to centralization of tuberculous growths, as, for example, in the lungs (pulmonary variety) or in the meninges of the brain and spinal cord (meningeal variety).

Pathology.—The fact is to be emphasized that somewhere in the body there is an old tuberculous focus. Apart from this primary lesion, the anatomic changes consist in the widely disseminated miliary tubercles. Their most frequent seats are the lungs, liver, and spleen; less frequently, the marrow of the bones, the heart, the choroid, and the meninges. In some of the organs, particularly the meninges, lungs, etc., the tubercles may be readily perceived by the naked eye, while in others they frequently cannot be detected without the aid of the microscope. It must not be forgotten that in some of the more protracted cases the nodular tubercles may grow into foci of considerable size, ranging from that of a lentil to that of a pea.

Etiology.—This has been, in the main, given in connection with the general etiology of tuberculosis (*vide supra*), though a few special points remain to be adduced. The acute forms of tuberculosis are decidedly more frequent during infancy and childhood than during adult life, and with few exceptions the cases are secondary to a local tuberculous form in one or more lymph-glands (tracheal, bronchial, mesenteric) or in the lungs. More rarely a pre-existing tuberculous focus in the kidneys, the bones, or the skin may give rise to the affection, as may the occurrence of certain other acute infectious diseases—such as measles, whooping-cough, and influenza, in children, and typhoid fever and lobar pneumonia, especially with delayed resolution, in adults.

Modes of Infection.—Most frequently there is established a fistulous connection between the local tuberculous focus and a vein, especially the pulmonary vein. Under these circumstances there may be large numbers of bacilli discharged into the blood-stream; but oftener only small numbers of bacilli enter and subsequently multiply, inducing general infection (Ribbert and Wild[1]). A second mode of infection, though decidedly more rare than the above, is the rupture of a tuberculous focus into the thoracic duct, in which case the tuberculous material passes almost directly into the subclavian vein. In these cases, according to Ponfick, the disease is less rapid in its course.

Clinical History.—That miliary tubercles may exist in many organs of the body (liver, heart, etc.) without giving rise to symptoms is a noteworthy fact. Cohnheim and Manz have discovered miliary tuberculosis of the choroid when the condition was only detectable with the aid of the ophthalmoscope.

The following forms of the disease may be distinguished:

GENERAL MILIARY TUBERCULOSIS.

(a) TYPHOID FORM.

The **symptoms** are those of a general infection of the body, there being in most cases a period of *incubation*, during which the patient complains of malaise, headache, chilliness, feverishness, and increasing debility. Rarely, the onset is comparatively *sudden*. The reaction of the nervous system against the poison, which is now scattered to all parts of the body, is shown by such symptoms as the *fever*, which rapidly increases, a *rapid, feeble pulse*, and *mental dulness* or *delirium*. The *tongue* becomes dry, and sometimes also brown. The *respirations* are accelerated, and there is more or less *cyanosis*, with which symptom is associated a peculiar and *characteristic pallor* of countenance. Coincidently with the febrile exacerbations the *cheeks* may wear a circumscribed blush. Among the rarer early symptoms is epistaxis. The patient soon becomes profoundly prostrated or experiences a feeling of anxiety: if, as sometimes happens, the course is protracted, *weakness*, *anemia*, and especially *emaciation*, are well marked and assume diagnostic importance. These cases sometimes pass into the pulmonary or the meningeal form, the patients often succumbing speedily to such localized developments.

Fever.—The temperature usually pursues a high range, although there are a few cases in which the entire course is afebrile. Again, it occurs not infrequently that the temperature is normal or nearly so for a short period. The usual temperature-curve ranges at first between

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[1] *Deutsche medicinische Wochenschrift*, Dec. 30, 1897.

102° and 104° F. (38.8°–40° C.), and then continues to rise, with the development of the serious general condition in a way exactly similar to that observed in typhoid fever. In many instances the fever is irregularly remitting, at least at intervals, if not so constantly. Thus, periods of irregular fever may alternate with others of continued, and later deeply remittent or distinctly intermittent, fever.

Nervous Symptoms.—In most cases the nervous symptoms are not prominent. In a smaller number headache, vertigo, delirium, and often stupor, become marked at an early stage and may persist. They are due to the general infection.

Circulatory System.—The pulse is small, and its rate is out of proportion to the fever, varying from 100 to 140 or higher. It may become irregular, particularly if the meninges be involved.

Respiratory System.—The breath is somewhat hurried and labored; there is a cough, but it is not annoying as a rule; and there is a slight expectoration, which is not characteristic. If there be present simultaneously in the lungs an old tuberculous focus, the expectoration may be more profuse and typical. The bacilli are also absent from the sputum unless an old tuberculous lesion exist in the lungs.

The physical signs are those of a moderate, diffuse bronchitis, though local signs of consolidation or pleurisy may develop late in the course of the affection. On the other hand, such signs may be evidences of an old tuberculous affection.

Digestive System.—As before noted, there are anorexia and a dry tongue (symptoms due to the systemic infection), while vomiting may occur at the outset and excessive thirst is common. The spleen usually becomes enlarged, though only to a moderate extent.

Ocular Symptoms.—The important symptom presented by the eye is the presence of choroid tubercles, which may be determined by a careful ophthalmoscopic examination. Their absence does not militate against the diagnosis of general miliary tuberculosis, since they may be too few to be detected, or possibly absent altogether. Their demonstration is always exceedingly difficult, and only possible with the skilled ophthalmologist.

Diagnosis.—This form of tuberculosis is often with difficulty discriminated from typhoid fever, but in the following table I have endeavored to contrast points of dissimilarity:

ACUTE GENERAL MILIARY TUBERCULOSIS.	TYPHOID FEVER.
Family history of tuberculosis, or presence of an old focus.	Coexistent with an epidemic or following previous cases of typhoid.
Evolution of the disease not characteristic.	Evolution of the disease is characteristic.
Epistaxis rare.	Epistaxis a common early symptom.
Fever-curve of decidedly irregular type.	Temperature-curve of the continued type.
Pulse rapid, out of proportion to fever.	Pulse often dicrotic; slow in proportion to fever.
Respirations rapid and labored.	Respiration moderately increased.
Face dusky, with peculiar pallor.	No duskiness of face.
Abdominal symptoms are not suggestive.	Abdominal symptoms (stools, enlarged spleen, tympanites, etc.) suggestive.
No characteristic eruption.	The eruption (appearing in successive crops) is pathognomonic.

ACUTE GENERAL MILIARY TUBERCULOSIS.	TYPHOID FEVER.
Widal reaction absent.	Present.
Knee-jerk may be absent.	Knee-jerk never wanting.
Leukocytosis may be present.	Leukocytosis absent unless complicated by a suppurative process.
Choroid tubercles may be detected.	Choroid tubercles absent.
Tubercle bacilli rarely demonstrable in the blood.	Cultures from punctured spleen may show typhoid-bacilli (dangerous procedure). They may be found in the stools.
Hemorrhage from bowels exceptional.	Hemorrhage from the bowels common.
Perforative peritonitis absent.[1]	Perforative peritonitis often present.

The tuberculin test may prove an aid to diagnosis in cases pursuing an apyrexial course.

(*b*) PULMONARY FORM.

Though all gradations between the typhoid and the pulmonary types occur, the latter should be recognized and briefly described. It may develop *suddenly*, the ushering-in symptom being sometimes a *chill*, though more frequently there is a *premonitory period*, during which the general health fails materially. Some acute illness, as measles or whooping-cough, in which there has been marked catarrhal bronchitis, often constitutes the point of departure for this variety.

The *respiratory symptoms* are early prominent, and later preponderate in the clinical picture. From the start there is dyspnea, and this gradually increases until the respirations become rapid (40 to 60 per minute). When dyspnea becomes pronounced the face assumes a characteristic cyanotic pallor. The cough at first is moderately severe, but it soon becomes troublesome, being frequent and attended with a slight expectoration, which, however, is non-characteristic.

The **physical signs** are those of broncho-pneumonia, and the latter may or may not be preceded by the signs of generalized bronchitis. With the onset of consolidation there appear spots that yield either dulness or a tympanitic resonance on percussion, and broncho-vesicular breathing with numerous subcrepitant râles on auscultation.

The general symptoms are marked from the beginning. The *fever* is high—from 103° to 105° F. (39.4°–40.5° C.) or often higher. The pulse ranges from 100 to 140, is small, feeble, and sometimes irregular, and it may be more rapid still during the advanced stage of the affection (see Fig. 23). Cerebral symptoms rarely appear.

The **course**, as a rule, is more prolonged than that of general miliary tuberculosis, except in children, in whom it often runs an exceedingly acute course. As the end approaches the signs of suffocation are gradually intensified, and finally lead to a fatal termination.

Diagnosis.—The diagnosis is difficult; but a family history of tuberculosis, a knowledge of the pre-existence of a tuberculous focus or of an antecedent predisposing affection, will aid in its recognition. Tubercle bacilli are perhaps not demonstrable in the sputum unless an old tuberculous lesion is present. In doubtful cases, however, an attempt should be made to detect the bacilli in the blood. Occasionally either tuberculous meningitis or peritonitis supervenes, and aids in removing the doubt, and in a small percentage of the cases choroid

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[1] See also Differential Diagnosis of Typhoid Fever.

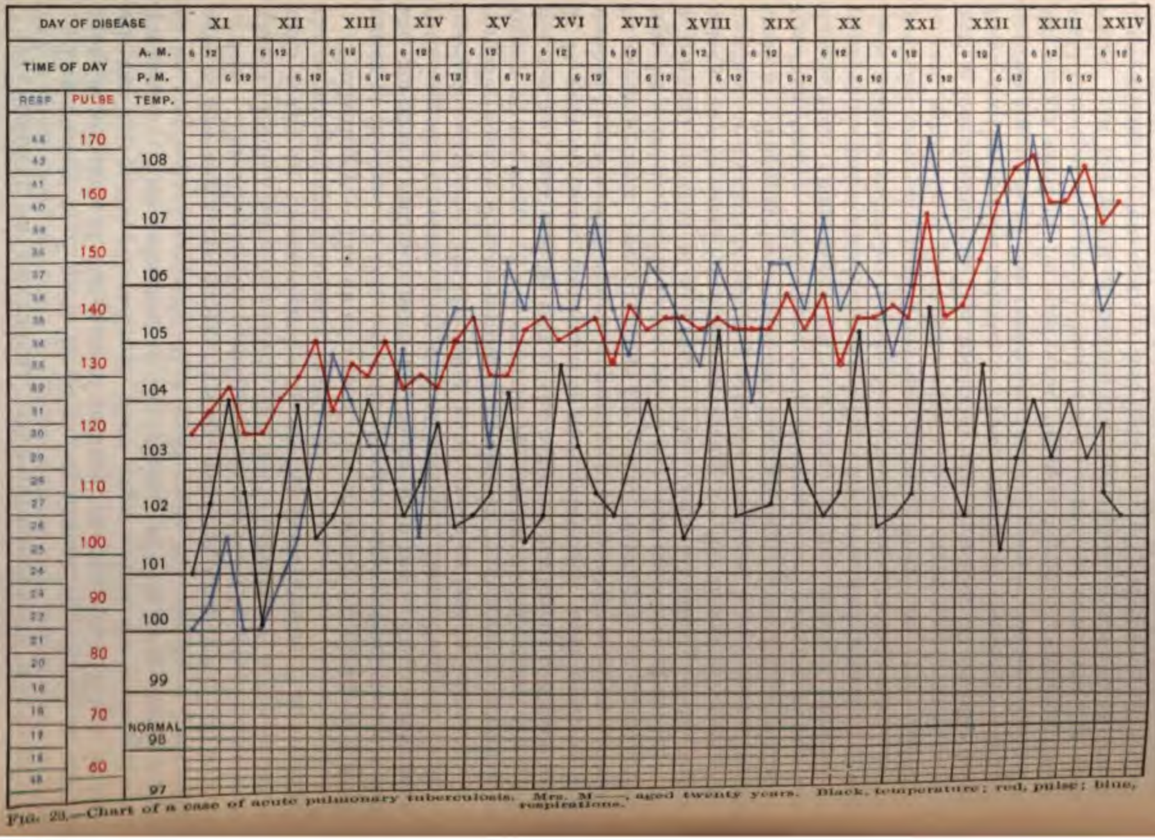

FIG. 23.—Chart of a case of acute pulmonary tuberculosis. Mrs. M——, aged twenty years. Black, temperature; red, pulse; blue, respirations.

tubercles are detectable. These points, together with the more marked general symptoms, will usually enable the clinician to distinguish this variety of tuberculosis from *non-tuberculous broncho-pneumonia.*

(*c*) CEREBRAL OR MENINGEAL FORM (TUBERCULOUS MENINGITIS).

This variety is of quite frequent occurrence, appearing in not less than 50 per cent. of the cases of miliary tuberculosis. When it develops the symptoms referable to other organs than the meninges are in abeyance. With reference to the etiology of this form one fact needs to be emphasized—namely, that most cases are observed between the ages of two and seven years; it may, however, be met with at any time of life. The affection frequently has its origin in tuberculous bronchial glands (Jacobi), and the history of a fall is common. A few cases have been found to be associated with erythema nodosum. Exceptionally the meninges are primarily involved.

Pathology.—The chief site of the tubercles in children is the pia mater at the base of the cerebrum (basilar meningitis), whilst in adults the pia at the vertex is more apt to be involved. The membrane surrounding the tubercles may not be inflamed, there being a simple tuberculous deposit. On the other hand, more or less inflammation, with sero-fibrinous or fibrino-purulent exudation, is generally present in the region of the base. This exudate is usually abundant in the Sylvian fissures, and may find its way to the external surface of the hemispheres. It is gray in color, transparent, and gelatinous, and contains in its meshes the tubercles, which appear as grayish-white bodies, and which, in cases of equal severity, may be either numerous or scanty. They may be scarcely visible to the naked eye, but may vary from the size of a pinhead to that of a French pea. The branches of the Sylvian artery may be implicated, either owing to the direct pressure of the exudate or to the obliterating arteritis produced by a tuberculous infiltration. The pia looks like wet blotting-paper over the quadrangle at the base (Gray). Elsewhere it is thickened and opaque, though easily detachable. Osler says: "The arteries of the interior and posterior perforated spaces should be carefully withdrawn and searched, as upon them nodular tubercles may be found when not present elsewhere. In doubtful cases the middle cerebral arteries should be very carefully removed, spread on a glass plate with a black background, and examined with a low objective. The tubercles are then seen as nodular enlargements on the smaller arteries." Involvement of the chief vessels that nourish the walls of the ventricles and the ependyma, and stretch from the vermis cerebelli forward over the quadrigemina, explains the constant presence of a turbid fluid in the ventricles, with softening of their walls. As the result of undue intraventricular pressure the cerebral convolutions become more or less flattened, with effacement of the sulci. The cortex, to a variable depth, is generally the seat of red softening, and more rarely of white softening alone. The tuberculous infiltration involves the cranial nerves.

Histology.—The tubercles grow in the perivascular sheaths, which are often distended with lymphoid and epithelioid cells, and there is observed not infrequently a thrombosis of the arteries and of the venules of the pia, obliterating their lumen. The pia mater is gradually thickened through cellular infiltration, and in a small proportion of the cases the spinal meninges are similarly involved, chiefly in the cervical portion of the cord.

Symptoms.—There is a *prodromal period* which lasts one or more weeks, during which the patient (usually a child) is pale, peevish, has headache and photophobia, and grinds its teeth during sleep; the tongue is coated, appetite impaired, and there may be occasional vomiting, either propulsive or regurgitative. Constipation is present and may be marked. Among rare premonitory symptoms are slight hyperesthesia of the abdomen and a diminished urinary secretion. A tendency to *emaciation* is quite constant. These prodromal symptoms present variations as to their number and combinations in different cases. In few instances only is the onset acute. The symptoms usually indicate basic meningitis, and at first there is associated considerable mental excitement; later there are pressure-symptoms (caused by the exudate), with total loss of the mental faculties.

(1) **Stage of Cerebral Excitement.**—The *invasion* is generally gradual, or even quite insidious, its most characteristic phenomena being *severe vomiting, marked headache*, and *chills followed by fever*. Certain other symptoms now arrest the attention, such as extreme irritability, screaming, and great obstinacy, and occasionally drowsiness appears early. When the onset is sudden the disease may be disclosed by convulsions, paralysis, wild delirium, or coma. The established disease exhibits certain distinctive features. The pain is often most excruciating, causing the child to utter short penetrating screams (hydrocephalic cry), and in rare instances the sharp cries may be continuous and lead to physical exhaustion. The headache is increased by light, noise, or movement. Vertigo is common; the pupils are contracted at this period; the face pales and then flushes; the pupils alternately dilate and expand; and the expression is sometimes sad, though more often stupid. Generally hyperesthesia or dysesthesia may appear, and there may be a slight mind-wandering at night, though active delirium is rare. *Tâches cérébrales* may be obtained, but are not characteristic. The patient is intolerant of every form of disturbance. All the symptoms of the prodromal stage are now aggravated; slight muscular twitchings and sleep-starts occur; the vomiting is apparently causeless, and may be frequently repeated; and constipation persists.

Fever is present, but is of slow development, and rarely rises higher than 102° or 103° F. (39.4° C.) in the evening. The *skin* is dry and harsh. The pulse is slow or moderately accelerated, but soon quickens to 120 or even 130, and later it may be irregular. At times the *pupils* are unequally contracted, and ptosis is usually an early sign.

(2) **Second or Transitional Stage.**—The symptoms of cerebral irritation now abate, the patient becoming more quiet, while mental dulness often supervenes. The vomiting and headache gradually subside, and the child rarely cries out. The abdomen is now distinctly scaphoid and the head occasionally retracted. Constipation is obstinate. The evidences of localized organic foci, such as slight twitchings of the muscles of the face, followed by strabismus, ptosis, or paralyses of the face or limbs, may appear. Generalized convulsions may occur, and

muscular tremors and athetoid movements may appear. Both pupils (or one only) may be dilated as intracranial pressure develops; patchy flushing of the face is common. The respiration is now irregular and sighing.

(3) **The Stage of Paralysis.**—On account of the exudation the mental faculties are abolished, so that the patient is comatose, though convulsions or localized spasms of the muscles in different parts of the body (neck, back, limbs, etc.) may be observed. Optic neuritis develops, while the paralysis of the ocular muscles above noted deepens. The pupils are dilated, the eyes are partly closed, and the eyeballs at intervals slowly and alternately move in a lateral direction. Hemiplegia sometimes develops, and more rarely monoplegia, affecting the face or one of the extremities. There may be paralysis of the third nerve, with involvement of the face, hypoglossal nerve, and limbs on the opposite side (a combination of symptoms first observed by Weber), consequent upon a lesion localized in the internal inferior portion of the crus. Monoplegia of the right side of the face has been observed in a few instances, associated with aphasia. Exceptionally aphasia and brachial monoplegia have been combined. The temperature in the early part of this stage usually rises to 103° F. (39.4° C.) or higher, but later it may drop to a subnormal level, and in rare instances as low as 94° F. (34.4° C.). Immediately preceding the fatal termination the temperature may rise to 106° or 107° F. (41.6° C.), the pulse becoming frequent, small, and irregular. Anesthesia comes on with general muscular relaxation.

Occasionally a *typhoid state* (great prostration, dry tongue, diarrhea, etc.) may develop, and Cheyne-Stokes respiration is almost invariably present, preceding the fatal event. Leukocytosis has been observed.

Ophthalmoscopic Examination.—The ophthalmoscopic appearances are—hyperemia of the disk, later the changes belonging to neuritis (swelling and striation) appear, and choroidal tubercles may be detected.

Diagnosis.—This is based: (1) On the reaction to tuberculin; (2) Examination of eyes, which present the characteristic appearance of the choroid coat (Jacobi). Post-basic meningitis gives the same symptoms, and lumbar puncture is the only means of diagnosis. In tubercular meningitis the diplococcus intracellularis is not found. *Syphilitic meningitis* and meningitis due to *trauma* may bear a close resemblance to the tubercular form, but the history should prevent confusion.

Clinical Types.—(*a*) **Mild Type.**—The marked or alarming symptoms (tetanic rigidity of the muscles, convulsions, and paralysis) develop at a late period. In this class should be placed those cases in which the meningitis is but feebly indicated—*e. g.* when it is but a small factor in the condition of acute general tuberculosis.

(*b*) **Malignant or Rapid Form.**—This type is comparatively rare, occurring most frequently in adult life, while the lesions have their seat almost exclusively upon the convexity. The onset is marked by the most frightful tetanic convulsions, which precipitate a fatal termination in a couple of days.

(*c*) **Chronic Type.**—Cases pursuing a chronic course are rarely encountered, and the symptoms usually point to localized cerebral lesions (Jacksonian epilepsy, etc.).

Prognosis.—The disease lasts from two to four or five weeks, though chronic cases may continue for several months. When the convexity is implicated, however, the duration is only one or two weeks. It should be emphasized that frequently in the course of well-marked cases a decided remission in the leading symptoms occurs, so that convalescence is suggested; but this is deceptive, and is almost invariably followed by a renewal of the unfavorable features of the affection. A few cases only are recorded in medical literature as ending in recovery. Freyhan has reported a case with recovery in which the diagnosis was proved by puncture of the spinal canal and the withdrawal of fluid, in the sediments of which tubercle bacilli were found. A. Jacobi has met with 2 cases that terminated favorably, and Leube has also reported a case in which the symptoms were characteristic, and at the autopsy, some years later, old tuberculous lesions were found in the meninges. It is to be recollected, however, that the course of tuberculous meningitis is probably uninfluenced by human agency.

ACUTE PNEUMONIC PHTHISIS.

(*Acute Phthisis; Florid Phthisis; Galloping Consumption.*)

This may be primary or secondary, the latter form being consequent either upon a localized tuberculous area in the lung, tuberculous pleurisy (acute or chronic), tuberculous peritonitis, or tuberculous disease of some other organ. Acute phthisis may occur at any age, though it is relatively more frequent in childhood and early adult life, but whether primary or secondary, the infection of the lungs is rapid.

Pathology.—Two forms may be recognized: (1) This reveals the appearances of an *acute lobar pneumonia*, one lobe only being implicated, as a rule, though sometimes the whole lung is involved. The process leads to a destruction of lung-tissue, so that a section may show the existence of cavities. The latter are usually small, while surrounding them may be seen tubercles in hepatized tissue, and here and there caseous masses of a yellowish-white color may be visible. These often indicate old or pre-existing foci. It is sometimes exceedingly difficult to distinguish a tuberculous croupous pneumonia from the ordinary form, and the most careful inspection may fail to reveal the presence of elementary tubercles in the acutely consolidated tissue. In cases in which this disease is suspected, however, the opposite lung, the bronchial glands, the peritoneum, and other organs should be carefully examined.

The lesions presented by cases that have run a long course are somewhat characteristic, though not always the same. If the case has had a duration of eight or ten weeks, apical softening with more or less extensive cavity-formation often occurs. Less frequently, a lobe or an entire lung is found to be consolidated throughout, "and converted into a dry, yellowish-white, cheesy substance, in which condition it may remain till the end."

(2) *Presenting the Appearances of Broncho-pneumonia.*—This variety is more common than the previous, especially in children. The evidences of bronchitis affecting the finer tubes, together with consolidation of the lobules to which the tubes lead, are striking. As in ordinary broncho-pneumonia, so here, the solidified areas appear as grayish-red masses in the early stage, while later they are of an opaque-

white. The products that fill the air-cells may caseate and break down, with the formation of irregular cavities that vary in size. When large areas are involved they are the result of the fusion of contiguous smaller areas of hepatized tissue. The trouble often begins in the upper lobes and spreads downward, though not infrequently the lower lobes are most extensively involved.

In not a few cases the masses are small, multiple, and widely disseminated throughout the lungs, and miliary tubercles in the lungs or pleuræ are associated with the broncho-pneumonic lesions before described. In nearly all cases signs of pleurisy may be noted, as is shown by pleural adhesions or by deposits of lymph on the pleura. The bronchial glands are also usually infected, and, particularly in children, are the seat of tuberculous processes.

Bäumler has called attention to a type of *tuberculous inhalation pneumonia* consequent upon hemoptysis, the blood and contents of the cavities being drawn into the finer tubes in respiration. This form of broncho-pneumonic phthisis sometimes follows pulmonary tuberculosis in the early, though more often in its late, stage. On microscopic examination tubercle bacilli are found, though rarely in abundance, in the infiltrated masses and in the walls of the cavities.

Clinical History.—(1) **Acute Cases.**—Preceding the attack, the patient may have "taken cold" or have been in a run-down state; more often, however, he has been apparently healthy. The *onset* is sudden, marked by a *rigor*, *pain in the side*, *fever*, *cough*, and *systemic prostration*, and there may be *bronchial hemorrhage* which may last one or more days. The total amount of blood expectorated may be considerable. In the majority of cases the *expectoration* is mucoid at first, and then becomes rusty-colored, often containing tubercle bacilli, though at first they may be absent and, indeed, not appear until late in the disease. *Dyspnea* appears early, and may soon become extreme, and the fever quickly rises to 104° F. (40° C.) or over. It may be of the continued type, or it may early assume the remittent or hectic type, and with the latter forms of fever, which usually begin about the end of the first week, are associated *night-sweats* and *rapid emaciation*. The prostration of the vital powers is now extreme. The expectoration is more abundant, muco-purulent, and often greenish-yellow in color.

In the course of one or two days after the onset we obtain *physical signs* that vary with the extent of the lesions. Usually, as before stated, these cases present the anatomic appearances of acute lobar pneumonia—viz. the complete consolidation of one or more lobes, which is usually followed by signs of softening, provided the patient survives the first week or ten days. The physical signs during the stage of consolidation are precisely the same as in lobar pneumonia. The signs of softening and of cavity will be given in detail in the description of Chronic Phthisis.

The *course* is usually rapid, occupying from two to six weeks on the average, though rarely cases that reach the stage of cavity-formation are protracted to three or even four months. Considering the brevity of the attacks, the extreme degree of emaciation (shown especially by the hollow cheeks and temples, pinched nose, and thin hands) is truly remarkable. The patient usually maintains a hopeful state of mind, notwithstanding the rapid downward course of the affection, and it may be admitted that recovery is possible. The parts involved are in such cases destroyed and replaced by fibrous tissue, and it should be remembered that the apex is involved in most cases. It commonly happens that consolidation only is present in the second lobe affected, while in the upper lobe one or more cavities have already been developed. The pleural crepitating friction is often audible before consolidation is complete.

Diagnosis.—The onset, symptoms, and course during the first week may be those of ordinary lobar pneumonia, but in some cases certain symptoms may arise which will excite suspicion of their tuberculous character in the early stage. Thus, hemoptysis rarely occurs in a pneumonia due to pneumococcus infection, and the appearance of the patient, as well as his previous and family history, may also be of a confirmatory character. The points of discrimination have been fully set forth in the section on Lobar Pneumonia (pp. 153, 154).

(2) **Subacute Cases** (rarely acute).—The *onset* is less sudden than in the former type, while the patient's antecedent condition may either be good or below the *standard*. At the beginning he has *repeated chills*, though *hemoptysis* may be the first symptom which indicates a pre-existing tuberculous focus. The *fever* rises high, and is apt to be irregular from the start; the *pulse* and *respirations* are rapid, and there is a *muco-purulent expectoration* which may either be profuse or scanty. Occasionally it is fetid, and the sputa may early contain *elastic* fibers and *tubercle bacilli*, though more often these are noted after the affection has become fully established. During the progress of the case, also, hemoptysis may arise. Later, drenching *night-sweats* increase the exhaustion and emaciation, which speedily reach an extreme degree, and soon or late a typhoid condition of the system is developed.

The physical signs are, at first, those of general bronchitis, with or without indications of pleurisy. Later, small areas of consolidation, which often increase in size, are indicated by impaired percussion resonance or dulness and by broncho-vesicular (rarely tubular) breathing, with subcrepitant râles. These signs may be unilateral, though more often they occur bilaterally. In many cases softening with cavity-formation ensues, with the usual physical signs of this condition.

Course and Duration.—For some time the patient may remain out of bed, although in most instances the disease constantly progresses. Less frequently there are exacerbating periods and remissions. Rarely these cases recover with a loss of more or less lung-tissue, though the condition may pass into chronic phthisis. It is important to recollect that the local lesions may become extensive, as the result of fusion of small consolidated masses, until an entire lobe is involved, and when this occurs the symptoms and course simulate those of the acute type. The *duration* ranges from two to eight weeks or more.

Diagnosis.—This variety is frequently confounded with non-tuberculous broncho-pneumonia, and the chief distinctions will be mentioned in connection with the latter disease. *Bronchiectasis* may be accompanied by emaciation, fetid expectoration, night-sweats, and the signs of cavity, and this disease has been mistaken for acute phthisis. Important in the recognition of the latter, however, are marked fever and emaciation. Moreover, the physical signs are more frequently referable to the apices, and the disease is more steadily progressive, running shorter course than bronchiectasis.

Acute Broncho-pneumonic Phthisis in Children.—The belief that the form of broncho-pneumonia that so frequently follows certain infectious diseases (measles, whooping-cough, etc.) is in the majority of instances tuberculous has been steadily gaining. Osler recognizes three groups of cases: (*a*) Those in which the child suddenly becomes ill while teething or during convalescence from fever, with high temperature, severe cough, and the signs of consolidation of one or both apices. Death may occur within a few days. To the naked eye the lesions do not appear to be tuberculous. (*b*) In this group the children show the ordinary symptoms of broncho-pneumonia, and the cases are more protracted, death occurring about the sixth week. (*c*) The child feels ill during convalescence from an infectious disease, fever, cough, and dyspnea being present. The intensity of the symptoms abates within a fortnight, and the physical examination shows the presence of diffuse bronchitis with scattered minute areas of consolidation. Many of these cases develop into chronic phthisis.

CHRONIC TUBERCULOSIS.

(*Chronic Pulmonary Tuberculosis; Chronic Ulcerative Phthisis.*)

This form is much more common than the acute, the term embracing sub-varieties to which attention will be incidentally directed. Its most typical clinical form follows a mixed infection as a result of a septic element superadded at some time to the primary tuberculous infection.

The Causal Factors have been detailed under General Etiology.

Pathology.—The pathologic characters of tuberculosis in general have been already presented, but it will be necessary to describe briefly the special anatomic conditions met with in chronic ulcerative phthisis.

The post-mortem appearances of the lungs in chronic pulmonary tuberculosis are remarkable for their great diversity, not only in the extent of tissue involved, but also as to the character of the morbid processes. Often the associated lesions form no unimportant part of the picture. In nearly all fatal cases the most advanced and extensive lesions are found near the apex, and, as a rule, the entire upper lobe of one of the lungs is implicated. In addition, it is observed that the destructive process has extended to the lower lobe of the same side as well as to the apex of the opposite lung, the lower lobe of the primary lung generally being invaded before the upper part of the other. Though both lungs are affected in fatal cases, they represent different stages of the disease. The case is very different in an old and cured tuberculosis of the lungs, such as is frequently met with in persons who have died of some other affection. Here the lesions may occupy but small part of one lung, and usually near the summit.

Kingston Fowler has investigated the question of the points of election and paths of distribution of the lesions in chronic phthisis, an has found that the primary lesion is not, as a rule, at the summit of th upper lobe, but that it occurs from 1 to 1½ inches (3.79 cm.) belo this point and near the postero-external borders. Favored by norma respiration, the lesions advance downward, so that on physical examination the first evidences of disease are to be found posteriorly over the lower part of the supraspinous fossa, while anteriorly the early signs are met with immediately below the middle of the clavicle, extending along a line running about 1½ inches (3.79 cm.) from the inner end of the second and third interspaces. The starting-point, though less frequently, may also be indicated by physical signs in the first and second interspaces below the outer third of the clavicle, with subsequent downward extension.

From personal observation of the post-mortem lesions of this disease, and from my studies at the bedside, I feel convinced that the initial lesion is frequently located anteriorly and near the apex, corresponding on the chest-walls to the clavicle and the supraclavicular spaces. This site has seemed to me to obtain more often on the right side than on the left. Kingsley has shown that when the lower lobe becomes involved the consolidation begins about 1½ inches (3.79 cm.) below its apex posteriorly, and corresponding externally to a spot opposite the fifth dorsal spine. From this point it spreads downward and laterally in a line following the border of the scapula "when the hand is placed on the opposite scapula and the elbow rests above the level of the shoulder." The middle lobe on the right side is usually invaded by direct extension from the upper. The seat of primary infiltration may even be the lower lobe, but this is an occurrence of great rarity.

The relative frequency of involvement of the two sides varies according to different authorities. A careful analysis of my own records, and the results of some statistical investigations into the subject, show that out of a total of 1236 cases 726 occurred on the left side and 510 on the right.

In all cases the primary lesions are due to *tuberculous infiltration*, which at first is confined to certain lobules, though it may later involve extensive areas of lung-tissue (*tuberculous broncho-pneumonia.*) In most instances the starting-point of the morbid changes is in the smaller bronchi and also, according to Payne, the inside of the alveoli. Soon the bronchioles and the corresponding air-cells become blocked with inflammatory products. These areas then undergo caseation and present the usual opaque, grayish-yellow appearance, a cross-section of these yellow nodules showing the central bronchus usually plugged with exudate and surrounded by caseous matter. Softening and sometimes complete liquefaction, with expectoration or absorption of the altered morbid products, may take place, and this disintegration is associated with *ulceration* in the wall of the bronchus, consequent upon secondary pyogenic infection, and a resulting formation of small *cavities*. Ulcers may form in the bronchioles before necrotic processes supervene, and they are generally shallow, with sharply-defined edges. Recovery may ensue as the result of *calcification* with encapsulation of the cheesy masses, or the affected area may undergo *fibroid transformation*—a conservative process and one that may lead to actual cure. It often happens, however, that old and apparently healed tuberculous lesions undergo ulceration, when the calcareous masses (pulmonary calculi) may be dislodged and expectorated, and the more rapidly the caseous masses are formed the more liable are they to softening. Surrounding the healed areas the tissue may be the seat of atelectasis, though more often of emphysema. Destruction of lung-tissue also results from interstitial inflammation with the formation of new connective tissue, the latter in turn compressing and finally obliterating the alveoli.

Cavities (*Vomicæ*).—These result chiefly from progressive necrosis and ulceration. They are formed mostly by dilatation of the bronchi, whose walls are tuberculous and suppurating. But they may also arise independently of the bronchi. Cavities vary largely in number, size, form, and in other characteristics. They are often multiple, though usually not far removed from one another, and unite as they increase in size. In this way large cavities, involving the whole of one lobe and even an entire lung (except the extreme anterior margin), may be formed, and a variable number of small pockets connecting with the bronchus may thus originate. The walls of the cavities are almost invariably irregular.

Vomicæ may be classified as (1) progressive and (2) non-progressive.

(1) The **progressive** are divisible into (*a*) New cavities and (*b*) Old cavities.

(*a*) *New cavities* have soft, necrotic, friable walls so long as the destructive processes are rapidly progressing, and the same state of things prevails in the cavities of acute phthisis. They may develop near a healed focus or near old cavities with limiting walls, and when situated near the periphery of the lung they may rupture into the pleura, causing pneumothorax.

(*b*) *Old cavities*, as a rule, have sharply-defined walls that vary considerably in thickness. At first they consist of a fibro-vascular zone, which has an inner suppurating surface; subsequently the lining of this zone is converted into an exfoliating membrane. The contents of vomicæ are muco-purulent or purulent, and often consist of a shreddy and sometimes a bloody fluid. Rarely they are gangrenous. Cavities also contain tubercle bacilli and other micro-organisms. Percy Kidd has studied the question of the relation of tubercle bacilli to tuberculous pulmonary lesions, and states that they are invariably present in newly-developed tubercles and fresh cavities, but frequently absent in old nodules. Trabeculæ composed of blood-vessels and remnants of pulmonary tissue often traverse the cavities. In old cavities excavation may be complete, not a vestige of normal or diseased tissue remaining in them, though the blood-vessels, many of which are beaded by small aneurysmal dilatations along their course, are the last to disappear. Their removal is effected by an obliterating inflammation. Rupture of these miliary aneurysms or the erosion of a large vessel is an event that gives rise to copious hemoptysis. Cavities having dense walls may also increase in size by encroaching upon and destroying the surrounding tissue, huge cavities often having thin, tense walls. But, wherever situated, they usually begin toward the summit of the upper lobe. Another common seat is the mid-dorsal region.

(2) **Non-progressive Cavities.**—Quiescent cavities are usually small, though variable in size, according to the stage at which the process of contraction is arrested. Medium-sized and large vomicæ do not become totally occluded. They may be multiple, though more often perhaps single, and associated with them may be observed dense, fibrous

dules representing healed foci. Their interior may be lined with a ooth, cuticular structure resembling mucous membrane.

Interstitial Pneumonia.—In the course of chronic phthisis interstitial flammation of two sorts will most probably arise: (*a*) A consolidation cited by the tubercle bacilli themselves, and hence manifesting a *destructive* tendency; (*b*) A slowly-developed *interstitial pneumonia* which ims at *arresting* the progress of the affection. It develops in close roximity to caseous masses and around cavities. The new connective issue thus formed in obedience to the well-known pathologic law tends contract secondarily, and thus vomicæ are often partly, though seldom entirely, obliterated. The shrinking of the connective tissue may also result in compression, and finally in the destruction of pulmonary tissue, just as in a tuberculous inflammation. The process in this instance, however, is on the whole conservative and reparative.

Disseminated Tuberculosis.—*Miliary Tubercles.*—This form has for its chief characteristic miliary tubercles, which are scattered not only about the tuberculous area, but also throughout the rest of the lung, and usually in the lower lobe. Most of the tubercles undergo fibroid or fibro-caseous change. These minute, hard gray or grayish-yellow nodules vary in size from a mustard-seed to that of a pea, and lung-tissue that is more or less studded with chronic miliary tubercles is apt to look pale, while the surrounding air-cells are *emphysematous.* The condition may lead to pneumonia, and the whole aspect then becomes altered. Here, as before described, fusion of miliary tubercles results in larger masses which become caseous, and hence the method of cavity-formation is identical with that observed in tuberculous broncho-pneumonia. In the disseminated form tubercles may also be found in many other organs than those indicated (pleura, trachea, larynx, bronchial and other lymphatic glands, peritoneum, spleen, kidneys, liver, brain, mucosa, testes, etc.).

Lesions of the Pleura.—This membrane is hyperemic and coated with fibrinous exudation coextensively with the affection of the parts in chronic ulcerative phthisis. The pleural membranes are only more or less thickened by organized adhesions, but in the latter and also in the pleura tubercles or cheesy masses may be found. Simple and other forms of pleurisy are also met with—sero-fibrinous, purulent, and hemorrhagic.

Lesions of the Bronchial Glands.—At first these are enlarged and edematous, containing tubercles, and later they present foci which often undergo purulent disintegration and sometimes calcification. Other lymphatic glands than these may be affected (mesenteric, etc.).

Lesions of the Larynx.—The larynx is frequently the seat of tuberculous infiltration and ulceration, particularly in certain parts, such as the vocal cords, posterior wall, ary-epiglottidean folds, etc.

Lesions of the Heart.—Tuberculous endocarditis is present in about 5 per cent. of the cases, and congenital stenosis of the pulmonary orifice is noted in not a few instances (Chevers). The right heart is often hypertrophied or dilated.

Other organs may present lesions in chronic phthisis, and these will be spoken of in connection with the clinical history.

Tuberculosis of the intestinal canal is a common though late lesion.

Amyloid degeneration of certain organs is a not unusual secondary event, especially of the kidneys, liver, spleen, and intestinal mucosa, and in like manner enlargement of the liver due to *fatty infiltration* is noted not infrequently.

Clinical History.—The modes of invasion are quite diverse, but with few exceptions the onset is either (1) gradual or (2) abrupt, and, as a rule, the health has been previously undermined for a longer or shorter period.

(1) **Gradual Onset.**—(*a*) The disease often originates in a manner similar to the origin of *ordinary bronchitis*, and combined with the symptoms of broncho-catarrh in some cases are those of pleurisy. Tuberculous bronchial affections often follow certain acute infectious diseases—influenza, typhoid, measles, whooping-cough, etc.—and in this form are rarely curable. The *physical signs* may be negative for some time, and then appear in the apex region, and the most characteristic grouping of physical signs during the incipient stage may be thus summarized: "Lagging" or defective expansion, as noted in inspection and palpation, a localized increase in the tactile fremitus, slightly impaired percussion-resonance, enfeeblement of the normal vesicular murmur, with (at a later period) prolongation and sharpening of the expiration. The fact that the lesions are commonly detectable in the suprascapular fossa must be remembered. At this period obvious *constitutional disturbances* are present (debility, emaciation, fever).

(*b*) *Onset with Pleurisy.*—This may be sudden, as in an acute pleurisy with effusion, but often the latter condition develops insidiously. Of 90 cases of pleurisy with effusion, one-third terminated in chronic phthisis (Bowditch). It may begin as a dry pleurisy at the apex, either anteriorly or posteriorly, or the evidence of pleurisy may be associated with the more common, bronchitic onset.

(*c*) *With Gastro-intestinal Symptoms.*—There is impaired digestion, and soon the patient becomes anemic, loses flesh, and is debilitated. Later, the first indications of pulmonary tuberculosis develop in the lungs. It is most important to keep in remembrance that close scrutiny of the data entering into the early history of cases of pulmonary tuberculosis usually reveals some perversion of the general health before distinctive pulmonary phenomena arise.

(*d*) *With indefinite peritoneal symptoms*, lasting for months or years.

(*e*) *With Laryngeal Symptoms.*—This is a rare form. It begins with hoarseness, more or less aphonia, and considerable cough; there is also a slight muco-purulent expectoration. Laryngoscopic examinations may detect tuberculosis of the organ, and tubercle bacilli may be found in the sputum before involvement of the lungs is discoverable.

(2) **Cases with Abrupt Onset.**—(*a*) The most important group under this category is heralded by the symptoms and signs of *acute pneumonia*, more commonly of the lobular variety. As compared with ordinary pneumonias, these present some peculiar features: the fever is irregular and the expectoration is more abundant, is blood-stained, and contains bacilli. The signs are usually located in the apical region. Resolution may occur, but recovery is not complete, and the condition is likely to pass into chronic phthisis.

(*b*) *Onset with Fever.*—Chills and fever are apt to arise in all instances the advanced stage of pulmonary tuberculosis, and these symptoms ay also initiate the attack. There is no mistake in diagnosis more ommonly made in malarial regions than to ascribe such cases to paldism.

(c) *With Hemoptysis.*—This symptom may be the first to invite attention to lung-trouble. In the majority of cases the amount of blood lost is considerable, and, less frequently, repeated slight hemorrhages occur. Pulmonary symptoms may be absent, sometimes temporarily, and in rare instances, perhaps, permanently; but in a great proportion of cases the clinical picture of incipient pulmonary tuberculosis is revealed pursuing its accustomed course immediately after the occurrence of the hemorrhage. The physical signs may be latent for a time, and, whilst they are usually located in the subapical area, they may assume the guise of a pleurisy in the scapular or infrascapular region. A slight tuberculous lesion is most probably present in these cases preceding the occurrence of the hemorrhage.

The **symptoms** are (1) *local* and (2) *general.*

(1) **Local.**—(*a*) *Pain.*—This is absent in many cases of chronic phthisis and in others it may be moderately severe. It is seated usually at the base, laterally or anteriorly, and not rarely there is pain of a lancinating character in the interscapular region in the early stages of the affection. This symptom is of diagnostic worth only after other forms of pain (rheumatic, neuralgic) have been excluded. The most common cause of pain is pleuritis, with or without pleuritic adhesions; it is increased on deep breathing and coughing. Intercostal neuralgia and pleurodynic stitches may also develop in the course of this disease.

(*b*) *The Cough.*—This may be looked upon as an essential feature, though in a few instances it may be slight or even wanting throughout. Its severity bears no constant relation to the extent of the pulmonary lesions, but rather to the degree of sensitiveness of the patient. It is dry and hacking at the beginning, and, if the larynx be involved, the cough is marked and takes on a hoarse quality. It is most pronounced at certain periods of the day—viz. on lying down at night and on awaking from sleep. Paroxysms may also occur after meals, and these occasionally induce vomiting. The cough is at times distressing and debilitating in its effects.

(*c*) *Expectoration.*—At the beginning the sputum is scanty and mucoid, rarely hemorrhagic, or it may be merely streaked with blood; later it may become muco-purulent, and the appearance of small gray or grayish-yellow flocculi first suggests the nature of the affection. With the onset of the stage of cavity-formation the sputum becomes more abundant and more distinctly purulent, and, after the formation of cavities of any size, airless, opaque, and nummular (coin-shaped) masses are expectorated. The latter are greenish-gray or greenish-yellow in color, and sink rapidly when discharged into water. They are often mingled with more or less bronchial secretion, and are not entirely characteristic of tuberculous cavities, being sometimes observed in pure bronchitis. They may even be absent, and the expectoration be merely purulent. The opening of a fresh cavity may be followed by very free expectoration. The sputum is sometimes fetid, and exceptionally it is horribly offensive, varying greatly in amount in different cases and at different stages of the disease. In certain cases it is absent throughout the greater portion of their course, and is especially apt to be slight in children and old people. In such instances it may be impossible to collect sufficient sputum to examine for bacilli.

Microscopic examination discovers alveolar epithelium (particularly in the earlier stages), pus-cells, blood, fat-globules, elastic fibers, and *tubercle bacilli*, the detection of the latter being the most important factor in the diagnosis. It may be safely stated that the finding of bacilli in the sputum is *prima facie* evidence of chronic phthisis; on the other hand, however, their absence in the early stage does not exclude the disease. It is often needful to make repeated and delicate examinations of the sputa. It is also of the utmost importance to select for examination the small grayish masses that are usually to be found, since they early contain the bacilli. In tuberculosis in the aged tubercle bacilli are not always detectable in the sputum.

Method of Examining the Sputum.—"A small amount of the purulent portion of the sputum is spread in a thin and uniform layer on a perfectly clear cover-glass by means of forceps, needles, or the Ohse, which must previously be held a moment in the flame of a Bunsen burner or a spirit lamp, or by pressing a small amount of sputum between two cover-glasses, then sliding them apart. It is then dried in the air, or more quickly by holding the cover-glass with forceps some distance above the flame of a burner or lamp. Finally, it is to be passed three or four times through the flame, and so 'fixed'" (Musser).

The preparation may be stained with carbol fuchsin (basic fuchsin 1, alcohol 10, 5 per cent. solution of carbolic acid 90), either by dropping a few drops of the stain on the smeared side of the cover-glass and holding it above the flame until it steams, or by floating its face downward upon a watch-crystal containing the solution. It must then be decolorized either with a 30 per cent. solution of nitric acid, allowing it to remain until the red color has entirely disappeared (about fifteen seconds), and then washing and counter-staining with methylene-blue, or with Gabbett's solution (methylene-blue 2 gm., sulphuric acid 25 c.cm., water 75 c.cm.), in which it must remain until the red color has been replaced by a faint blue (thirty seconds or more). Instead of carbol-fuchsin, anilin gentian violet may be employed (add a saturated alcoholic solution of gentian violet to a filtered saturated solution of anilin until a metallic luster appears on the surface). The specimen may lie either several hours in a cold solution or a few minutes in one that is steaming. Decolorize with the nitric-acid solution and counter-stain with rubin or Bismarck brown. It is often much simpler to smear the sputum directly upon the slide, and then examine, when stained, without the intervention of a cover-glass. A much larger amount of sputum can thus be prepared at a single operation.

In the microscopic examination use a $\frac{1}{12}$-inch (2.11 mm.) oil-immersion lens and Abbe condenser, or, at the least, $\frac{1}{7}$- or $\frac{1}{8}$-inch (0.36 cm. or 0.31 cm.) objective. If carbol-fuchsin has been used in staining for the bacilli, and methylene-blue as a contrast, the former will be found as red rods in a blue field (background), while if gentian violet has been used, the tubercle bacilli appear as dark blue rods, with all other bodies rown, if Bismarck brown is used for the contrast stain. There may e visible in the field a few bacilli only, particularly during the early art of the case. In the stage of cavity their number is usually increased, and sometimes they are quite numerous. Dorset[1] describes a apid method of great practical value for staining the organism in tissues, and for the purpose of differentiating between the tubercle bacilli and the smegma-bacillus (the latter not taking the stain), as follows: Coverglass preparations are made and fixed in the ordinary way, and then immersed in a cold, saturated 80 per cent. alcoholic solution of sudan III for five minutes. Any excess of stain is then removed by washing in several changes of 70 per cent. alcohol for five minutes. The bacilli are thus stained a bright red and the beaded appearance is quite distinct.

The demonstration of *elastic fibers* is also an important aid to diagnosis. Fenwick's method is the following: Boil the sputum with an equal quantity of a solution of caustic soda (gr. xv–℥j—0.972–32.0); pour the product into a conical glass and fill with cold water. The sediment is subsequently examined with care for elastic fibers.

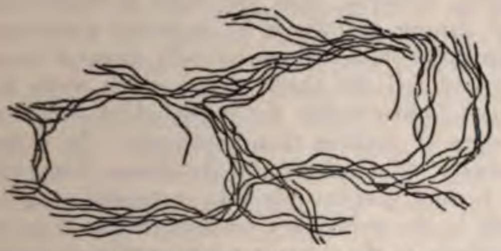

FIG. 24.—Elastic fibers (after Strümpell).

The *form* and *appearance* of the elastic threads differ according to their special source. If they come from the alveoli, there is an interlacing of the fibers which may preserve the globular contour of the air-cells. If they come from the blood-vessels, they are single and elongated, or two or three of the fibers may be arranged side by side. Elastic tissue derived from the bronchi has a similar appearance.

The presence of elastic fibers furnishes incontestable proof that destruction of lung-tissue has taken place. To show that this loss of structure, however, is due to tuberculosis, we must exclude abscess (an exceptional event) and gangrene of the lungs—diseases in which it also occurs.

(*d*) *Hemoptysis.*—This symptom of phthisis will be spoken of under Diseases of the Lungs, but its importance as a diagnostic feature of this disease makes special reference to it here absolutely necessary. It is present in the majority of cases. Gabrilowisch[2] found that of 380 patients 213, or 56 per cent., had hemoptysis. The sputum may be merely blood-stained, or the hemorrhage may be excessive and prove rapidly fatal, though hemoptysis is rarely the direct cause of death in tuberculosis. Slight hemorrhages are usually produced by mere hyperemia, and are most apt to occur during the early stages; while severe bleedings are produced by the erosion of a blood-vessel or rupture of a small aneurysm, and are most prone to occur during the stage of cavity. In certain cases hemoptysis is frequent.

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[1] *New York Medical Journal*, Feb. 4, 1899.

[2] *Berliner klinische Wochenschrift*, Jan. 2, 1899.

A third or capillary form of hemorrhage may occur in phthisis with cavity-formation, and in this variety, which is of a rather frequent occurrence, the purulent sputum is uniformly stained with blood. It may also be nummular, but presents a reddish-brown or chocolate color. The *exciting cause* is seldom obvious, though in not a few instances aggravation of the cough, and in others great mental excitement, would appear to excite bleedings. Slight hemorrhages often, and severe ones rarely, afford more or less relief to the pulmonary condition. On the other hand, severe bleedings usually exert an unfavorable influence, being followed by debility and anemia. Moreover, in numerous cases hemoptysis is followed by a more rapid extension of the local lesions, with corresponding aggravation of the local and general manifestations. The fact remains, however, that the effect of severe hemoptysis upon the progress of chronic phthisis is by no means always untoward. In a case of my own there occurred periodically copious spontaneous bleedings (in spring and fall) for three years, which were as regularly followed by marked improvement for a period of three or four months. The physical signs of phthisis then developed. In a large number of cases of pulmonary tuberculosis the transition from warm to cold or cold to warm seasons corresponds with increased cough, hence with increased pressure in the pulmonary circulation; and so bleeding is also favored, particularly in those having a hemorrhagic tendency.

(*e*) *Dyspnea* is present, but is not a marked feature, as a rule, despite advanced pulmonary lesions. Perhaps the chief reasons for a lessened demand for oxygen on the part of the system are—first, the slow and gradual manner in which the lesions develop; and second, the pronounced bodily wasting. The *respirations*, however, are moderately increased in rate, averaging from 20 to 30 per minute, and this compensates admirably for the diminished breathing-space. The dyspnea may be greatly intensified, however, as the result of intercurrent pneumonia, pleurisy, active exertion, or great mental excitement, and toward the close of fatal cases the most intense dyspnea may be manifested.

Physical Signs in the Stage of Consolidation.—*Inspection* gives most important results. The paralytic or phthisical thorax is generally presented to view. It is flat, particularly the upper half; the intercostal spaces are wide; the ribs slope at a sharp angle from the sternum, making the epigastric angle acute and producing elongation of the chest. The same sharp inclination downward from the vertebral column i observed laterally and posteriorly. The angle of Louis is prominent and the depressions (supra- and infraclavicular, intercostal) are deepened, the costal cartilages being often prominent and the sternum, particularly in the lower part, sometimes much depressed or even concav (funnel-breast). The scapulæ stand out prominently and may be distinctly winged. A second type of paralytic thorax is narrow and long Pulmonary tuberculosis may, however, arise in chests of apparentl normal build. The paralytic thorax is often a resultant of develope phthisis. In subjects of obesity the phthisical thorax may be concealed

Again, the deformity due to occupation, as leaning over a desk, may ape the paralytic chest, and, finally, it may be the result of extreme emaciation. With the development of phthisis at the apex the depressions of the side affected are relatively deeper, while the clavicle often stands out more prominently.

Defective expansion is observed early, and usually at the apex of the side first affected; subsequently this may be more general, and finally bilateral. To note the motions of respiration with precision the examiner should occupy a position exactly in front of the median line of the patient's body. The difference in the movement of the two sides often becomes more apparent on deep respiration than on quiet breathing, and while at rest the respirations are almost normal, but exertion decidedly increases their frequency.

Palpation.—Testing the expansion by palpation gives better relative results than does inspection. To determine the comparative movements of the apices the extended hands should be so placed (by allowing them to diverge below) that the tips of the fingers touch the lower border of the clavicle, and then the patient should be asked to breathe deeply, though slowly. The expansion in the supraclavicular spaces is tested by standing behind the patient and using the tips of the fingers, or by allowing the two first fingers of each hand to pass parallel with the clavicles. In this way "lagging" over the apex will be the first symptom recognized, and may for some time be the only one.

Tactile fremitus is early increased with oncoming consolidation, though it is normally more marked at the right than at the left apex. If there be thickening of the pleura, however, it is diminished, and if there be pleural effusion it may be absent.

Mensuration.—The difference between the measurement of the chest in inspiration and expiration in any person of average health should be not less than three inches, and a difference below two and a half inches points strongly to tuberculosis. The data thus gained are more important than the shape of the *thorax.*

Percussion.—Resonance is deadened more and more as consolidation progresses. If the consolidated areas are minute, however, the percussion-note may be unchanged, and as the air-cells surrounding the latter are often emphysematous and relaxed, the note may be somewhat tympanitic. In many cases the tympanitic sound and deadness are intermingled, giving rise to the so-called tympanitic deadened sound. Slight dulness is, as a rule, noted first below the clavicle, though in not a few cases it is first detected upon and above the clavicle. The corresponding regions of the two sides must be compared during a held inspiration, and also during a held expiration. The degree of dulness can sometimes be better estimated by comparing the apical note with that obtained lower down on the same side, allowing for the normal topographic differences of intensity. The latter method is especially applicable to cases in which both apices are involved. Light and single percussion blows must be used. Impaired resonance may be detected early in the supraspinous fossa, and less frequently in the interscapular space if the subject is not too stout, though slight dulness in the absence of other signs has little diagnostic value. As the lung-tissue becomes airless throughout an area of considerable size it is markedly deadened, until dulness is heard; finally, with extensive consolidation the note may be wooden and the feeling of resistance be much increased.

Auscultation.—The vesicular breathing may be sharpened, owing to narrowing of the smaller bronchi, but more often perhaps it is diminished by the swelling and secretion. The corresponding regions on the two sides must be compared—first during quiet, and then deep breathing, and it should be remembered that prolonged expiration is an early and important diagnostic sign, at first being somewhat sharpened, and later distinctly bronchial. Tuberculous bronchitis may cause interrupted or jerking inspiration at the apex. If heard elsewhere, it has small value. With lobular consolidation at different points in the region affected, the conditions favor the transmission of the bronchial sounds, but these are toned down by the remaining intact air-cells; hence there is "transition" or broncho-vesicular breathing. With complete consolidation pure bronchial breathing is audible, and with the latter two forms of breathing crepitant or subcrepitant râles are heard. A clicking râle, although not commonly present, is an almost conclusive indication when observed. Sometimes the first râles which accompany the long expiration have a low whistling sound: with liquefaction they become more moist, are louder (somewhat ringing), and often bubbling, and may be heard on inspiration and expiration. If scanty, they may be audible on inspiration only; they are increased by coughing. If the moist crepitant and subcrepitant râles, often due to concurrent bronchitis, be very numerous, the breath-sounds will be obscured, but after free expectoration as the result of coughing the quality of the breath-sound is appreciable.

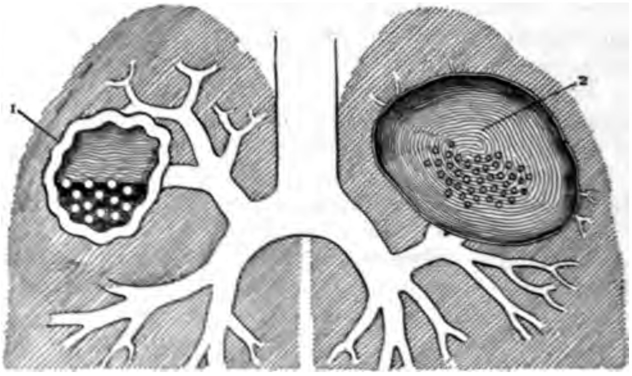

FIG. 25.—1. Small cavity near periphery, with thick relaxed walls, containing secretion and communicating with a bronchus (*vide* subjoined table). 2. Large parietal cavity, with thin, tense, smooth walls, communicating with a bronchus (*vide* table).

Pleuritic friction-sounds may be heard, due to accompanying pleuritis sicca, and these may be audible before the bronchial râles reveal the disease. Friction-sounds and râles often occur together. *Pleuro-pericardial friction* is present when the "lappet" of lung over the heart is affected, while clicking râles, occasioned by the heart's systole, are audible when the same area is pneumonic. The vocal resonance increases with the progress of the consolidation, and when the latter is complete *bronchophony* (rarely *pectoriloquy*) is present. In the subclavian arteries a systolic murmur is not uncommonly heard, the latter being supposed to be due to pressure exerted by the thickened pleura upon these vessels.

Physical Signs.	*Physical Signs.*
(*a*) Percussion-deadness on a strong blow, mere impairment of resonance on a light blow; Wintrich's interrupted change of sound, detectable when patient is upright, but not when recumbent.	(*a*) Amphoric percussion-resonance, cracked-pot sound, and Wintrich's change of sound.
(*b*) On auscultation low-pitched cavernous (hollow) breathing; gurgling râles.	(*b*) On auscultation, high-pitched amphoric (musical) respiration and metallic râles.
(*c*) Pectoriloquy indistinct, owing to small size of cavity and the contained fluid.	(*c*) Amphoric (musical) voice and amphoric whisper.

Physical Signs of Cavity.—*Inspection* shows a more marked retraction and a more decided lack of local motion than during the previous stage. The degree of shrinking is proportional with the extent of fibrous-tissue formation.

Palpation corroborates inspection as to lack of motion, and gives increased tactile fremitus if the cavity connects with an open bronchus and if it contains but little secretion. Excessive secretion interferes with conduction of sound.

Percussion.—Resonance is generally more or less impaired in consequence of the consolidation of the surrounding lung-tissue. The note may be somewhat tympanitic, but varies with the position of the cavities, the amount of fluid secretion contained by them, the condition of their walls, and the vibratory capacity both of the latter and of the individual thorax. Cavities of the size of a walnut situated in the apices usually give a distinctly tympanitic note, while cavities of the same dimensions, or even larger, in the lower portion of the lung do not. The metallic tone is especially noticeable over large cavities with smooth walls. The tympanitic sound may be deadened by closure of the connecting bronchus and by temporary filling of the cavities with secretion, and, again, if they are surrounded by thickened lung-tissue or by a large thickened pleura, there may be impaired resonance or absolute dulness even. Certain *special conditions* change the tympanitic sound over a cavity. Thus the note will be louder and exalted in pitch when the mouth is opened wide, and lowered when the mouth is closed (Wintrich's sign), there being dulness when the mouth is closed and tympanitic resonance when the mouth is open. If the cavity communicates freely with the bronchus, a tympanitic note may change in pitch with change in posture (Gerhardt's change of sound). If the patient changes from the dorsal to the upright position, resonance may give way to more or less flatness over the lower portion of the cavity, since the fluid contents of the latter are thus brought into contact with the chest-wall; this, although an almost certain sign of a cavity when present, is exceedingly rare. The so-called cracked-pot sound is often elicited over large parietal cavities with thin walls, and may be quite intense; but, since it also occurs in many other pathologic conditions, its diagnostic significance in this disease is subordinate. There may even be normal resonance if the cavity is covered by a layer of unaffected air-cells of considerable thickness.

Auscultation over small vomicæ with lax walls reveals *cavernous* (low-pitched) breathing, while over large cavities with tense walls (if parietal and communicating with a tracheo-bronchial column of air) it gives amphoric (higher-pitched) respiration. Moist râles (bubbling and gurgling, according to the consistency of the secretion) may be present, and these correspond in the main to the amphoric breathing, hence being heard most frequently over large, smooth-walled and peripherally-located cavities. The gurgling and slushing sounds caused by the air bubbling through the secretion in a cavity are always intensified by coughing.

The sounds of falling drops (metallic tinkling) may be heard over large vomicæ with tense, smooth walls containing thin secretion. *Pectoriloquy* and *amphoric whispers* are the vocal sounds heard over huge cavities, and to the latter should be given the greatest diagnostic significance.

General Symptoms.—(*a*) **Fever.**—Whilst the disease is progressing fever is a constant, significant, and, it may be, the earliest, symptom. If a two-hourly record be kept for a few days, from time to time an accurate conception of the course and type of the fever can be formed. In the first and middle stages the highest temperature occurs about 4 or 5 P. M., the lowest about 4 or 5 A. M. The fever may be continuous, remitting, or intermitting, and in a general way these types, in the order named, correspond to the stages of tuberculization, softening, and cavity-formation. Modified types, due to the fact that the lesions may simultaneously represent different stages, are also observed. Apyrexial periods are met with in the early as well as the late stages of chronic phthisis, and indicate cessation of the processes of tuberculization and caseation.

A continued fever is most apt to be met with during the initial period of phthisis, the evening temperature sometimes registering but a degree higher than the morning. A similar curve may be presented at any later time if acute pneumonia supervene, though it is to be recollected that the remissions in such cases are usually greater than in primary lobar pneumonia.

A remittent fever is more common than the preceding type. It may be present from the start, but is oftener seen in the middle, and less frequently in the advanced stages of phthisis. This form of fever points to softening (see Fig. 26).

An intermittent fever is also frequent, and is characteristic of cavity-formation, suppuration being invariably associated with the latter process. The temperature may be intermittent from the start, suggesting malaria to the unguarded; but it is due to sepsis, the temperature rising during the day, beginning usually shortly before noon, and reaching its maximum at from 5 to 8 P. M. It now falls slowly until about 4 or 5 A. M., and then rapidly reaches the minimum—a subnormal point—usually at from 6 to 10 A. M. For a considerable portion of every twenty-four hours the temperature may be below the normal (see Fig. 27), sometimes dropping as low as 95° F. (35° C.).

(*b*) **Night-sweats** occur in a large majority of cases. They may appear during any part of the course of phthisis, though most apt to occur and be most marked during the process of cavity-formation; they show themselves in the early morning hours simultaneously with the rapid decline in the temperature, and may appear during sleep at any period of the day. They may be light and limited to the neck and upper por-

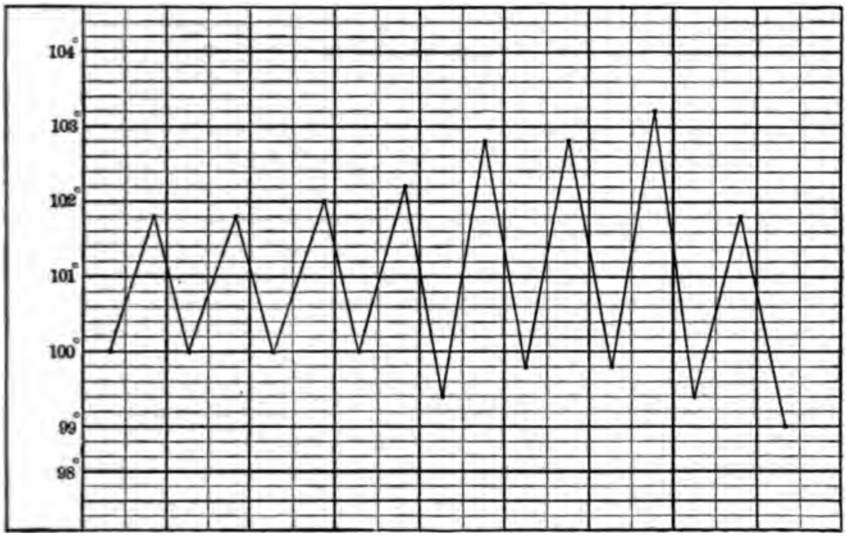

FIG. 26.—Temperature-chart of a case of phthisis. Quiescent cavity in right apex, and commencing excavation in left apex. Robert G——, aged 21 years; dyer.

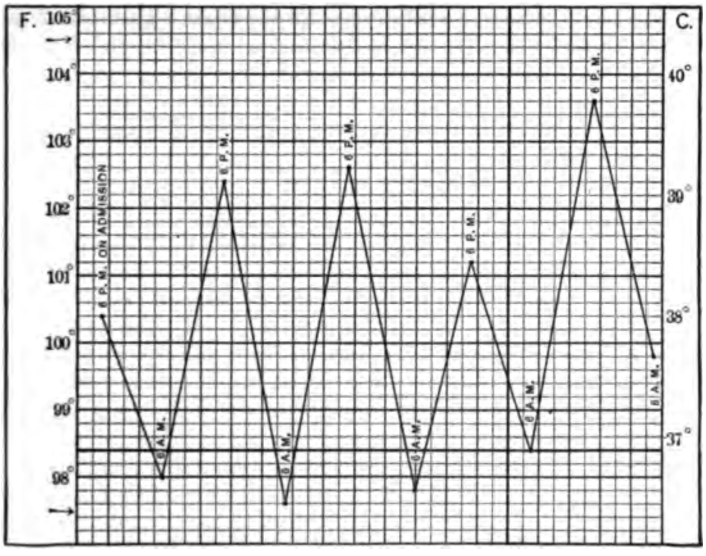

FIG. 27.—Temperature-chart of a case of phthisis. Cavity in left apex, giving cracked-pot sound, Wintrich's sign, etc. George C——, aged 22 years; glass-worker.

tion of the thorax; on the other hand, they are often excessive, saturating the bed-clothes and inducing great exhaustion. The drenching sweats are dependent partly upon the fever and partly upon the existing weakness, though slight exertion may also engender free perspiration.

(*c*) **Emaciation** occupies a prominent place in the symptomatology, the muscular and fatty tissues being involved to an equal degree (Strümpell); the extremities and soft parts of the thorax are most affected. It must be remembered that an exalted grade of emaciation may be present at an early period, and in such cases it may be assumed that the thinness of flesh was a precursory state. In nearly all cases an extreme degree of emaciation, reducing the patient to a slightly covered skeleton, is reached before the end. The causes of emaciation are chiefly the persistent fever, the loss of appetite, and the feeble digestive and assimilative powers. It is an almost invariable rule that during the afebrile periods, associated as they are with improved appetite and digestion, the patient gains in flesh and strength.

(*d*) **The pulse** is increased in frequency, is of good volume and regular in rhythm, though of low tension (soft). When suppurative fever sets in the pulse becomes very frequent, small, and compressible, and the capillary pulse is often observed; rarely venous pulsation may be noted in the backs of the hands.

(*e*) **Anemia** is one of the symptoms evidencing impaired nutrition, and seems to appear in certain cases before the more obvious local lesions. It is often associated with an afternoon rise of temperature, impaired digestion, and loss of flesh and strength (chloro-anemia)—a grouping of symptoms that should arouse strong suspicions of this disease. The objective changes pointing to anemia are pronounced (pallor of lips, visible mucous membranes, and skin). The *blood* presents nothing characteristic. In the early stage it may be chlorotic in type, the hemoglobin being decidedly deficient; but when there are cavity-formation and hectic fever, considerable leukocytosis, as many as 50,000 leukocytes per cubic millimeter, may be observed. The differential count shows a great excess of the polymorphonuclear cells. The condition is due to secondary infection by the pus-forming organisms.

General debility is complained of in all cases, and is progressive.

Symptoms and Complications presented by Other Organs.—(*a*) **The Heart.**—The pulse and the blood-appearances have already been described. With retraction of the upper lobe of the left lung the area of the heart's impulse is obviously increased, particularly upward, so that pulsation may be visible in the fourth, third, and even second interspaces, near the sternum, while the normal apex-beat may be wanting. The physical signs noted may be rarely those also of displacement of the heart to the right, while the necropsy may show the heart to be in its normal position. Functional murmurs both at the apex and at the pulmonary orifices are often audible. Disease of the tricuspid segments is not infrequent in phthisis, and pulmonary stenosis predisposes to the latter disease. Conversely, the old view that there is a discoverable element of antagonism between pulmonary tuberculosis and chronic valvulitis is still widely entertained.

(*b*) **Gastro-intestinal Tract.**—The *tongue* may be furred: more often it and the mouth and throat are red, showing increased irritability.

The *pharynx* may be the seat of tuberculous lesions, which may interere greatly with deglutition, *aphthous ulcers* being common, while *thrush* may also appear in the later stages. The appetite is impaired or lost: thirst is annoying, and the symptoms of chronic gastritis often obtain. A catarrhal ulceration and dilatation may be associated conditions; and the stomach may be extremely irritable. Vomiting may be troublesome during the last stage of the affection. A study of the gastric secretion gives variable results, there being an early hyperacidity, while later the secretion is subacid. Croner has found normal motility constantly present in the early stages.

The *causes* of gastric symptoms are not clear. The mucosa is the seat of venous engorgement, and may thus occasion the catarrhal changes that are present in many instances. The symptoms may be serious without adequate local anatomic changes to explain them.

The intestinal symptoms are but little less important than the gastric. During the early stage constipation is a frequent condition, and yet few cases run their entire course without manifesting diarrhea. The latter symptom may occur at any time, but is most prone to appear at an advanced period, and may pursue an intermittent course. Occasionally it alternates with periods of "hectic fever," and late in the affection a watery discharge may develop (*colliquative diarrhea*). The intestinal lesions are of three sorts: (*a*) *catarrhal*, (*b*) *ulcerative*, and (*c*) *amyloid*. These often arise in the order enumerated, but are not infrequently combined in various ways. Hemorrhoids and anal fistulæ are among the common complications.

(*c*) **Genito-urinary Organs.**—There is frequently an albuminuria that may either be of an ordinary febrile nature or due to chronic nephritis (*productive* and *non-productive*). *Chronic nephritis* is usually a late development, and is associated with a rather marked albuminuria, the presence of tube-casts in the urine, and dropsy. *Amyloid changes* may also set in toward the close, attended by their characteristic urinary phenomena. As secondary events tuberculous *pyelitis* and *cystitis*, with the appearance of pus and rarely blood in the urine, may develop. Hematuria may also result from temporary congestion. The testes may be implicated, and a routine inspection of these organs, as suggested by Osler, should not be neglected.

(*d*) **Cutaneous System.**—*Cyanosis* occurs, but, being of a moderate degree, it is often veiled by a decided pallor. The *cheeks* often wear a "hectic flush," and the skin, late in the affection, is apt to be dry, harsh, and scaly. Among the cutaneous appearances are pigmentary stains over the chest (chloasmata phthisicora), and seated in the same regions as well as upon the back are frequently seen brown stains (pityriasis versicolor). The *hair* over the chest often becomes gray; that of the head and beard, long and harsh. The *finger-ends* are often bulbous (clubbed), with incurved nails, though this is not peculiar to chronic phthisis, and cracking of the finger-nails is also often observed.

(*e*) **Nervous System.**—The mental attitude is characteristically hopeful and buoyant, even in the advanced stages. Hence the patients are readily encouraged by the unscrupulous to believe that their condition is improving, despite the steadily unfavorable progress of the disease, and, indeed, they may be in an utterly helpless state, and yet confidently expect to recover. The *cerebral symptoms* are rarely marked, and the mind, as a rule, is exceptionally clear. Tuberculous meningitis and meningo-encephalitis may develop near the close. I have met with cerebrospinal meningitis in one of my own cases. Focal lesions, due to the presence of tubercles, may produce forms of paralysis (aphasia, hemiplegia, etc.) according to their location. Rarely peripheral neuritis (usually an extensor paralysis of the leg) and insanity are observed.

(*f*) **Chest-muscles and Mammary Glands.**—The former are abnormally irritable, and sometimes even painful on percussion, and the mammary gland is in rare instances hypertrophied, males suffering most; but, as pointed out by Allot, the affection is a chronic non-tuberculous mammitis.

Diagnosis.—The early recognition of chronic pulmonary tuberculosis often tests severely the diagnostic acumen of the physician. The general and local symptoms, including the physical signs, may afford merely a strong suspicion of the existence of phthisis, and in such instances repeated examinations of the sputum for the bacilli are imperative, and only when they are found is the diagnosis set at rest. Repeated staining of the sputum may be necessary for the detection of tubercle bacilli. It is also desirable to determine whether they are constantly present by re-examinations at intervals. There are cases in which the physical signs are obvious, yet the bacilli are either not detectable or only so after several examinations; but a continued search for these micro-organisms is the first duty of the physician in such instances. An absence of the bacilli, however, does not justify a denial of the existence of phthisis, and is of little negative value. The symptoms of greatest diagnostic value are cough, expectoration, fever, progressive emaciation, and the constant presence of certain physical signs in the subapical region on one side (flattening of the front of the chest, defective expansion, slight deadening of the percussion-note, enfeeblement of the normal vesicular murmur, and prolonged expiration, with or without adventitious sounds). Francis H. Williams, of Boston, has recently produced skiagraphs that show the presence of tuberculous deposits and pleuritic exudates, and these may, at times, give the earliest positive information in regard to these conditions.[1] In the hands of an expert more reliable knowledge can be gained in the initial stage by the fluoroscope than by practising the physical signs.

The *tuberculin-test* is warmly commended by Trudeau, Otis, Klebs, and others. It is fairly accurate, and out of a total of 1470 injections in dubious cases, 71.9 per cent. reacted positively.[2] Its use should be limited to suspicious cases, after other methods of diagnosis have failed us, and medium-sized initial doses are to be employed. A positive reaction demands an elevation of temperature to 101° F., and this rise usually occurs within twenty-four hours, but it may be delayed until thirty-six or even forty-eight hours. The possibility of reaction occurring in cases of syphilis, leprosy, chlorosis, hysteria, actinomycosis, and other affections, will not lead to error, if it is noted that such reactions re less intense. It is important to determine the diagnosis early, and his is often practicable before the sputum-test gives a positive result. A slight rise of the evening temperature (99.6° F. or over) is, if associated with any disturbance of health, an almost infallible diagnostic symptom. In the more advanced stages of phthisis the diagnosis is rarely difficult.

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[1] For illustrative cases see "Diagnosis and Treatment of Prebacillary Stage of Pulmonary Tuberculosis," *The Journal of the Amer. Med. Assoc.*, January 12, 1901, by the writer.

[2] "The Value of the Tuberculin-test in the Diagnosis of Pulmonary Tuberculosis," by the writer, *New York Medical Journal*, June 23, 1900.

In the very early stage the local condition may be obscured by the symptoms of impaired digestion, loss of flesh and strength, fever, and pronounced anemia (chloro-anemia, *vide* p. 302; also *Modes of Onset*).

Differential Diagnosis.—*Bronchial catarrh* is with great difficulty discriminated from beginning phthisis. If the temperature is elevated from 2 to 5 P. M., and not at all or only slightly above the normal night temperature in the evening, the probabilities are greatly in favor of tuberculosis (Barlow). In bronchial catarrh there is no dulness, and moist râles, that vary in intensity from one day to another, are heard equally on both sides. From time to time râles may also be heard at the bases in bronchitis. In phthisis one apex is more involved than the other, the moist sounds not being heard equally low, and after repeated coughs with subsequent deep inspiration the rales are more apt to remain than in ordinary bronchitis. In phthisis, also, there is a gradual loss of flesh and strengh, and repeated microscopic examination of the sputum will demonstrate the presence of the bacillus. If *hemoptysis* be the first symptom observed, then all other causes for the spitting of blood should be patiently excluded, unless the associated evidences of commencing phthisis are conclusive. Phthisis in the stage of cavity may be confounded with *bronchiectasis* (*vide* Diseases of the Lungs).

FIBROID PHTHISIS.

Definition.—Fibroid phthisis implies induration followed by contraction of the affected lung-tissue, due to an increase in the connective-tissue elements. There are cases in which it cannot be distinguished pathologically from chronic pulmonary phthisis, but from a clinical point of view the two affections present distinctive peculiarities. The majority of instances are primarily tuberculous, though manifesting a strong tendency to the formation of fibrous tissue—a conservative process; in other instances the fibroid change may be primary, followed by tuberculous infection (*vide* Pneumonokoniosis). The usual form arises variously as a sequel of other morbid processes, such as—

(1) Pneumonias, lobar (rarely) and catarrhal pneumonia (commonly).

(2) Pulmonary lesions, such as a tubercle in the stage of consolidation or cavity.

(3) Chronic tuberculous pleurisy.

(4) Bronchial catarrh from inhalation of irritants (steel-, coal-, or mineral-dust). This condition may be found to be a tuberculous process accompanied by fibroid change.

Pathology.—The process in the beginning is very often localized in one apex, and less frequently in the middle portion of the lung or in the bases. It may remain circumscribed, but more often it extends downward, and gradually invades the entire lung. It is unilateral. Secondary to the induration and contraction there is dilatation of the bronchi.

The lung-tissue is hard and dense, the alveoli being obliterated. It resists cutting and creaks, and the section presents a smooth, dry, gray, often marbled aspect, though the fibrous tissue may undergo caseation. Tuberculous lesions may also develop in the opposite lung.

The pleura is thickened as a rule, often to a marked degree, and its layers are adherent; the unaffected portions of the lungs frequently become emphysematous. The right ventricle is, as a rule, hypertrophied.

Symptoms.—These may be briefly stated, since they do not differ from those of cirrhosis of the lung (*vide* Diseases of the Lungs). The *onset* is extremely insidious: a persistent *cough* that is apt to occur in severe paroxysms in the mornings, and a *purulent expectoration*, are for a long period the leading features. If bronchiectasis is present, the sputum may be fetid. *Dyspnea* is marked, particularly on exertion. *Fever* is slight or absent, hence emaciation progresses slowly or may even be absent.

The physical signs are obvious, and are identical with those of fibroid induration of the lung (*vide infra*).

The **course** of this disease is exceedingly long, ranging from ten to twenty or even thirty years, and, as I have before stated, both lungs may become the seat of tuberculous disease. Again, as in chronic pulmonary tuberculosis, prolonged suppuration may lead to amyloid changes in the liver, spleen, kidneys, and intestines. *Dropsy*, due to secondary dilatation of the right ventricle, often closes the scene.

Differential Diagnosis.—*Chronic bronchitis* may be mistaken for fibroid phthisis. In the latter disease, however, there are unilateral retraction and the signs of consolidation or of an apical cavity, and the sputum-test may settle the doubt.

Complications of Pulmonary Tuberculosis.—*Lobar pneumonia*, and less commonly *lobular pneumonia*, may develop and cause a fatal termination. In a study of 100 cases H. M. King found the principal complications of a non-tuberculous character were lobar pneumonia and nephritis.

Erysipelas may arise in the course of chronic pulmonary tuberculosis, though the proportion of cases is not formidable. Out of 1165 cases of erysipelas, 15 coexisted with pulmonary phthisis.[1] Some contend that its occurrence in this disease may be beneficial, but my own observations tend to show that the gravity of both conditions is increased when occurring together.

Typhoid fever may rarely be met with in sufferers from chronic phthisis, though I have seen but a single instance come to autopsy. In another fatal instance the characteristic symptoms of typhoid were present during life, but no post-mortem examination was allowed. It is important, however, not to overlook this occasional association of two such common affections.

Chronic nephritis and pulmonary tuberculosis are often found in the same subject, and with these arterio-sclerosis is quite commonly combined.

Chronic endocarditis, particularly of the tricuspid segments, may also occur in phthisis, and from time to time cases of valvular heart-disease are reported, in which it is evident that passive congestion must have existed for some time before the tuberculous condition developed. The old doctrine of the mutual antagonism between disease of the left heart and pulmonary tuberculosis finds not a little support from these cases, as in a large proportion a very considerable tendency to encapsulation of the tuberculous lesions exists.

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[1] "Points in the Etiology and Clinical History of Erysipelas," *Journal of the American Medical Association*, July 2, 1893.

Course and Duration.—Both as to course and duration this disease exhibits unusual variations. If not promptly treated during the time in which there is hope of therapeutic and climatic efficiency, it frequently progresses with more or less rapidity toward the grave. On the other hand, it is common to observe periods during which the disease is arrested or improved. Generally, the improvement, though followed by an exacerbation, endures for a long time, and permanent cures, even in the advanced stage, are by no means rare. The duration of pulmonary tuberculosis varies exceedingly, though from the collective investigations of different authors and from all the statistics available I find the average duration to be about three years. The late Austin Flint long ago directed attention to the innate tendency of a considerable percentage of the cases to spontaneous recovery—a fact that simply indicates a victory for nature's silent defensive processes in the struggle for supremacy.

In fatal cases death is by (*a*) *gradual asthenia* (most frequently), with retention of consciousness until the end approaches.

(*b*) *Complicating conditions* (bronchitis; pneumonia; pleurisy; pneumothorax; amyloid degeneration of the intestines, liver, spleen, kidney; Bright's disease; diabetes, etc.).

(*c*) *Tuberculosis of other organs*, particularly the meninges, intestines, and genito-urinary tract.

(*d*) *Hemorrhage*, due commonly to rupture of an aneurysm in the lung-cavity; less frequently to erosion of a large vessel. Fatal hemorrhage may, when the vomica is of large size, occur without hemoptysis, as in a case of Roland G. Curtin's at the Philadelphia Hospital.

(*e*) *Syncope.*—Though of comparatively rare occurrence, there are a number of events that may lead to sudden, fatal syncope—*e. g.* hemorrhagic embolism or thrombosis of the pulmonary artery, pneumothorax, thoracentesis for pleural effusion, walking about in a moribund state, etc.

(*f*) *Asphyxia* often closes the scene in acute pneumonic phthisis, and rarely in chronic phthisis complicated with pneumo-thorax, or with a large undiscovered or neglected empyema, or with sero-fibrinous pleurisy.

TUBERCULOSIS OF THE ALIMENTARY TRACT.

(1) **Lips.**—Whilst tuberculosis of the lip is quite rare, the possibility of its occurrence must not be forgotten. In a case of my own it assumed, as is usual, the form of a small ulcer, and the diagnosis was made by an examination of the labial mucus. It followed an accidental lesion of the lip from biting, and was not associated, as are most cases, with laryngeal or pulmonary tuberculosis. In *diagnosticating* the condition chancre and epithelioma must be excluded, the former by the history, and the latter chiefly by a microscopic examination for tubercle bacilli.

(2) **Tongue, Palate, and Tonsil.**—The work of Orth, Hanan, Schlenker, Kruckman, and others has shown that the tonsils, owing to their frequent inflammation, serve as the door of entrance of the tubercle

bacilli. The fact that tuberculosis of the tonsils has repeatedly been found, and when other lesions of the disease were absent, points to the not infrequent occurrence of primary tuberculosis in this site. The infiltrated areas often present small grayish spots, but the appearance of the ulcers is not characteristic, frequently bearing a strong resemblance to epithelioma and to the syphilitic ulcer. The *diagnosis* demands either inoculative experiments or a microscopic examination of the oral mucus, the latter being oft repeated if necessary. E. D. Smith records 5 rare cases of tuberculous ulceration of the soft palate and adjoining tissues, most of them secondary to pulmonary tuberculosis.

(3) **Pharynx and Esophagus.**—Both miliary tubercles and ulcerative lesions may rarely arise on the posterior wall of the pharynx by direct extension from laryngo-pulmonary tuberculosis or as the result of secondary inoculation. The chief symptoms occasioned are the excessive secretion of pharyngeal mucus and muco-pus, and painful deglutition. Tuberculosis of the esophagus is extremely rare. Osler, however, saw a case in his wards in which the ulcer perforated the esophagus and caused purulent pleurisy.

(4) **The Stomach.**—Tuberculous lesions appear only exceptionally in the mucosa of the stomach, notwithstanding the fact that marked gastric symptoms are of frequent occurrence. It should not be forgotten that nausea, vomiting, and other gastric symptoms may be directly due to involvement of the larynx. I have been able to find reports of 4 such cases in addition to the 12 collected by Marfan.[1] The ulcers may be single (as in Musser's case) or multiple (as in Osler's case). The *symptoms* are not characteristic, but hematemesis occurring in patients suffering from tuberculosis of other organs should excite a strong suspicion of the existence of ulcer. Pain coming on soon after meal-time is more marked in tuberculous ulcer of the stomach than in ordinary gastric lesions. Perforation has taken place in some cases, with its usual dire consequence.

(5) **Intestines.**—The lesions may be (*a*) primary, or (*b*) secondary to tubercles of other organs (lungs, peritoneum, etc.).

(*a*) Primary tubercle of the intestines is chiefly met with in children, for the reason that they are more likely to swallow the tubercle bacilli with their food, and especially in milk. The intestinal route of infection is, according to my own observation, more common in adults also than is supposed. Many cases during life present the features of both intestinal and peritoneal tuberculosis, and it is often impossible to determine which of these was the primary condition; and the same difficulty arises when the cases come to autopsy. I have never seen an instance (post-mortem) of intestinal tuberculosis in which the peritoneum and mesenteric glands were not involved to an equal degree.

(*b*) The secondary variety occurs in more than one-half of the cases of pulmonary tuberculosis, the chief seats of the lesions being the lower part of the ileum, the cecum, and the upper part of the colon. The rectum is also the seat of secondary tuberculosis in a small proportion of the cases of chronic phthisis and it may be rarely a primary seat of the affection.

The morbid process begins in the solitary glands in Peyer's patches, where at first grayish, firm tubercles grow and form little prominences.

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[1] *Paris Thesis*, 1887.

These caseate, becoming yellow in appearance, and then soften and disintegrate, producing ulcers. Osler thus describes the characteristics of the tuberculous ulcer: "(*a*) It is irregular, rarely ovoid or in the long axis, more frequently girdling the bowel; (*b*) the edges and base are infiltrated, often caseous; (*c*) the submucosa and muscularis are usually involved; and (*d*) on the serosa may be seen colonies of young tubercles or a well-marked tuberculous lymphangitis." In all *acute* cases the surface-lesions show little tendency to repair (Senn).

In *chronic* cases attempts at healing are the rule; and the cicatrices are extensive and often pigmented, and as they undergo contraction may produce incomplete or even complete stricture of the bowel. At a point corresponding to the seat of the ulcers local peritonitis invariably develops. The serosa is thickened and adherent, and the ulcer may penetrate through this coat without causing perforative peritonitis, while rarely a fistulous connection is established between the different parts of the intestine.

Symptoms.—In children the symptoms are those of a protracted catarrh of the intestines, or they may be absent. Among prominent features are diarrhea, colicky pains, and the presence in the stools of pus, blood, and particles of mucus resembling sago-grains. In many cases there is constipation, which may be due either to peritonitis or cicatricial stenosis. The general symptoms are irregular fever, wasting, and a lack of development; they may antedate the local, and are especially valuable for diagnosis.

In adults intestinal tuberculosis generally gives rise to symptoms similar to the above, and when they arise in the course of pulmonary phthisis they are highly significant. If diarrhea be present, it stubbornly resists treatment, and it must not be forgotten that this symptom may also be due either to catarrhal colitis or to amyloid change, both of which processes may be associated with chronic phthisis. Constipation is common and often marked, and local tenderness and colicky pains are complained of frequently. The pulmonary signs, however, may be in abeyance.

If the abdominal and general symptoms are such as to excite suspicion of this disease, then a rigid physical examination of the lungs should be made. The chief seat of the lesions may be for a long time in the cecum, or in the appendix, when the symptoms—both local and general—will be those of appendicitis.

The *diagnosis* of primary intestinal tuberculosis is beset with special difficulties. Sawyer[1] has in special instances demonstrated the presence of clusters of tubercle bacilli in the rectal mucus, and in this way the recognition of intestinal tuberculosis at an early date, or before diarrhea sets in, is rendered possible. The mucus is obtained after placing the patient in a position as if to examine for piles, and directing him to bear down as though at stool, by gently removing a small quantity from the everted membrane with a sterile loop. It is then spread upon a clean cover-glass and treated exactly as sputum in the ordinary examination. The same method is applicable to cases of secondary intestinal tuberculosis, but here the history and associated tuberculous lesions usually serve to remove all doubt.

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[1] *Medical News*, May 23, 1896.

TUBERCULOSIS OF THE SEROUS MEMBRANES.

General tuberculosis of the serous membranes *secondary* to pulmonary and intestinal tuberculosis is of common occurrence, and that a *primary* form of tuberculosis of the serous membranes also occurs is undoubted. Unfortunately, accurate means of discriminating the secondary from the primary form are wanting, since often in the secondary variety the primary lesions in other organs are insignificant.

The anatomic alterations resemble those of ordinary inflammation of these structures, plus the presence of nodular tubercles. The latter may be observed, as a rule, only over small, scattered, circumscribed areas, though not infrequently they are both numerous and diffuse (general miliary deposit). The effusion is in most instances sero-fibrinous, though sometimes it becomes purulent, and not uncommonly it is hemorrhagic. Most instances of so-called hemorrhagic pleurisy are due to pleural tuberculosis.

Clinically, cases are divisible into (1) acute serous membranous tuberculosis and (2) the chronic form. The *acute* form results from inoculation of the peritoneum or pleura, induced by limited foci in the bronchial, tracheal, or mediastinal lymph-glands, or in the Fallopian tubes in women. The *chronic* type is apt to result from a direct extension of a tuberculous process from some organ adjacent to the pleura or peritoneum, though it may attack the serous membranes primarily. Belonging to this class of diseases are two groups of cases: those attended by sero-fibrinous or sero-purulent effusion and the presence of caseous masses, and those in which there is a tuberculous deposit with increased density and great thickening of the pleural layers, and slight exudation. The pericardium may be similarly involved.

(*a*) **Tuberculous meningitis** has been described fully in the present section (*vide* Miliary Tuberculosis).

(*b*) **Tuberculous Pleuritis.**—This subject will be referred to in the section on Diseases of the Pleura. Its import, however, is such that brief special consideration is demanded, and from a clinical view-point the cases may be grouped under two heads—namely, *acute* and *chronic tuberculous pleurisy.*

The *acute* form often has a sudden onset, the initial symptoms being a rigor or repeated fits of chilliness, a stitch-like pain in the side affected, shallow, catching breathing, a cough, and fever. The ushering-in symptoms sometimes suggest lobar pneumonia, and a fatal termination is not uncommon, though apparent recovery or a transition into chronic tuberculous pleuritis also occurs.

Chronic tubercular pleurisy is vastly more common than the acute form, and it is sometimes primary, though more often secondary to pulmonary tuberculosis. In all cases of the latter disease in which the periphery of the lung becomes involved the visceral layer of the pleura is invaded. This leads to plastic pleurisy with adhesion, and the membranes contain disseminated tubercles, or to sero-fibrinous tuberculous pleurisy. As above stated, the effusion may be hemorrhagic and may also become purulent. When the tuberculous pulmonary focus perforates the pleural sac, pyopneumothorax is produced. In tuberculous pleurisy, as opposed to simple pleurisy, there is usually an absence of leukocytosis.

Symptoms.—The *onset* is very insidious and often unnoticed. There may be few symptoms, and yet a physical examination reveal a large sero-fibrinous exudate. The cough and other symptoms are frequently due to a coexisting tuberculosis of the lungs, and the presence of subcrepitant and dry râles is strongly confirmatory of tuberculous pleurisy. By and by the evidences of pulmonary tuberculosis are of diagnostic importance, or the supervention of acute general miliary tuberculosis makes clear the nature of the case. The *subacute variety* with effusion may terminate, after absorption of the exudate, in chronic adhesive pleurisy with great thickening of the membrane. The latter may also originate as a primary proliferative process.

(*c*) TUBERCULOSIS OF THE PERICARDIUM.

The morbid lesions are analogous to those of tuberculosis of the pleura. The effusion may be enormous on the one hand or insignificant on the other, and it is often hemorrhagic, while in the chronic form there is marked thickening of the membrane with the deposit of tubercles and cheesy masses. The affection is less common than tuberculosis of the pleura, yet not so rare as was formerly supposed, and occurs in the acute and chronic forms.

Acute tuberculous pericarditis is rarely a primary affection, and, as a rule, originates secondarily to pulmonary, pleural, or glandular tuberculosis. It is especially prone to arise in tuberculosis of the bronchial and mediastinal lymph-glands, and, as the latter condition is frequent in young children, so tuberculosis of the pericardium is relatively frequent at this period, though it may occur at any time of life. Pericardial tuberculosis also results from direct extension from a contiguous focus. The symptoms will be detailed in the discussion of Pericarditis. In the *diagnosis* of the affection the history and any associated tuberculous processes detectable must be taken into account, and a point of some diagnostic value rests in the fact that tuberculous pericarditis does not show the usual inflammatory leukocytosis.

Chronic Tuberculous Pericarditis.—This may be a part of the general tuberculosis of the serous membranes, or it may follow an infection of the bronchial and mediastinal glands (most frequently), lungs, pleura, or peritoneum. Undoubtedly, cases of primary origin also occur, but they are exceedingly rare, the neighboring lymph-glands being very generally involved. This form is also dependent upon direct extension from the spine, and sternum.

From personal observation I am convinced that the cases naturally fall under two heads, when considered clinically: those without effusion, in which the pericardium is adherent; and those with more or less effusion. The former are the more frequent, though often entirely latent, the adherent pericardium leading to hypertrophy of the heart, followed sooner or later by dilatation. The signs are therefore those of adherent pericardium, with the occasional difference that the dulness may extend higher up over the sternum, in consequence of the presence of firm, cheesy masses at the base of the heart and also encircling the aorta. The smaller group of cases (in which the effusion is present) resembles dilatation of the heart in its clinical manifestations. I recall one instance of this sort that occurred in a male aged about sixty years at the Episcopal Hospital, the autopsy revealing extensive pulmonary tuberculosis and chronic tuberculous pericarditis, with the presence of eight ounces of hemorrhagic effusion.

(*d*) TUBERCULOSIS OF THE PERITONEUM.

This is dependent upon infection by means of the bacilli circulating with the blood, or upon extension of tuberculous inflammation or ulceration from adjacent organs. Mention has already been made of the fact that the intestines are often invaded by tuberculosis, and that the serosa is quickly involved in such instances. The condition may rarely be primary. This involvement may remain circumscribed and undergo spontaneous cure if the intestinal lesion cicatrizes, as post-mortem findings frequently indicate, but in extensive peritoneal involvement spontaneous resolution is out of the question. These cases may be subdivided into acute and chronic. The *very acute cases* are those forming a part of acute general miliary tuberculosis, or due to perforation into the peritoneal sac from adjacent organs, and Adlebert's classification is as follows: (*a*) the ascitic form, (*b*) the ulcerous form, and (*c*) the fibroid form. Though these groups do not present sharp clinical distinctions, the courses they run vary considerably, as do the results of treatment. In the *ascitic* form the exudate is purulent or sero-purulent, and is often encapsulated. In the *ulcerous* the tuberculous new-formations, which may be quite large, undergo caseation and ulceration, the latter process being progressive, so that it may perforate the walls of the intestines. This and the ascitic variety may be combined.

In the third or *fibroid* form the peritoneal surfaces are adherent. There is little if any exudation; the tubercles may be numerous and diffuse, or may be found only in scattered localized areas, and are often pigmented. The lesions may represent the concluding stage of acute or subacute tuberculous peritonitis.

Etiology.—Most cases are produced by extension of tuberculous inflammation from adjacent organs, and of 107 cases analyzed by Phillips the lungs were involved in 99, the pleura also in 60, and the bowel in 80. Children are frequent victims to intestinal tuberculosis, and the bacilli often reach the peritoneum through the intestines, as they are also apt to do in adults suffering from chronic phthisis. Extension from the pleura to the peritoneum is frequent (pleuro-peritoneal), but from the pericardium is of rare occurrence. In females the starting-point is often the Fallopian tubes, and in either sex it may be the appendix.

Predisposing Factors.—*Age.*—During the period from fifteen to forty years the incidence is most frequent, although it is not uncommon in children under ten years, nor between the fortieth and fiftieth years of life Subsequently, it rapidly decreases in frequency. I agree with Osler i stating that in America negroes are more prone than whites.

Sex has a tolerably potent disposing influence. Abdominal surgeon have taught us that the disease occurs more frequently in females tha males, owing to the fact that the Fallopian tubes are a favorite seat fo primary tuberculous infection. The ratio based upon sex is as 3 to 2 i favor of females.

Symptoms.—Some cases develop *abruptly* with *severe symptoms*, as fever, marked constitutional disturbance, rapid small pulse, abdominal *pain*, *vomiting*, and sometimes *diarrhea*. The *temperature* may be quite high (103° to 104° F.—40° C.), or it may be only slightly elevated even in the worst cases. There follow quickly such symptoms as *anemia*, *marked emaciation*, and a pronounced *typhoid condition*. The signs of *peritoneal effusion* (rarely large) are soon in evidence, and are attended sometimes by a suppurative type of temperature, sweats, etc., indicating the presence of *pus* in the peritoneal sac. A few cases are unattended by ascites, and here nodular masses are palpable, while on auscultation friction-sounds may be audible in the umbilical region. *Tympanites* due to intestinal paresis, is common in cases having an acute onset.

The *acute stage* may be absent, the affection then being marked by slight local and general symptoms (low fever, anemia, slight belly-pains, and a sense of distention). The skin is sometimes pigmented, and usually in patches. There are not a few instances in which the affection is latent, and in one case of this sort with ill-defined general symptoms pigmentation of the skin first directed my attention to the peritoneal condition.

The **physical signs** of moderate ascites frequently, and those of enlarged mesenteric glands sometimes, are present. These conditions are often combined in children, constituting the so-called *tabes mesenterica*. I cannot conceive of the occurrence of this association of symptoms without simultaneous involvement of the peritoneum, and doubtless co-involvement of the latter membrane and intestines usually occurs. The tuberculous new growth in the peritoneum may also form a distinct tumor not unlike that produced by glandular enlargement, while the intestinal coils with their now thickened walls are sometimes knotted together so firmly as to simulate a dense new growth. The exudation may be loculated owing to adhesions between peritoneal layers of the intestinal coils, etc., producing a localized tumor varying in size and position. Such sacculated exudations most frequently occupy the pelvic or umbilical regions, though they may also be found elsewhere in the abdomen. They may be multiple, and are not infrequently too small to be recognized by the physical signs, being often discovered during laparotomy. On the other hand, they may occupy a large portion of the abdomen. An *omental* tumor of characteristic elongated form (produced by a shrinking and curling up of this membrane) is demonstrable, its long axis generally taking a transverse direction just above the umbilicus. Gardiner has observed this tumor to disappear by spontaneous resolution in children.

The dry, fibrous variety, which is not infrequent, is often latent, and the condition may be general or localized. It is decidedly more frequent in adults than in children. The *symptoms* are far from characteristic. Among local features are pains, abdominal distention (giving rise to a tympanitic note on percussion), tenderness on pressure, and sometimes a tumor-ridge extending across the upper abdominal region. Among general symptoms are usually anemia and emaciation, with or without fever. Indeed, the temperature may be subnormal, and these cases may show a tendency to spontaneous recovery.

Diagnosis.—Unless tuberculosis of other organs can be demonstrated the diagnosis is often impossible. This is particularly true in cases in which there is no abdominal pain nor tenderness. Fever and the presence of a tumor, especially if the latter be elongated and lies transversely in the umbilical region, are important aids; but if tuberculosis of the lungs, pleura, pericardium, appendix, and the tubes, in women, can be excluded, the rectal mucus and the urine should be examined for tubercle bacilli. From the *acute form* several affections must be discriminated:

(*a*) *Internal Hernia.*—This comes on suddenly; the pain is strictly localized and paroxysmal; stercoraceous vomiting appears in a few hours; the constipation is absolute, and tympanites is marked, but ascites is absent.

(*b*) Similar symptoms belong to *volvulus* and to the quick incarceration of loops of intestine under bands of adhesions; on comparison they will be seen to differ from those of acute tuberculous peritonitis.

(*c*) *Enteritis* is discriminated *from acute tuberculous peritonitis* by the presence of copious mucous discharges, and by the absence of associated tuberculous lesions, ascites, tumors, and the phenomena of the typhoid state.

Chronic tuberculous peritonitis often closely simulates *cancerous peritonitis*, owing to the fact that the elongated omental tumor may be met with in both, associated with ascites, abdominal pain, and slight fever. In carcinoma, however, there is an absence of the tuberculous history and lesions, and the presence, sometimes, of a gradually increasing tumor of primary growth, the slowly oncoming intestinal obstruction from pressure, and the cancerous cachexia. Moreover, tuberculous peritonitis occurs more commonly in younger subjects, and is more apt to be interrupted by periods of improvement, followed in turn by rather alarming symptoms.

Locular exudations must be distinguished from *ovarian tumors*, and here the history, together with tuberculous lesions elsewhere in the body, the occurrence of febrile attacks, and intestinal disturbance with pain, are of great diagnostic significance. Such cases should be examined by a gynecologist, since, however expert the examiner, when the saccular exudations are located in the pelvic region an exploratory laparotomy must often decide the nature of the condition. Finally, it must not be forgotten that the vast majority of cases of chronic peritonitis are tuberculous.

TUBERCULOSIS OF THE LIVER.

The liver was formerly overlooked in many instances of tuberculosis, because the lesions, particularly in acute tuberculosis, are often microscopic. In the chronic disseminated variety, however, grosser changes are observed, the organ being slightly enlarged, pale, and fatty, and presenting an irregular surface like that of an orange. On section, the parenchyma cuts with great resistance, being very dense (tuberculous cirrhosis). Minute gray and larger yellow masses are seen, especially just under the capsule, and small cavities, the result of a breaking down of the cheesy masses and containing pus and bile, are also observed. These changes are most pronounced about the bile-ducts.

Etiology.—The liver is implicated in all instances of acute miliary tuberculosis. It is also involved secondarily in chronic tuberculosis of the lungs, pleura, peritoneum, spleen, lymphatics, etc.

Symptoms.—This is a common condition, the organ being appreciably enlarged and its surface presenting irregular, palpable prominences. The clinical features of *perihepatitis* and *peritonitis* are often found in combination. Ascites may be present, but is rare.

TUBERCULOSIS OF THE GENITO-URINARY SYSTEM.

(1) **Tuberculosis of the Kidneys.**—This may be primary or secondary, the secondary form being the more common, and it may be either unilateral or bilateral. Infection occurs through the blood in some instances.

Pathology.—The process begins in the calices and apices of the pyramids (papillæ), thence proceeding to the pelvis of the kidney, so that early the condition may be pyonephrosis. The morbid changes then extend to the ureters, and sometimes to the bladder and prostate, and instances are even met with in which the process seems to have crept from below upward, starting from the bladder or prostate. The tubercles pass through the usual stages of caseation, necrosis, and suppuration, and destruction of the renal tissue to a greater or lesser degree occurs, with the formation of cysts containing cheesy material in which lime-salts may be deposited. When the process invades the kidneys through the blood, it may be limited largely to the cortical layer and give rise to nodular tuberculosis with caseous masses, yet with little loss of renal substance. There are not a few cases in which the chief lesion is a tuberculous pyelitis. In the latter class the ureters show extensive involvement, such as thickening of the coats with caseation and ulceration of its mucosa; the bladder, deep urethra, and prostate may also be involved. While it is difficult to judge of the relative ages of the lesions in different organs, I cannot escape the conviction that in this group of cases renal tuberculosis is an *ascending* process and follows uretero-cystic tuberculosis. As before stated, however, most instances are *descending*. Although both kidneys are finally involved in most instances, the lesions are usually much more advanced in one kidney than in the other, and hence for a considerable period the disease is probably unilateral.

Etiology.—Of disposing factors *age* and *sex* deserve especial mention, most cases occurring during middle life, though they are by no means rare both at an earlier and a later period.

Sex.—The disease is much more frequent in males than in females. As stated, the bacilli reach the kidneys with the blood-stream, producing primary renal tuberculosis, but invasion may also take place through the lymphatics or through direct extension from adjacent structures.

Symptoms.—In many cases there are either no renal symptoms or none until a late stage is reached, but the symptoms of pyelitis are usually present. *Pyuria* may be the only symptom for a long time, and this symptom, according to certain authorities, points directly to cystitis. When the latter condition is present, however, the micturition becomes frequent and there is vesical tenesmus. *Pain* in the side chiefly affected is complained of, and is sometimes not unlike renal colic; hematuria is not rare; and a cystoscopic examination may show the blood to be of renal origin (Tuffier). It is useful also in showing the state of the bladder-mucosa. The demonstration of tubercle bacilli in the urine, especially if arranged in S-shaped groups, is diagnostic (Frisch). When the bacilli cannot be found, inoculation-experiments upon guinea-pigs and rabbits furnish an accurate criterion, though it must not be forgotten that tubercle bacilli may find their way into the urine from more distant tuberculous foci. In women catheterization of the ureters may determine which kidney is involved. *Polyuria* is sometimes present, as well as *albuminuria;* the urine may also show *tube-casts* (rarely) and pus-cells. Macroscopic cheesy masses are occasionally found.

The *general features* are often marked, but not until the affection becomes advanced, chills, fever of a suppurative type, emaciation, and increasing debility being the principal symptoms. Associated tuberculous lesions, especially of the lungs, are constantly observed.

Physical Signs Inspection may show a tumor-like prominence on the side chiefly affected, though rarely of large size. Palpation often detects tenderness, and the outline of the organ may be defined by careful firm pressure with the finger-tips.

Diagnosis.—It is difficult to discriminate *calculous pyelitis.* In the latter, however, the pain is severer, the tumor-mass larger, and the hemorrhage more frequent than in tuberculous nephritis. The discovery of tubercle bacilli or the demonstration of tuberculosis of the lungs or other organs would remove all doubt. The tuberculin test may be used.

(2) **Tuberculosis of the Ureter and Bladder.**—This is almost always secondary to tuberculous disease of the pelvis of the kidney above, or of the deep urethra, testes, or prostate below. When primary, as rarely happens, the bladder is in most instances invaded last. The *symptoms* are those of chronic cystitis, and in all cases in which no other cause for the latter can be found the primary tuberculous lesion must be sought for and the urine carefully examined for bacilli. The smegma bacillus, sometimes present in normal urine, can be distinguished by decolorizing with absolute alcohol, which will take place in about two minutes, while with the tubercle bacillus a very much longer time is required. Others say this is not sufficient, and that only their methods of culture-growth or inoculation will distinguish them. A catheter specimen should be obtained if possible (Ogden). With the development of ulcerative lesions hemorrhage is apt to arise.

(3) **Tuberculosis of the Vesiculæ Seminales, Prostate, and Testes.**—The prostate gland and testes are frequently invaded in genito-urinary tuberculosis, and the vesiculæ seminales somewhat less frequently. The morbid process leads to the formation of cheesy nodules, which may, though comparatively rarely, disintegrate, causing excavations or perforation. Rarely, the tubercle does not pass through the stage of caseation, but merely shows the presence of numerous embryonic cells.

Etiology.—The condition is usually secondary, but the existence of primary tuberculosis in these organs cannot be denied. Testicular tuberculosis may begin at any period of life, and is of rather frequent occurrence in infants. When it occurs in the latter, it is part of a more general tuberculous infection, and is in many instances undoubtedly congenital. In some cases it may be a late hereditary affection.

Symptoms.—In the *testicle*, tuberculosis, as a rule, induces a painless, protracted orchitis, though when cavernous lesions occur the symptoms are more acute. In *prostatic tuberculosis* the bladder is highly irritable, there is great distress felt in the thigh and groin, and micturition is very painful. Catheterization, particularly if the urethra (as is very rarely the case) is the seat of tuberculous ulceration, causes most excruciating suffering, and there may be signs of stricture. *Rectal palpation* detects in the prostate firm nodules varying in size from a pea to a bean, together with enlargement of the organ.

Diagnosis.—The diagnosis of tuberculosis of the prostate is easily made from the vesical symptoms, the presence of tuberculosis in other organs, the result of rectal examination, and the detection of bacilli in the urine. Syphilitic involvement of the testicle is sometimes excluded with difficulty; in the latter disease, however, the surface of the swollen organ presents greater irregularities, and is even less painful than in tuberculosis. The absence of the history of syphilitic infection and the presence of tuberculosis in other organs, particularly in the uro-genital system, are valuable points in the discrimination.

TUBERCULOSIS OF THE FALLOPIAN TUBES, OVARIES, AND UTERUS.

Tuberculosis of the tubes in women is a not infrequent condition, and may be primary.

Etiology and Pathology.—The tubes, as a result of infiltration, are thick, hard, and bound down by false membrane. Their ends are generally closed, but the intervening portion is dilated, and contains mucus, pus, and cheesy material. A catarrhal salpingitis is generally in association. *Uterine tuberculosis* is rare, and its origin is usually attributable to similar involvement of the tubes.

The disease is most common during the period of greatest sexual activity, but young children may suffer (*vide* literature of Hennig), and in them the ovaries and uterus may be implicated without participation of the tubes, as in cases reported by Gusserow. At any period of life the lesions may be microscopic; they usually, however, excite marked local peritonitis, which may become general, with the development of ascites. The process may extend to the vagina.

Diagnosis.—The age, family history, and signs of the tuberculous diathesis must be noted. The disease does not distinguish itself from other tubal tumors by anything characteristic on bimanual palpation. Cases occur with ascites and also without, and in the latter variety plaque-like thickening of the subperitoneal tissue is an aid to diagnosis. The uterine secretions should be examined for bacilli in all obscure cases. Ashton advises an exploratory incision or puncture and examination of the contents of the peritoneum or tubes for bacilli.

TUBERCULOSIS OF THE MAMMARY GLANDS.

This is rare; the affected glands present fistulæ and ulcers, with induration of the organ and retraction of the nipple. Warden[1] reports the finding of 58 authentic cases in the literature. Nearly 90 per cent. of the patients were females, and most cases developed in the third decennium.

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[1] *Medical Record*, October 1, 1898.

The *symptoms* are sharp and lancinating pains radiating to the arm, and tumor, the latter consisting of one or more nodules. Pyogenic secondary infection, leading to obstinate fistulæ, is common. The axillary glands are often enlarged. A positive *diagnosis* rests crucially upon the finding of the bacilli in the pathologic secretions.

TUBERCULOSIS OF THE BRAIN.

Pathology.—Tuberculosis of the brain occurs in two forms, one of which, acute tubercular meningitis, has been previously described, while the other is a chronic tuberculous infection, usually localized, of the meninges and cortex, and causing meningo-encephalitis. Very rarely the membranes remain intact. The so-called solitary tubercle is an irregularly round mass, varying in size from a small pea to an apple or even larger. It is generally single, though sometimes there are two, and rarely even three, nodules. The tubercle may be imbedded in, and be contiguous with, the brain-substance, or may be separated from the latter by cysts. The peripheral zone is formed largely of connective tissue, is lighter in color (often translucent), and may contain miliary tubercles, while the central portion, which is cheesy as a rule, may liquefy and thus form a small cavity containing a purulent-looking material. They are seen with greatest frequency in the inferior portions of the brain.

The new growths may compress the longitudinal sinus, inducing thrombosis; they may interfere markedly with the circulation, causing cerebral softening; and, finally, they may excite acute tuberculous meningitis. Tuberculosis of other organs is usually found as an associated condition.

Etiology.—The disease occurs with especial frequency in young subjects, and, according to the statistics of Pribram, in about three-fourths of the cases before the fifteenth year. The symptom-picture is identical with that of brain-tumor (*vide* p. 1128).

TUBERCULOSIS OF THE SPINAL CORD.

The lesions are those of solitary tubercle of the brain. It is an extremely rare condition, and almost invariably secondary. (For symptoms, *vide* Spinal Tumor and Meningitis.)

TUBERCULOSIS OF THE HEART.

In acute miliary tuberculosis gray granulations or larger yellow tubercles may be found throughout the tissues of the heart. More frequently than was formerly supposed does cardiac tuberculosis also result from chronic tuberculosis of adjacent organs. Illustrative cases have been reported by Townsend and Waldeyer. Constantin Paul has in these cases observed tubercles in the wall of the left auricle, as well as in the infundibulum of the pulmonary artery in several instances.

Valvular tuberculosis is, I believe, even more common, though few cases have been reported. Londe and Petit in one instance found the heart much affected, and discovered on the mitral valve several vegetations the size of lentils, which showed tubercle bacilli. I have seen two cases—one in which the mitral, and another in which the tricuspid, valve as affected, associated with latent pulmonary and mediastinal tuberculo- s. Doubtless there are cases in which the valve-lesions form the central nd most prominent part in the picture.

TUBERCULOSIS OF THE ARTERIES AND VEINS.

This may arise consequently upon extension of a tuberculous process into the vessel, as in chronic phthisis. It causes infiltration of the arterial wall, resulting in thrombosis, or the vascular tubercles may caseate and soften, thus leading to hemorrhage. In tuberculous meningitis the arterial lesions are conspicuous. The perforation of a vein by an old focus is followed by a distribution to all parts of the body of numerous bacilli and acute miliary tuberculosis. Infection of the arteries may also occur through the blood. Of 1778 cases of pulmonary tuberculosis, thrombosis occurred 19 times, most commonly in the veins of the lower extremities (H. Ruhl and Hierokles).

General Prognosis.—The prognosis is best reached as in other infectious diseases—namely, by taking into account (*a*) the severity of the type of the disease; (*b*) the presence or absence of frequently associated diseases or complications; and (*c*) the numerous circumstances connected with individual patients.

(*a*) **The Severity of the Disease.**—Though there are no accurate criteria, we may judge of the severity of the disease by its progress, by the result of proper treatment, and from certain symptoms. If the fever be high, the prostration marked, and the local lesions rapidly advancing, we may safely infer that the disease is of aggravated type. With these certain other considerations are closely connected—the stage of the affection and the extent of the local lesions. Thus at an early stage the prognosis is more hopeful than at a late period, and, similarly, when the lesions are strictly localized at one apex it is more hopeful than when they have reached the stage of extensive cavity-formation or are bilateral. As already stated, a certain proportion of the cases manifest an inherent tendency to spontaneous arrest or even cure, and this may occur even after the stage of excavation has supervened. Notwithstanding this truth, however, it is well to make in all undoubted instances of the disease a guarded prognosis. A common error is the mistaking of a temporary for a permanent arrest of the tuberculous process, and in the natural history of the affection the fact was emphasized that its course was interrupted by periods of comparative comfort and noticeable improvement, followed by sharp exacerbations.

(*b*) **Associated Diseases and Complications.**—These unfavorably modify the prognosis—rapidity of pulse, marked temperature-variations, a positive diazo-reaction, chronic nephritis, gastric complications, and intestinal and laryngeal involvement. Some of the accidents of the disease may also precipitate a fatal result (*vide* Modes of Death). The sudden appearance of intercurrent acute pneumonia, whether tuberculous or not, is indicative of danger. Other complications presented by the lungs and other organs have been detailed in the Clinical History.

(*c*) **Circumstances connected with Individual Patients.**—(1) A feeble, delicate constitution, either acquired or inherent (tuberculous diathesis), increases the gravity of tuberculosis.

(2) When the general symptoms show marked improvement, and especially if the fever subsides and the patient gains flesh and strength, the outlook at once brightens.

(3) *Hygienic Surroundings.*—When the hygienic regimen under which the patient lives is of the best, the prospect is more hopeful than when it is faulty. A proper diet often decidedly aids favorable progress, while a defective one often turns the scales against recovery. Equally influential for good is a pure atmosphere, while, *per contra*, a vitiated one is most injurious.

(4) *Age.*—In young subjects from five to fifteen years of age tuberculosis often pursues an acute course and the mortality-rate is exceedingly high. Chronic tuberculosis may, however, form a sequel, and under appropriate surroundings may lead to recovery. In chronic phthisis "the younger the patient the shorter the duration." I have frequently observed that patients who give a history of pleurisy or other phthisical manifestations early in life do not bear chronic phthisis well should it develop at a later period. During old age—a time of life at which tuberculosis is not uncommon—pulmonary tuberculosis is usually more or less latent, and, owing to coexistent emphysema and chronic bronchitis, pursues a slow course.[1]

(5) The gravity of tuberculosis may be determined with some degree of accuracy by the use of creasote in gradually ascending doses. Hence this agent has a prognostic value.

TREATMENT OF TUBERCULOSIS.

Prophylaxis.—(1) This embraces thorough and prompt disinfection of the sputum as the best preventive element. To this end the patient must be taught to expectorate at all times into a spittoon or spit-cup which contains a proper disinfectant solution, and when the breaking-down stage has arrived portable flasks (*e. g.* Dettwiler's) containing an antiseptic solution must be worn by the patient, even while out of doors. Afterward the sputum is to be destroyed by boiling or burning and the spit-cup sterilized. The sweat of tuberculous patients should be removed at intervals, and the surface of the body bathed with appropriate antiseptics.

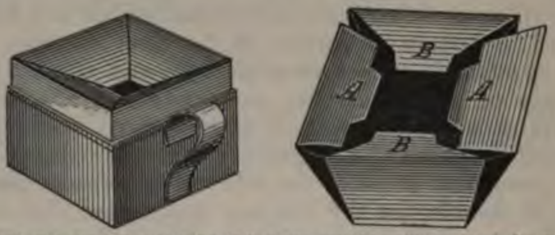

FIG. 28.—Pasteboard spit-cup for receiving infectious sputum. When used the pasteboard can be removed from the steel frame and burned.

(2) **Isolation.**—After the stage of softening is reached the patient should invariably occupy a separate apartment, since, despite great care, the room and bed occupied by the consumptive become in time a source of infection. Hence, unwashable hangings and upholstered furniture,

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[1] A physician should not neglect to examine the sputum in suspicious cases for bacilli.

s well as other objects that facilitate the harboring of the bacilli, should e removed from the sick-room. The floor of the apartment should ot be carpeted, but may be in part covered with rugs that can be frequently taken up and shaken in the open air. For like reasons, special hospitals and sanatoria for the treatment of the tuberculous poor are a necessity. Flügge's important researches (*vide supra*) show that phthisical patients should wear a mask day and night, that should be removed only for eating and to expectorate. Tuberculous patients in the infectious stage of the disease should be retired from occupations in which they may infect others (Flick). Kissing by the patient must be prohibited, and all things used or worn by him should be kept apart from those used by the family or his friends. The prevention of auto-infection, which often results from the swallowing of sputum, is most important.

(3) **Compulsory registration** of tuberculous (pulmonary) patients is desirable. This insures thorough disinfection by health officers of houses in which deaths from phthisis have occurred, and serves to cut off many of the varied channels of transmission of the tubercle bacillus, provided that the measures applied be not rigorous.

(4) **Government Inspection of Dairies and Slaughter-houses.**—This is the serious business of the State, and, since infection through food, especially milk, is quite common in infants, skilled veterinary inspection of dairies is of prime importance. Of the greatest benefit would be the killing of all tuberculous cattle, and of less though decided efficacy the confiscation at the abattoirs of all carcasses that present marked lesions.

(5) The **popularizing of information** relating to the dangers of, and the means of stamping out, this great scourge. This may be in part accomplished by mural placards, stating simple, plain facts about the way in which the disease is spreading. Armaingaud suggests the placing in the homes of the people printed matter in a form suitable for preservation.

(6) **The Removal of Known Predisposition to the Disease.**—The tuberculous diathesis, whether inherited or acquired, must be overcome, if at all, by vigorous measures or by better hygienic living. In attempting to remove the phthisical tendency the physician must place chief reliance upon the most favorable environment attainable. The value of a change of residence—from the city to the country, the seaside, or the mountains, according to circumstances in individual cases—cannot be overestimated. It often renders predisposed persons immune. For some, and particularly young subjects, an equable climate (Southern California or Florida), that will enable them to live an out-door life, is to be preferred. Attention to the food must not be forgotten. Milk is excellent and should be used freely. Daily sponging of the neck and thorax with cold water is beneficial, and appropriate light gymnastics should be instituted if the subject be old enough. In-door occupations are to be forbidden, and the ventilation of living- and bed-rooms must be looked after carefully.

Tuberculosis is apt to develop especially in children while convalescing from acute fevers, and hence during this period the child should be strengthened by vigorous feeding, pure air, and tonics. In children predisposition often results from obstructions in the nose and from persistently enlarged tonsils; and they should be promptly removed. All local foci of tuberculosis in children—glandular, osseous, and articular—must be attacked surgically.

Treatment of the Disease.—The treatment of tuberculosis, regarded as a parasitic disease, presents two leading indications. One has reference to the destruction of the specific cause, the tubercle bacilli, by the use of antiseptic inhalations or of some parasiticide taken internally. Of the numerous substances used by inhalation, few have given satisfactory results, this being largely due to our inability to convey them to the smaller bronchi in a sufficient degree of concentration. They are best adapted to, and most efficacious in, cases in which the larynx is involved.

While the antiseptic treatment, both by inhalation and by means of the introduction into the blood of antiseptic substances, is to be carried out, it accomplishes nothing more than the arrest of the growth and development of the bacilli, and that in an indirect manner. The inhalation of antiseptic substances may be accomplished in various ways—by inhaling vapors, by the use of the steam-atomizer, or by some form of "respiration-inhaler." I have long employed the Robinson inhaler, the sponge of which is moistened with a few drops of a mixture made of equal parts of creasote, chloroform, and alcohol, the patient wearing the inhaler nearly all the time when not eating or sleeping. Unfortunately, most patients object to the constant use of this instrument. The chief among other antiseptics thus employed are carbolic acid, terebene and terpin hydrate, turpentine, thymol, iodoform, oil of peppermint, and a spray of a solution of sulphurous acid. These agents may be variously combined. The injection of antiseptics into the diseased areas in the lungs, as recommended by Pepper, has been for the greater part abandoned. As pointed out by Osler, however, the remarkable results that surgeons have recently obtained in the treatment of joint-tuberculosis by injections of iodoform point to this as a remedy which will probably prove of service when injected directly into the lungs.

The most common, because least objectionable, mode of introducing this class of substances is by internal administration. According to the results reported from all quarters of the world, creasote thus employed alone enjoys the confidence of the profession; and in common with numerous other observers I have found its continued use to be followed by lessened cough and expectoration, lessened fever, and by a lessening or cessation of the night-sweats, with a gain of strength and weight as the natural consequence. In my own hands its beneficial effects have been manifested at the end of two or three weeks. It must be borne in mind that the dose is to be gradually increased to the point of gastric tolerance, which in my experience usually does not exceed 15 to 20 drops (0.999) three times a day. Larger doses are, however, sometimes tolerated.

Following, in the main, the practice of Trudeau, who has used this drug quite as extensively as any other American physician, after reaching the point of tolerance I gradually reduce the dose to and maintain it at 5 or 6 drops (0.333), three times daily. Among the best vehicles are hot milk, hot water, and diluted alcohol. Recently I have ordered it in capsules, which the patient himself fills at *the time of using*, and have found it a popular and ready mode of administration. When creasote is not well borne by the stomach and its inhalation is seriously objected to by the patient, it may be given by enema, the dose being 20 to 30 drops (1.332), in peptonized milk or mixed with a little egg-white. It has also been employed hypodermically in a 10 per cent. solution in oil of sweet almonds, the dose of which is 1 dram to 1½ drams (4.0–6.0). Lastly, it has in rare instances been employed by inunction.

Guaiacol, particularly in the form of the carbonate, has of late been quite extensively employed in place of creasote, of which it is the chief active principle. It may be administered in pill or capsule, the dose being slightly less than that of creasote. It is well tolerated by the stomach, and is broken up in and absorbed from the intestinal canal.

Among other remedies prescribed for their supposed parasiticidal effect are arsenic, mercuric chlorid, and alcohol, but they are clearly inferior to creasote in this therapeutic rôle.

Tuberculin was at one time supposed to exert a specific influence upon the tuberculous processes, but this view has been largely abandoned. Its chief value, as well as employment at the present day, is as an agent in forming the diagnosis.

I am of the opinion that all antiseptics used internally in this disease have for their chief influence a modification of the soil-conditions on which the growth and multiplication of the bacilli depend. They are, in truth, of great value in fulfilling the second leading indication of treatment, which is to overcome the bodily receptivity for the specific bacillus, or to aid the natural defensive processes in limiting the destructive work of the latter. All forms of tuberculosis, however, may heal spontaneously in any stage, this being especially true of the local varieties so common in children, affecting the lymph-glands, joints, and bones.

A large proportion of pleurisies are tuberculous in nature, and although most sufferers from this disease develop pulmonary tuberculosis later in life, many of them apparently heal without the aid of the physician. This is shown by the old pleuritic lesions that are constantly met with at autopsies in persons dying suddenly of other diseases. Spontaneous recovery is seen oftenest in cases that have not progressed to the stage of cavity-formation. Indeed, in the instances in which vomicæ of considerable size have formed, cicatrization or complete cure is out of the question, though they may become encapsulated (quiescent). The percentage of cases in which encapsulated and obsolete tuberculous lesions have been observed at the post-mortem table in persons dying of all causes differs widely with the statistics of different observers. If we consider the cases that are latent from an early period in life, together with those of all ages after childhood, it is doubtless true that in more than 50 per cent. of the human family the bacilli not only gain entrance into the body, but also effect a lodgement. And, since about 14 per cent. of the deaths from all cases can be ascribed to tuberculosis, it follows that unless the conditions are favorable for the growth and development of the bacilli there is manifested a strong tendency to limitation and healing.

In removing the diathesis medicines are unquestionably of less value than the hygienic treatment, the latter in the widest sense of the term aiming to reinforce Nature's efforts at spontaneous recovery, and embracing four main elements: (1) Climate; (2) Feeding; (3) Special Remedies; (4) Treatment of Leading Symptoms.

(1) **Climate.**—The all-powerful influence of environment has already been pointed out. Experience and observation have shown that certain climates, selected with particular reference not only to the stage of the affection, but more particularly to the individual, stand foremost as suc-

cessful modifying influences of the tissue-soil. In any case of tuberculosis that climate is most suitable in which the patient "feels well, eats well, sleeps well, and gains flesh and strength" (Delafield). Until the patient finds such a climate, or if he finds no single climate to produce these results, he should travel from place to place unless special contra-indications (excessive debility, etc.) exist. If active tuberculosis has existed, the stay in a suitable climate should not be less than two full years; and if the patient receive benefit from a high altitude, he should remain permanently. Cases in which hemoptysis is severe and of frequent occurrence, those complicated with weak hearts, and neurasthenic subjects should not be sent to the higher altitudes.

The climatic requisites for a consumptive are (*a*) pureness, (*b*) equability, and (*c*) abundant sunshine. Less beneficial, though important, are (*d*) dryness and (*e*) altitude.

(*a*) *Pureness.*—This requirement is of paramount importance, and thus is explained the fact that mountain air and that of the virgin forest are so helpful in phthisis. Forests, and particularly pine-groves, favor atmospheric purification, since they generate ozone, which oxidizes the impurities contained in the air.

(*b*) *Equability* has reference to the absence of rapid variations of temperature. On the whole, a relatively low is better than a high temperature, the former being stimulating, and the latter sedative, in effect. It should be pointed out that forests also greatly favor the quality of equability,[1] both as to temperature and relative humidity. They tend to maintain an almost unvarying degree of moisture in their vicinity, thus minimizing the diurnal variations of temperature—a point that is of far greater importance than the question of seasonal variations. Forests intercept and temper the bleak winds of winter, while by their shade and leaf-surfaces they afford a cooler temperature in summer.

(*c*) *Abundance of sunshine* is demanded by the consumptive. The advantages of sunshine are obvious from the observations made by Munn[2] in the year 1892, when in Denver there was sunshine in 62 per cent. of the possible hours during which it could occur. A *dry* atmosphere has advantages, but that dryness is not an essential element is shown by the fact that patients often do well at places having comparatively high relative humidity, such as Florida, Southern Georgia, Southern California, and the resorts on the south coast of England. The *rarefied* atmosphere of high altitudes, on account of its stimulating effect upon the respiratory function, aids in producing good results, but the pulmonary changes induced (enlargement of the air-cells, with augmentation of the size of the chest) make it necessary for patients to remain for the rest of their lives. That it is not an essential factor is shown by the excellent results obtained in the ofttimes purer atmospheres at lower levels.

The essential climatic factors mentioned are found in certain American and European resorts. Of the former, the Adirondack region, Colorado, Arizona, and New Mexico are especially to be mentioned, combining as they do in winter a uniform cold, much sunshine, and purity of atmosphere. A camp- or tent-life in the open air cannot be too strongly advocated. According to my own experience, the Adirondacks meet the indications best in early cases or in patients who have strength enough to lead an outdoor life, and in whom the breaking-down stage is not too far advanced. Some cases, in the early stage, also do well at Thomasville, Ga., Southern California, and at Lakewood, New Jersey. Some of the latter resorts possess the added advantage of affording an opportunity of gaining a livelihood. Among foreign resorts, Davos possesses about the same advantages as may be met in Colorado, New Mexico, and the Adirondacks, while the resorts in Southern Italy and France are comparable to Southern California, Southern Georgia, Florida, and the Bermudas in this hemisphere. Good culinary and home comforts are considerations of little less importance than the climate.

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[1] *House-plants as Sanitary Agents; Sanitary Influence of Forest Growth*, p. 312, by the author.

[2] *Medical News*, Aug. 18, 1894.

Briefly, the atmosphere of forest resorts possesses certain unmistakable advantages for this group of sufferers. Hence they should be sent into the neighborhood of the nearest forest in mild latitude (if they cannot enjoy the advantages of more remote resorts), where reasonably good food and other comforts of life are obtainable. In this connection the superior value of the highly ozonized and terebinthinized atmosphere of the pine-groves cannot be too strongly emphasized. It is especially serviceable in cases in which the laryngeal or bronchial element is marked.

Sanatorium Treatment.—Whilst it is essential to send patients to suitable resorts, the most satisfactory results are obtained from the combined climatic and sanatorium treatment. Sanatoria are warmly advocated by Trudeau, Knopf, Bowditch, and others. As elsewhere stated: If properly situated, properly officered and well-equipped, they show results that surpass all other known methods of treatment in the earlier or incipient stages of the disease. These sanatoria should take the form of cottages and pavilions. The principal advantages offered are due to a rigid system of hygiene under the close supervision of competent medical officers. There are *three groups of cases* among the middle and lower classes that require institutional treatment:

Group I.—The numerous cases that have progressed to an advanced and practically hopeless stage. These require every comfort and kind care, such as can be furnished by *special hospitals* for consumption in a healthful urban locality.

Group II.—Incipient cases among the pauper element. For such, sanatoria conveniently located in close proximity to large municipalities, though with special reference to such factors as purity of atmosphere and protection from chilly blasts, by natural elevations or the woodland, should be provided. It is not possible to secure for them the most salutary climates.

Group III.—Phthisis pulmonalis among the middle and working class, or persons having small means The members of this group will find themselves compelled to depend principally upon private philanthropy, and probably to some extent also upon semi-State institutions; they need sanatorium treatment in the best climates, and there is no valid reason why the combined sanatorium and climatic treatment should not be attempted, since such an undertaking could be made to be almost self-sustaining.[1]

Among *home sanatoria* are the Adirondack Cottage Sanatorium, the Sharon Sanitarium, near Boston, the Loomis Sanatarium, at Liberty, N. Y., and the Winyah Sanitarium, at Asheville, N. C. *Foreign sano-*

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[1] "Sanatoria and Special Hospitals for the Poor Consumptive and Persons with Slight Means," by the Author.

toria are to be found at Falkenstein, near Frankfort-on-the-Main, Goerbersdorf, and Hohenhonnef. Solaria, in connection with city hospitals for advanced cases, would, I am certain, yield gratifying results.

Home sanatoria can be readily improvised by stocking living apartments with growing plants. The beneficial influences arising from the presence of the latter are ascribable to two functions—the generation of ozone and transpiration.[1] Here should be mentioned the so-called *open-air treatment* of phthisis. It is of inestimable value to patients who must perforce be treated at home. They should be submitted to a *regimen* similar to that of a sanitorium, and constantly kept in the open air, and for the more part at rest.

(2) **Feeding.**—The diet should be both nutritious and generous. Too close attention cannot be bestowed upon the feeding. Above all, when the remedies prescribed (cod-liver oil, creasote) embarrass in the slightest degree the function of the stomach they must be stopped.

Such albuminous articles as milk, eggs, flesh, fish and fowl, together with an abundance of fats, should be taken. The hydrocarbons are urgently needed, but they must be taken with care lest they derange the digestive function. An early breakfast in bed, including a pint of milk "with an egg stirred into it," is often advisable. Otis asserts that the milk should be sterilized, as there is no more reason why one should use raw milk any more than raw meat. The appetite is often poor or even lost. When this is the case the patient should keep in the open air or try a brief change of air by going, to rural districts or the seashore. It is, however, generally needful to resort to a rigid system of feeding, giving a small quantity of food, such as milk, meat-juice, egg-white, and the like, at brief intervals. The French method of forced feeding deserves a trial if there be absolute loathing for food. It consists of first washing out the stomach with cold water, and then introducing the following mixture thrice daily: 1 liter of milk, an egg, and 100 grams of very finely powdered meat. As a rule, the patient cannot be induced to swallow this, and it then must be poured through a stomach-tube. When the temperature is above 100° F. (37.7° C.) the patient should be kept at rest. In a minority of the cases the appetite is ordinarily keen, often as a result of change of air, and these usually pursue a favorable course. The following combination will be found useful in assisting the appetite:

℞.	Sodii bicarb.,	ʒiss	(6.0);
	Tr. nucis vomicæ,	fʒiiss	(10.0);
	Glycerini,	f℥ss	(15.0);
	Inf. cascarillæ,	q. s. ad f℥iv	(120.0).

Sig. ʒij (8.0) t. i. d., in water, fifteen minutes before meal-time.

Other simple bitters and mineral acids may be tried, and there are many cases in which the judicious use of stimulants, particularly wines and malt liquors, aids the appetite and digestion materially. The chief indications for the exhibition of alcohol are loss of appetite, feeble digestion, and weak, rapid action of the heart. Brandy or whiskey in the form of milk-punch may be given freely in the advanced stage, and more especially during the morning hours. Strychnin is a valuable remedy in the later stages; it should be administered over a long period

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[1] *Loc. cit.*, p. 168.

Lavage has helped some of my cases immensely. Lastly, an orderly method and sound judgment must be brought to bear in arranging the diet and drink.

(3) **Special Remedies.**—These increase the bodily resistance by improving the chief nutritive processes, but do not directly affect the tuberculous lesions; among them cod-liver oil occupies first place. It seems to have more than a mere food-value. It is, however, exceedingly difficult to estimate the therapeutic importance of the oil while other measures —dietetic, hygienic, etc.—are being brought to bear. It sometimes causes further impairment of the appetite and digestion, or sets up intestinal disturbances, and under these circumstances its effects are harmful; on the other hand, when well borne it may be very properly employed. The commencing dose should be small (ʒj—4.0, once or twice daily, to be increased after a time to ʒij—8.0, two or three times daily). It should be taken about meal-time. When the pure oil is not well borne, it may be given in combination with an alkali (lime, soda). Should the oil be found to disagree after it has been used for a considerable length of time, it should be temporarily discontinued. As a substitute for cod-liver oil, cream, preferably Devonshire, may be tried (ʒij to ℥ss—8.0 to 16.0, three times daily).

Serum-therapy.—The definition of this term, as commonly understood, embraces also the employment of toxins and modified toxins produced in various media. These agents, however, are all used for the establishment of artificial immunity, as opposed to the object of the methods of treatment described above—the maintenance of natural immunity. Koch's tuberculin R, Maragliano's serum, and Paquin's antitubercle-serum have all been highly recommended as remedies in tuberculosis by certain clinicians. On the other hand, unfavorable results in cases apparently well suited to the treatment are recorded. A definite decision as to the practical value of this method cannot be reached at present.

The *hypophosphites* are especially serviceable in a certain proportion of the cases. The commencing dose should be 1 dram (4.0), increased to 2 (8.0) if it is well borne.

Arsenic is warmly advocated for its general influence in this disease. The dose should be small, so that it may be given for a long time without interruption. Jacobi speaks highly of digitalis in tuberculosis in children.

Iodoform- or *europhen-inunctions* are warmly commended by Flick, who asserts his belief that incipient cases almost always can be cured in this way, and that advanced cases can be improved.

The *advent of an acute disease* may arrest and cure a tuberculous process. Thus, the symptoms and signs of advanced tuberculosis have disappeared after an attack of virulent small-pox and acute rheumatism (Harris and Beales). Hysteria also exercises an ameliorating effect upon pulmonary tuberculosis, according to the observations of Gibotteau,[1] who advises against treatment of the former disease in tuberculous persons.

Treatment of the Acute Forms.—The treatment of acute tuberculosis is an expectant one. The special measures recommended above should be tried, but are rarely effective, and a change of climate is inadvisable. Supportive measures, such as stimulants and nutritious aliment, are required. The medicinal treatment must be adapted to the acute febrile condition, but all depressants are to be avoided. Special symptoms may be relieved in accordance with the principles laid down for the chronic form (*vide infra*).

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[1] *The Practitioner*, October, 1894.

(4) **Treatment of Leading Symptoms.**—(*a*) *Cough.*—This is often quite annoying. The special cause or causes of the coughing should be determined before any attempt is made to treat it. It may be attributable to catarrhal irritation of the upper air-passages or throat, when it is best treated by topical applications. The following substances may be inhaled: compound tincture of benzoin, combined with paregoric or carbolic acid; creasote, alcohol, and chloroform, in equal parts. For local applications by means of the spray sedatives and narcotics should be preferred, and a solution of cocain is sometimes most efficient. The cause may be found in pleurisy or pleuritic adhesions, and for this condition counter-irritants, as iodin, sinapisms, etc., may be used. Pleuritic coughs often demand codein or even morphin in moderate-sized doses. The cough is in most instances occasioned by the tuberculous bronchitis, and to a lesser extent by the vomicæ. Cough-mixtures are usually prescribed to meet these indications, but as usually formulated they are apt to disorder the digestive function, and in so far as they have this effect they are positively harmful. Syrups should be omitted from their composition. I have come to rely upon creasote by inhalation as the remedy *par excellence* for tuberculous bronchitis, and combine it with spirits of chloroform and alcohol. When expectoration is copious, preparations of terebene, terpin hydrate, and tar may be resorted to; and when the cough becomes sufficiently distressing to urgently demand relief, I employ codein (gr. $\frac{1}{8}$–$\frac{1}{4}$—0.008 to 0.016, every three or four hours) in the form of a granule. In the latter stages morphin is allowable, since it is at this time that constant coughing or severe paroxysms of cough, if not restrained, lead to utter exhaustion. Heroin, in doses of gr. $\frac{1}{2}$ to $\frac{1}{6}$ (0.03–0.01) three or four times a day, acts beneficially in allaying the cough that accompanies phthisis. Stimulant expectorants may be needful, and ammonium carbonate in the infusion of wild-cherry bark is perhaps most efficacious: a few drops of the deodorized tincture of opium or spirits of chloroform may be added.

(*b*) *Fever.*—Creasote has found a new field of usefulness in the treatment of the fever of tuberculosis. In my experience, at all events, the cases in which it has been used, as above indicated, have shown a greatly diminished febrile movement. Cold or tepid spongings of the body at intervals of one, two, or three hours, according to the intensity of the fever, should be tried. Internal antipyretics are rarely advisable, since during the period of high temperature the cardiac action is much enfeebled; but if urgently called for, the following may be employed: acetanilid (dose gr. ij–iij—0.129–0.194), phenacetin (gr. iij–v—0.194–0.324). These are to be administered about two hours before the commencement of the daily rise in temperature, and repeated every three or four hours if necessary. Other antipyretics worthy of trial are the mineral acids and zinc oxid, but not quinin, which has utterly failed in my hands. Keeping the patient at rest when the temperature is above 101° F. (38.3° C.) is good practice, though he should be wheeled into the fresh air for as long a time as possible during the day.

(*c*) *The Night-sweats.*—Among remedies that control the sweats most successfully may be mentioned—atropin (gr. $\frac{1}{120}$–$\frac{1}{60}$—0.0005–0.001);

zinc oxid (gr. ij–v—0.129–324); sulphuric or gallic acid; muscarin (♏iij–vj—0.399 of a 1 per cent. solution); agaricin (gr. $\frac{1}{8}$–$\frac{1}{4}$—0.008–0.016). Sponging with equal parts of alcohol and tincture of belladonna is very effective, but my own best results have been derived from the use of atropin (gr. $\frac{1}{120}$–$\frac{1}{90}$—0.0005–0.0007) in combination with agaricin ($\frac{1}{8}$—0.008).

(*d*) *Dysphagia* may be a troublesome symptom, especially from involvement of the larynx, and it is best met by local applications of a solution of cocain in glycerin and water (gr. x to ℥j–0.648 to 32.0), thrice daily before meals. In advanced cases I have resorted to hypodermic injections of morphin (gr. $\frac{1}{8}$—0.008) before meal-time.

(*e*) *Gastro-intestinal Disturbances.*—In nearly all cases of phthisis dyspeptic symptoms and diarrhea come on sooner or later, and for this gastric disorder nothing is so important as a proper regulation of the diet. Perhaps the medical treatment of the gastric symptoms has been dealt with at sufficient length, save that of vomiting, which may come on after meals and constitute a distressing concomitant. Those remedies giving the best results may be adduced as follows: cerium oxalate (gr. v–viij—0.324–0.518), in capsules before meals; calomel and soda in fractional doses; hydrocyanic acid (♏ij–iij—0.133–0.199); and chipped ice with brandy sprinkled over it, taken at short intervals, but especially shortly before meal-time.

(*f*) *Diarrhea.*—The most important factor in the treatment of this symptoms is a properly-restricted dietary. Alum whey, mutton and chicken essence are of service, but curds of milk, beef-tea, and solids are not suitable. Of the numerous medical measures that have been employed, the most useful are bismuth, acetate of lead, opium, thymol, salol, benzo-naphtol, and naphthalin. To these may be added the following acid diarrhea-mixture, each dose containing—

℞ Acid. acetici dil.	♏x	(0.666);
Morphinæ acetat.,	gr. $\frac{1}{8}$	(0.008);
Plumbi acetat.,	gr. j–ij	(0.0648–0.1296).

Complications when they arise must be dealt with according to accepted therapeutic principles.

SYPHILIS.

Definition.—A chronic infectious disease communicable from person to person by direct or indirect contact with a specific virus, or by heredity. According to its clinical course, it is characterized by six periods: (1) Period of primary incubation—the time which elapses between contact with the poison and the appearance of the chancre. (2) Period of primary symptoms (the chancre). (3) Period of secondary incubation—the time which elapses between the appearance of the chancre and the development of the skin eruptions. (4) Period of secondary symptoms (the skin eruptions). (5) Intermediary period characterized by the absence of lesions, although evidences of existing dyscrasia can still be found. (6) Period of tertiary symptoms. The congenital form of the disease is transmitted at the time of procreation, either by the sperm virile or by the ovum. Prince Morrow[1] points out that the important lesions of the disease are those that occur in the internal organs—*visceral syphilis.*

General Pathology.—(*a*) **Primary Lesion or Chancre.**—This appears at the site of inoculation, and is characterized by infiltration of the connective tissue chiefly with round cells, and also by larger epithelioid and giant cells. This is followed by a shallow ulcer of the size of a split pea or larger, of which the base is quite hard, the sclerosis being due partly to cellular infiltration and partly to a marked thickening of the adventitia of the arterioles and venules. Adjacent lymphadenitis develops and soon becomes general.

(*b*) **Secondary Lesions.**—Mucous patches are the most common. Their favorite sites are the points at which the mucous membrane and the skin are continuous (mouth, anus, etc.); their contour is more or less rounded or oval; their surface readily abraded and usually ulcerated; and their size varies from that of a pin's head to an inch or more in diameter. Like the Hunterian chancre, their periphery is more or less indurated. Secondary lesions also include skin and ocular lesions.

(*c*) **Tertiary Lesions.**—These are circumscribed inflammatory products, the result of the blocking up of the lymph-spaces by the broken-down round-celled infiltration of the secondary period (gummata), appearing in the connective tissue, bones, periosteum ("nodes"), skin, muscles, brain, liver, lungs, kidneys, heart, testes, etc. The gummata, though usually sharply circumscribed, may take the form of diffuse infiltrations of the affected parts, and vary greatly in size—from a pin's point to a hen's egg. Usually firm, they may be soft, and tend to form ulcers. Their color is grayish, and on section they show a caseous semiopaque center, with a fibrous, translucent periphery.

Microscopically, the gummata consist of small round cells. The mass thus formed may either be absorbed or persist; but in most instances coagulation-necrosis occurs in the center, due to local anemia, and there is a conversion of the peripheral zone into fibrous tissue. The gummata are enveloped in granulations and connective tissue, which contracts, forming cicatrices that often contain calcareous masses on their interior. The lesions of certain structures (skin, mucous membranes, bones, and cartilages) often lead to destructive ulceration and sloughing.

General Etiology.—**Bacteriology.**—Lustgarten in 1884 described the bacillus of syphilis, which closely simulates that of tuberculosis and the smegma bacillus. It occurs in rod-shaped or curved forms with slightly enlarged ends, and is from 3 to 5 μ long. From the smegma bacillus it is distinguishable by the carbol-fuchsin test; though demonstrable with difficulty in the hard chancre and in secondary lesions, its pathogenic potency is still very doubtful. Inoculation-experiments upon lower animals are not possible, since man alone is susceptible. Van Niessen[2] claims to have discovered a syphilis bacillus, although his results are unconfirmed by other observers.

Predisposing Causes.—Since acquired syphilis originates only by inoculation, it is obvious that a break in the cutaneous or mucous surfaces is essential to infection, such as a slight abrasion, fissure, or laceration, etc., particularly of the genital mucosæ. Other surfaces may also be the seat of infection, as the lips, hands, etc.

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[1] *Med. News*, March 23, 1901.

[2] *Centralb. f. Bakt. und Parasitenk.*, Bd. xxiii., No. 2, January, 1898, p. 49.

Susceptibility to the virus is universal, and no age is exempt. *Reinfection* is exceedingly rare, but does occur.

Contagion of Syphilis.—The blood of a syphilitic during the secondary period, and the secretion from the chancre or any of the secondary lesions, are contagious, the lesion at the point of ulceration always being a chancre. The physiological secretions, saliva, sweat, milk, and semen, do not convey the virus, unless contaminated by admixture with discharge from some of the lesions of the primary or secondary stage. The semen, while apparently not conveying the poison, is able to infect the embryo, and in turn the mother.

Modes of Infection.—(1) In a great proportion of the cases (about 70 per cent.) syphilis is transferred by illicit sexual intercourse.

(2) *Accidental Inoculation.*—This is not uncommon. (*a*) Most frequently it is accomplished through the pernicious custom of indiscriminate kissing (lip-chancre), and I have personal knowledge of not less than 8 instances in which infection has occurred through labial contact.

(*b*) The site of inoculation may also be the mouth and tonsils, the virus being conveyed during the low practices of sexual perverts. The wet-nurse may infect the mouths of suckling babes, or, *vice versâ*, the infant may infect the nipple of the nurse.

(*c*) The obstetric finger may become infected. Three instances of the sort have come under my own observation, and Fournier gives the details of 40 cases of primary syphilitic infection of the hand. In 30 of these the malady was acquired in medical practice (4 obstetricians, 20 general practitioners, 3 students, and 3 midwives).

(*d*) Humanized vaccine virus may rarely transmit the disease.

(*e*) Accidental infection has, though very rarely, taken place in a variety of other ways—*e. g.* handling foul rags from the hospital ward, by bed-clothing, drinking-cups, the pipe, and cigar.

Kraft-Ebing found that out of 3455 cases treated at the University of Christiania from 1867 to 1894, $15\frac{6}{10}$ per cent. were of extra-genital origin. The lesion was upon the lips in 51 per cent. of the cases.

(3) *Hereditary Transmission.*—Paternal transmission (through the semen) is much more common than is maternal, the period of greatest danger being immediately after the father has become infected or during the time of the secondary manifestations. The first-born, if the father be syphilitic, is apt to show well-marked lesions. Appropriate treatment of a syphilitic parent lessens the danger of transmission very materially, however, and in such instances there is little tendency to transmission shown after the third year. On the other hand, a syphilitic father or mother may beget healthy offspring, the infant having acquired some immunity which protects it from its mother (Profeta's law). Syphilitic children are also common to infected women. In the majority of instances of hereditary transmission, however, both parents are syphilitic, and under these circumstances the liability to infect the offspring is much augmented. A woman who has become infected after conception may bear a syphilitic child; though the latter may, on the other hand, escape infection.

Allusion may here be made to Colles's law—that a woman who bears a syphilitic child enjoys, owing to a sort of protective vaccination with the specific virus, perfect immunity, and this in the absence of all signs of the affection. Coutts[1] dissents from this opinion.

Clinical History of Acquired Syphilis.—(*a*) **Primary Stage.**—The typical *initial lesion* (chancre) appears about three weeks after infection, and is followed soon by swelling and induration of the nearest lymphatic glands. The primary sore begins as a *red papule*, which rapidly reaches its maximum, and then undergoes a central necrosis with the formation of a *small ulcer*. The adjacent structures become hard or cartilage-like—a characteristic to which the lesion owes its name of "hard chancre." A small chancre may often escape detection, especially if it be situated inside the meatus. When situated upon a mucous membrane it is always a *chancrous erosion*, which may be so mild and of such brief existence as to come and go without the knowledge of its bearer. Particularly is this the case in the female. The *general symptoms* are negative in this stage.

(*b*) **Secondary Stage.**—This is announced about six weeks after the appearance of the infecting chancre by *moderate fever* (101°–104° F. —38.3°–40° C.), accompanied by languor, headache, bone-pains, impaired digestion, and a slight degree of prostration. The patient, as a general rule, shows signs of anemia (syphilitic cachexia), and angina, with hyperemia of the fauces and hard palate, now appears. There may be minute elevated white patches upon the pharynx; the tonsils may enlarge, and on both shallow ulcers presenting a grayish sharply-defined border appear. They neither excite pain nor spread.

There is also an *eruption*, which is at first usually *roseolar* and widely distributed, coming out abundantly upon the trunk (especially the chest), buttocks, thighs, and forehead. Another early eruption is the *papular*. The papules are small, hard, and do not ulcerate, while their favorite seats are the scalp, chest, and dorsum of the tongue. The distribution of these early syphilids is symmetric; their outlines are rounded; their color like that of a slice of raw ham ("coppery"); they are polymorphous; and, as a rule, they excite neither pain nor itching.

Other and later-appearing eruptions may be *squamous*, *vesiculo-papular*, *pustular*, and *tubercular*. These show a tendency to bunch in certain areas, and hence are less diffuse than the afore-mentioned eruptions. Several sub-varieties, however, may appear simultaneously.

The visible mucous membranes (angles of the mouth, tongue, gums, pharynx, vulva, vagina, penis, and around the anus) and the skin may show painful condylomata or mucous patches, and especially is this the case in the mouth, where they often stubbornly resist treatment. *Recurrences* at varying and ever-decreasing intervals are quite common.

Other frequent symptomatic conditions arise during this secondary period, such as iritis, laryngitis (frequently), choroiditis, retinitis, epididymitis (more rarely), and alopecia. The hairs of the eylids and eyebrows may fall off and the finger-nails become brittle.

The *secondary* symptoms last from two to three months (the usual duration) to a year or more, and are followed by a period of apparent good health lasting for an exceedingly variable interval (from a few months to many years) before the tertiary stage sets in. During the secondary stage the symptoms may be severe, mild, or even absent. The severity of an attack of syphilis depends upon the *dose* of infecting virus on the one hand, and upon the condition (both local and general) of the vital functions on the other. This fact explains why a single organ or system, as the brain and cord, is attacked in one instance and some other organ or system in another, and the effect of traumatism in determining the topography of periosteal "nodes" is a good example.

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[1] "Hunterian Lectures," *Lancet*, 1896, No. 3889.

(c) **Tertiary Stage.**—As I have already stated, the secondary period is generally followed by a variable interval of freedom from symptoms, but to this rule there are numerous exceptions, and among not uncommon occurrences may be witnessed the appearance of tertiary symptoms during the secondary stage. Belonging to the third stage are certain skin-eruptions, especially the characteristic *rupia*, which first appears in the form of pustules that break and form ulcers that are covered with dry, laminated crusts "like an oyster-shell." To this stage also belongs *psoriasis*, especially of the hands and feet. *Pustules* (*tubercular*) which do not scale over also appear. These eruptions involve the true skin, and in healing leave scars, but, unlike the secondary cutaneous lesions, they are neither infectious nor contagious, are not, as a rule, symmetric, and are more liable to be attended by itching. True gummata may develop in the skin and subcutaneous tissue, and these break down and form kidney-shaped ulcers which tend to spread in a serpiginous manner. On healing (a process that is accomplished with difficulty), scars result. Gummata may occur in the mucous membranes, and pass through the stages of ulceration and cicatrization. When situated in the larynx or trachea their healing is attended with narrowing of the organ, and when in the lower bowel or the rectum dysenteric symptoms, followed by actual stenosis, may result.

In the *muscles* gummata occur and form small hard tumors. They may also cause periostitis and death of the bones, especially of the nose, palate, and skull; "nodes" are thus formed, which are situated chiefly upon the tibia and the skull in larger or smaller numbers, and also, though less frequently, upon other bones. These are exceedingly painful, particularly at night, and are very tender under the pressing finger. They may be true gummata, but more often, if not absorbed, they either become ossified or undergo fibroid change, while in rarer cases they suppurate. Chronic enlargement of the lymphatics and of the testicle, with little tendency to suppuration, may be noticed. The pregnant female is apt to abort or miscarry, either as the result of the action of the syphilitic virus upon the ovum or of the presence of gummatous growths in the placenta.

Gummata also occur in the internal organs (*visceral syphilis*), and of the latter I shall speak presently, taking up separately some of the various organs and systems of the body. *Amyloid degeneration* is frequently caused by the acquired form, particularly syphilis of the rectum in women, but very rarely by the congenital.

Malignant Syphilis.—By this term is meant a virulent and a fatal form of the malady, which is fortunately rare. The various stages manifest themselves early, and especially the tertiary, as on the forty-fifth day in a case of Mauriac. The course is rapid and the condition resists all forms of treatment. Roussel narrates a case in which death occurred about one year after the commencement of the disease.

Clinical Symptoms of Congenital Syphilis.—These may, though rarely, be identical with those of acquired syphilis, if we except the chancre.[1] Occasionally the characteristic symptoms are present at birth. On the other hand, in the vast majority of instances they appear between the first and fourth months of life (*infra*). The symptoms of *inherited syphilis* may be grouped according to the time of appearance:

(1) **In the New-born.**—There is a lack of physical development. The babe may be *greatly emaciated*, it has snuffles, and singultus occasionally sets in soon after birth. Skin-eruptions are rare, except *pemphigus neonatorum*, which appears as bullæ on the palms and soles; among exceptional cutaneous phenomena are gummata around the radio-carpal articulations, palmar psoriasis, and a fleeting roseola. Ulcers and fissures (*rhagades*) may be noticed around the outlets of the body (mouth, anus, etc.); the osseous system may show hyperostoses of the long bones; and the liver and spleen are enlarged. Comby reports 8 cases of pseudo-paralysis due to syphilis in the new-born.

(2) **Early Post-natal Symptoms.**—Most subjects of syphilis hereditaria are born *plump* and *without taint*. Romiceano[2] gives the results of his observations of 723 cases of infantile syphilis in which the disease appeared chiefly between the first and fifth months, and only 27 times in all after the sixth month. Rogers's statistics show that among 249 cases, 217 showed symptoms before the end of the third month. The first symptom is generally *coryza* (syphilitic rhinitis), which is betrayed by a sero-purulent or bloody discharge and a peculiar form of *obstructed breathing* (snuffles), rendering nursing difficult. The coryza may in some cases be preceded by singultus lasting ten or twenty days (Carini), and ulcers may form in the nose, leading to necrosis of the bones and producing at last a sunken and deformed nose which is highly significant. The coryza may extend to the middle ear and cause otitis media, with deafness and otorrhea as the chief symptoms. The *skull* may approach the natiform in shape, and the signs of diaphyso-epiphyseal inflammation develop.

The *cutaneous symptoms* appear early. The skin has a tawny hue, and an erythematous eruption of the nates and genitals is frequently seen; this is patchy, with well-defined margins, and has the characteristic coppery color. In the same localities papules may appear, while pemphigus may attack the palms and soles. Syphilitic onychia may be present, and the lips and angles of the mouth often show fissures that are of real diagnostic worth. Other symptoms are ulcerations of the skin and mucous surfaces, falling of the hair, and a moderate glandular enlargement.

Enlargement of the spleen is a frequent characteristic symptom, and White says that the enlargement of the organ when "painless, subacute, persistent, often preceding the eruptions, should be included in the list of significant symptoms."

Swelling of the liver may also be present, but is of little diagnostic import. Syphilitic infants occasionally manifest a hemorrhagic tendency.

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[1] With prenatal syphilis we are not concerned.

[2] *La Progrès médicale*, Paris.

At birth bleeding from the umbilicus may occur; later, into the subcutaneous tissue and from the mucous membranes (gastro-intestinal, vaginal, nasal, etc.). Hecker[1] considers an examination of the umbilical cord important for the early recognition of syphilis in the offspring of syphilitic parents; if the microscope shows characteristic changes, time may be gained for treatment; "these changes range from a decided endarteritis or periarteritis or phlebitis to a simple round-celled infiltration of the blood-vessel walls or the surrounding tissue." As pointed out by Osler, these cases must not be confounded with Winckel's disease.

Among *nervous symptoms*, restlessness, sleeplessness, and a harsh, shrill cry which may be almost constant for days together and due most probably to darting pains, are the chief. Anemia and other evidences of syphilitic cachexia soon supervene.

(3) **Late Symptoms.**—The symptoms of syphilis hereditaria tarda may be arranged in groups (Fournier):

(1) *Those Indicated by the General Appearance.*—There is a retarded general development, as shown by the small stature, undeveloped muscles, the graceful form, and infantile appearance at ages varying from four to twelve or more years. The skin has an earthen tint, and the hair may be scanty and late in its appearance on the face and genitals.

(2) *Skin-cicatrices.*—Cutaneous scars, particularly if multiple and extending over a circumscribed area, are important diagnostic signs. Their form is usually round or serpiginous, and their chief location the mouth, nose, soft palate, and lumbo-gluteal regions.

(3) *Lesions of the Skeleton.*—The natiform skull, "with a transverse enlargement, lateral bulgings, and the flattening in the middle," is almost pathognomonic. Asymmetric and hydrocephalic skulls are also to be considered, in many cases, as signs of hereditary syphilis, as is a sunken and deformed nose. The thickened, "sabre-shaped" tibia, due to gummatous periostitis, is capital evidence of the disease, while the chicken-breasted thorax is significant.

(4) The *testicles* show an arrest in development (infantile testicles). This is a sclerotic atrophy.

(5) *Hutchinson's triad*, under which title come (*a*) the Hutchinson teeth; (*b*) ear-conditions; and (*c*) affections of the eye.

(*a*) *The Hutchinson Teeth.*—The teeth may be late in appearing, and the dental arch may be malformed, the teeth presenting various irregularities in form and condition (dental dystrophy).

The incisors, especially the superior median of the second dentition, are notched, and show a thinness of the free edge, an atrophy of the summit, and crescent-shaped erosions. Fournier[2] calls attention to the absence of one, two, or more teeth in a great number of cases.

(*b*) *Ear-conditions.*—Otorrhea, secondary to naso-pharyngeal catarrh, has already been mentioned, and, in addition, at or about the time of puberty an incurable form of deafness may develop speedily, without the presence of pathologic lesions to explain the same.

(*c*) *Affections of the Eye.*—These are interstitial keratitis and iritis, affecting both eyes successively.

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[1] *Jahrb. f. Kinderh.*, Bd. li., Heft 3.

[2] *Gaz. hebdom. de méd. et de chir.*, January 18, 1900.

VISCERAL SYPHILIS.

Syphilis of the Brain and Cord.—Pathology.—The most characteristic and not infrequent lesions are the syphilitic new-growths. Their size varies from that of a bean to that of a chestnut, and they present irregular contours. They are usually situated either in the cerebral hemispheres or on the pons, and rather superficially, connecting directly or indirectly with the dura or pia mater. They may not infrequently originate in the dura mater. In gummata of average size a cut-section shows caseation in spots which are connected and surrounded by firm, translucent, gray or reddish-gray, fibrous tissue; and, according to Gowers, the more irregular surfaces and the irregular caseation serve as important distinctions from tuberculous tumors. When, as is usual, the gummata touch the membranes, meningitis—subacute or chronic, with much thickening—is combined.

As I have said, the condition may begin as a gummatous meningitis, while in fewer instances it may start as a gummatous arteritis. On the other hand, a gumma may secondarily involve a blood-vessel for a considerable distance, weakening its walls, with resulting rupture and intracranial hemorrhage; or it may bring about cerebral thrombosis.

Histologically "the cerebral gumma differs from other similar bodies chiefly in the presence of very large spider-like cells containing an exaggerated nucleus and a granular protoplasm which extends into the multiple, branching, rigid prolongations" (Wood). The arteries, particularly those of the base, may show syphilitic sclerosis; this renders them thick, hard, opaque-whitish, until their lumen is well-nigh obliterated.

Gummatous growths may attack the cord. In a case recently reported by Osler a new-growth occupied the cord opposite the root of the third cervical nerve. The other gross changes found in connection with cerebral gummata and their secondary lesions (softening, collateral inflammation) are also observed in syphilis of the cord.

Etiology.—Cerebral syphilis is usually a late (tertiary) manifestation—appearing from one to thirty years after primary infection—"in middle life" (Rothwell). Lydston and others have shown that nervous stigmata may become evident during the secondary stage, even as early as three months after initial infection. It oftenest develops in cases in which the secondary symptoms have been slight, and may occur in those in which both primary and secondary manifestations have been entirely overlooked. Inherited syphilis affects the nervous system less frequently than does the acquired form, but cerebral gummata have been noted at all periods from the time of birth until after puberty.

Symptomatology.—*Imbecility* and *idiocy* may be due to inherited syphilis, but they are probably too often attributed to this cause. The other features simulate those of the acquired form.

The symptoms of the acquired form are with few exceptions referable to three affections: (*a*) epilepsy, (*b*) brain-tumor, and (*c*) paralysis.

(*a*) *Epilepsy* coming on after the twenty-fifth year, not dependent upon alcohol nor uremia, is usually due to the ravages of syphilis, and a careful search for traces of scars and of the entire body-surface for bone-lesions, etc. should be instituted. The appearance of the disease may be preceded by psychic disturbance, headache, dizziness, and loss of memory. Hysteric manifestations may also be presented, being probably provoked by the specific lesions. On the other hand, a protracted torpor which may last for a few days or as many weeks may develop. In one of my own cases periods of marked mental excitement, that persisted for three or four days, alternated with periods of almost complete insensibility of about equal duration.

(*b*) *Brain-tumor.*—The symptoms pointing to brain-tumor will be discussed under this head in the section on Nervous Diseases. The syphilitic nature of the cerebral growth cannot be determined with any degree of certainty except in the presence of a clear history of syphilis —congenital or acquired—and the characteristic symptoms or traces of the primary, secondary, or tertiary lesions. In such cases the diagnosis is almost undoubted.

It must be remembered that the secondaries are either sometimes absent or go unnoticed, and if the patient has had a primary sore, the presence of the characteristic symptoms of brain-tumor (headache, optic neuritis, convulsions, etc.) make the existence of specific nerve-lesions highly probable. The chancre may also be overlooked or denied, and it is in such instances as the latter that the occurrence of convulsions in persons over thirty should excite suspicion, and lead to a trial of the antisyphilitic treatment for further confirmation.

(*c*) *Paralysis.*—This may take the form of hemiplegia (due to cerebral hemorrhage or tumor) or of general paralysis (*dementia paralytica*). The relation that these affections bear to syphilis will be indicated in its appropriate place in this work in the description of Nervous Diseases. The fact may here be pointed out that syphilis may induce precisely the same changes met with in general paralysis of the insane.

The history of syphilitic infection, together with symptoms of an *atypical* type of spinal tumor, points to *gumma* of the cord. Syphilitic myelitis usually develops within five years after infection, and may pursue an acute or subacute course, though oftener it takes the form of chronic myelitis. The latter attacks by preference the lumbo-dorsal section of the cord—a fact corroborated by the character of the symptoms. The clinical features, however, are not distinctively syphilitic; and the process is uninfluenced by the most vigorous antisyphilitic measures. When the etiologic influence of syphilis can be shown, especially in the absence of other causes, the diagnosis of syphilitic myelitis rests upon more certain ground. Acute syphilitic myelitis gives an unfavorable prognosis.

General Diagnosis.—The onset in nervous syphilis may be acute or subacute, and the symptom-complex embraces a multiplicity of phenomena, there being an especially erratic distribution of the ocular and other attending palsies and early marked impairment of the mind.

SYPHILIS OF THE LIVER.

In my experience the liver, with comparative frequency, bears the stress of visceral syphilis.

Pathology.—The lesions may be thus classified: (*a*) **Diffuse Syphilitic Hepatitis.**—This is met with chiefly in congenital cases, though I have seen an instance in an adult who died of cerebral hemorrhage, the occurrence of which in adult life has been questioned by some. The liver is uniformly enlarged, firm, and resists the cutting knife. Its color is grayish-yellow.

The microscope shows a marked increase in the connective tissue and a cell-infiltration throughout. From intense, focal cellular infiltration miliary gummata may result; these undergo contraction, diminishing somewhat the size and altering the shape of the organ.

(*b*) **Gummata.**—These may be seen in congenital cases (chiefly the miliary gummata). As seen in the adult, hepatic gummata are disseminated nodules, with the usual central, cheesy mass surrounded by a zone of grayish fibrous tissue and varying in size from a hazelnut to an apple. They form separate tumors, whose favorite seats are the convex surface of the organ, especially near to the suspensory ligament, and in the region of the portal vessels. They are usually tertiary lesions, and do not appear until a number of years (two, three, or four) after infection. These so-called syphilomata in the advanced stage contract, and the liver will be found smaller than the normal. Deep furrows due to contracting fibrous bands traverse the organ in different directions and divide it into lobes of various dimensions. Gummata frequently undergo fibroid change, but more rarely they soften and liquefy (Wilks). On the other hand, before contraction occurs the liver is increased in size and the gummata form protuberances on its surface.

(*c*) **Gummatous Arteritis.**—Briefly, this may affect both the portal vein and hepatic artery, though syphilitic endarteritis is situated chiefly in the smaller branches of the latter.

(*d*) **Perihepatitis.**—Here Glisson's capsule is thickened, owing to augmentation of its connective-tissue elements. From the latter there dip into the hepatic tissue cicatricial bands, particularly along the portal canals, which may change somewhat the shape of the organ. Section shows admirably the pale scar-like tissue (*vide* Diseases of the Liver).

Clinical History.—The affection may exist without symptoms. In the *congenital form*, however, we have signs of hepatic enlargement, with icterus, the spleen being likewise large and firm, as a rule. The history and associated lesions are necessary to a certain diagnosis.

In the *adult* syphilis of the liver does not usually attract attention until the gummata interfere with the portal circulation. As they undergo contraction they tend to occlude some of the portal branches, or they may, on account of their situation, exert pressure upon the vena porta itself. In either event the evidences (ascites and splenic enlargement) of portal obstruction will develop as in alcoholic cirrhosis. The gastro-intestinal symptoms common to the latter disorder are also present, and obstructive jaundice may supervene, though it is, comparatively speaking, rare. Pain, usually localized to some particular spot over the right hypochondrium, is sometimes complained of, and may be quite severe, while pressure over the painful area elicits great tenderness.

Physical Examination.—In the early stage, while the organ is enlarged, flattened, irregular protuberances may be detected by the palpating fingers. At a more advanced period ascites may interfere with palpation, and in such cases an aspiration of the fluid will enable one to feel the syphilomata. Finally, in the stage of contraction the results of palpation are obviously negative.

There is a group of cases in which the clinical picture is that of *advanced amyloid disease* of the viscera. The liver and spleen are enlarged, the urine is increased in amount and contains albumin and tube-casts, and finally dropsy supervenes.

Diagnosis.—This rests upon the etiology, the presence of scars in the throat or on the skin-surface, bone-lesions (especially irregularities of the tibial surfaces), or other evidences of the ravages of the disease, and upon moderately good general health. The most important local symptoms are the hemispheric prominences on the surface of the liver and the localized pain.

The diagnosis between syphilitic disease of the liver and *echinococcus-cysts* is sometimes extremely difficult. R. Lennhoff has noted in a number of cases of echinococcus-cyst that on deep inspiration a furrow forms above the tumor, between it and the edge of the ribs.

The clinical findings resemble those of cancer of the organ. I have contrasted the main dissimilar points in the subjoined table:

SYPHILIS OF THE LIVER.	CANCER.
History of heredity or of infection.	History of heredity or of primary growth.
Occurs congenitally, or, if acquired, at any age.	Never congenital. Usually occurs after the age of forty.
Often accompanied by symptoms of tertiary syphilis—alopecia, rupia, etc.	Often preceded by the primary growth in pylorus, uterus, mammary gland.
Jaundice and ascites are common, especially the latter. No cachexia.	Jaundice and ascites are rare. Marked cachexia.
The margin, on palpation, is markedly irregular, and neither nodular nor umbilicated.	Often the margin reveals the presence of umbilicated nodules.
Recovery may follow, or the affection may last for years.	Always fatal. Duration usually from a few months to a year.

The **course** and the *results* of antisyphilitic treatment are of value from a diagnostic view-point. The course is slow and often interrupted, while appropriate treatment may lead to recovery, as in three of my cases.

SYPHILIS OF THE ALIMENTARY TRACT.

The lesions in the mouth have been for the most part considered. In the tongue gummata often develop. A decidedly fissured appearance of the organ and whitish scar-like patches upon the surface may be observed in syphilis, but have no essential connection with that disease. Gummata also appear on the posterior wall of the pharynx and lead to ulceration, which may cause fatal hemorrhage by erosion of adjacent large blood-vessels (internal carotid, etc.). The walls of the esophagus may also be invaded, resulting usually in stenosis.

The stomach-walls may be infiltrated, though they are rarely ulcerated. Einhorn, Fournier, and others, have met gastric ulcer in syphilis; it was cured by the specific treatment. Syphilitic tumor of the stomach may rarely occur; the symptoms are those of malignant growth, resembling cancer, but curable. Syphilitic ulcers may appear in the intestines, and the condition may lead to perforation and peritonitis.

Gummatous infiltration of the rectum is a somewhat frequent, severe, and clinically important affection. It is much more common in women than in men, taking place in the "submucosa above the internal sphincter." It has frequently caused a fatal result in persons who failed to show post-mortem specific lesions in other viscera, and hence it is to be classed as one of the ravages. The result of the gummatous infiltration is the production of a funnel-shaped stenosis of the rectum which narrows from below upward. Above the stenosis, and directly dependent upon it, there is dilatation of the rectum and the descending colon. Here may also be found ulcers—some specific, and others the result of mechanical pressure exerted by the fecal accumulations.

Symptoms.—The clinical features are for the most part those of a gradually induced *stenosis* of the rectum. At first there may be hemorrhages, suggesting internal hemorrhoids. The action of the bowels is irregular, and is followed shortly by a tendency to dysenteric diarrhea, with pains, tenesmus, and scanty stools containing mucus and pus. Prolapse of the rectal mucosa may occur, and, owing to the presence of small hemorrhoids, the true nature of the case may be overlooked. The disease is most distressing, and leads slowly and gradually to extreme emaciation and asthenia. Death may be due to the latter or to some complication (perforative peritonitis, etc.).

Diagnosis.—This may be aided by a clear history of associated syphilitic symptoms or of specific lesions, including amyloid degeneration. A sure demonstration can only be made by rectal examination. The examining finger feels the sharp edge of the cicatricial ring. Cancer of the rectum can readily be eliminated on account of the absence in syphilis of the "crater-like" ulcer.

SYPHILIS OF THE LUNGS.

While undoubted cases occur, syphilis of the lungs is rare indeed.

Pathology.—The cases are pathologically divisible into four forms: (*a*) Gummy tumors; (*b*) Interstitial pneumonia; (*c*) Brown induration; (*d*) Fetal pneumonia.

(*a*) **Gummy Tumors.**—These appear as yellowish-white scattered nodules, varying in size from a cherry-pit to a hen's egg. Their centers are dry and caseous-looking, and their peripheral zones fibrous. They are relatively thicker set near to the root of the lungs. Cicatricial bands may be seen connecting not only the separate nodules, but stretching outward to the thickened pleura. Such growths may undergo softening and ulceration, thus forming a cavity that rarely attains to large measurements; or, on the other hand, in favorable cases the fibroid changes and cicatrization may lead to recovery.

A primary lesion is atrophy of the alveolar walls, with hyaline degeneration of the capillaries (Councilman). Broncho-pneumonia (not distinctively syphilitic) may be associated.

(*b*) **Interstitial Pneumonia.**—This is a fibrous infiltration, showing a predilection for the right lung. Its chief seat is the root of the lung, whence it extends along the bronchi and vessels, and usually involves a part of one or more lobes. Occasionally its starting-point is the pleura, from which the process advances along lines corresponding to the interlobular tissue. Bronchiectasis may be noticed. Gummata may also be associated, or may have been present and been practically obliterated during the process of cicatrization. I have seen an instance in which the merest vestige of gummatous material remained.

(*c*) **Brown induration,** simulating exactly that which is seen in association with organic valvular diseases, may be observed, but it may have no necessary connection with syphilis.

(*d*) **Fetal Pneumonia** (*Virchow's White Hepatization*).—This is peculiar to the new-born, in which miliary gummata first occur, followed by hepatization of large zones or an entire lung. The chief changes are an infiltration of the alveolar walls, while the air-cells are filled with desquamated epithelium; on section the tissue presents a grayish-white appearance.

Symptoms.—From what has just been stated it is clear that a certain limited number of cases present symptoms and signs that simulate ordinary ulcerative phthisis, but do not show bacilli in the sputum, and hence have no connection with genuine phthisis. There is another group of cases in which the picture presented to view is almost identical with that of fibroid induration though usually giving a distinctly syphilitic history. I am not prepared to say that there is an acute syphilitic broncho-pneumonia analogous to acute pneumonic phthisis, though I fail to see any reason why malignant syphilis may not attack the lung and take that form. The symptoms may be too few and too mild to afford ground for suspicion.

Diagnosis.—If a suspected case is treated early and accurately, the result may serve to corroborate the diagnosis, which is at first far from being final.

Bronchiectasis, dependent upon syphilitic peribronchitis or interstitial pneumonia, cannot be discriminated from other forms of that disease except there be a clear history of infection, and unless associated scars or active syphilitic lesions coexist. *Pulmonary tuberculosis* cannot be distinguished from *pulmonary syphilis* without a careful microscopic examination of the sputum. Moreover, it must not be forgotten that luetics often develop ulcerative phthisis, and hence these affections are often combined.

SYPHILIS OF THE SPLEEN.

Pathologically, syphilis of the spleen is to be classed with the general adenopathy of the disease. According to the statistics of Sée (relating to hereditary syphilis) and of Avanzini and Schuchter (relating to acquired syphilis), in about 25 per cent. of the cases of secondary syphilis hypertrophy of the spleen may be noted. This augmentation begins from two to four weeks after the appearance of the chancre, and gradually increases, persisting throughout the secondary period; it is not, however, observed during the tertiary stage. It is often accompanied by localized pain—syphilitic pleurodynia (Besnier).

SYPHILIS OF THE CIRCULATORY SYSTEM.

The Heart.—The pathologic divisions are—(*a*) *Gummata,* which attack chiefly the walls of the left ventricle. They are usually encysted.

(b) *A Fibro-sclerotic Myocarditis.*—The process begins in the perivascular tissue and proceeds from the vessel walls outward (Mracek). It is diffuse, as a rule, and leads to narrowing of the lumina of the coronary arteries and their branches or to aneurysmal bulgings, but the pathologic effects of these lesions are seldom detected clinically. Sudden death may occur.

(c) *Syphilitic Endocarditis.*—The changes are of the fibro-sclerotic variety, and not of the more acute verrucose or warty type. The symptoms to which the lesion gives rise are depicted under Organic Valvular Disease.

SYPHILIS OF THE ARTERIES.

Two forms are recognized: (a) *Obliterating Endarteritis.*—Here the syphilitic product consists chiefly of proliferated subendothelial tissue, which encroaches more and more upon the lumen of the vessel—a fact to which the disease owes its name. This so-called "Heubner's degeneration" is not peculiar to syphilis, but, as Osler says, "if, however, there are gummata in other parts, or if there be gummatous periarteritis in adjacent vessels, the process may be regarded as syphilitic."

(b) *Gummatous Periarteritis.*—This results in larger or smaller nodules or ovoid masses that may encircle the artery. Among common seats are the cerebral and coronary vessels, the growth starting in the adventitia and proceeding outward. According to the views of syphilographers of the present day, it is to be classed with the ravages of the disease, and hence is not due to the syphilitic virus.

Syphilis of the arteries has an important etiologic bearing upon atheroma and aneurysm (*vide* Diseases of the Arteries).

SYPHILIS OF THE KIDNEYS.

Renal syphilis belongs chiefly to the tertiary stage, though it may appear in the secondary.

Pathology.—(a) *Amyloid degeneration* is a common renal lesion.

(b) *Granular atrophy* (Jaccoud).

(c) *Gumma.*

Symptoms.—Except in the case of amyloid degeneration the conditions are impossible of correct diagnosis. Wagner describes a special form which he calls *acute syphilitic glomerulo-nephritis.* Clinically, it is characterized chiefly by hematuria, and ends rapidly with uremia.

SYPHILIS OF THE JOINTS.

The following division of the affection is made by Hutchinson of London:

(1) *Synovitis* appears during the secondary stage, but soon clears away under appropriate treatment, leaving no traces behind.

(2) *Perisynovial gummata.*

(3) *Arthritis*, due to osseous nodes or gummata in the neighborhood of the joints.

(4) *True Chronic Synovitis.*—This is the most common form of syphilitic arthritis.

(5) *Syphilitic chondro-arthritis* (Virchow).

The last four forms belong to the tertiary lesions.

Symptoms.—It is to be borne in mind that a joint-affection that does not yield to specific treatment is not necessarily non-syphilitic.

Perisynovial gumma attacks most frequently the tissues around the knee-joint; it is very chronic in its course and is more commonly seen in women than in men.

Arthritis due to osseous nodes has a special diagnostic feature in the severe nocturnal pains. The fourth form of syphilitic arthritis (true chronic) is the most common among the types due to acquired syphilis, while the symmetric synovitis of the knees occurring about puberty is perhaps peculiar to the congenital cases.

Syphilis of the Testicles.

The lesions are of two forms: (*a*) *Gummata.*—These produce hard, usually nodular, swellings, either single or multiple, and of moderate size, that occupy the substance of the testicle and sometimes the epididymis.

(*b*) *Interstitial Orchitis.*—This is a fibro-sclerotic change that leads to slow, gradual atrophy. Though bilateral, it is usually more marked on one side than the other. *Epididymitis* occasionally develops as a lesion of early secondaries. It is usually unilateral, painless, and quickly disappears under treatment. In the tertiary stage gummata may develop.

Diagnosis.—In gummatous orchitis the swelling of the testicle is painless, usually nodular, and feels much like a scirrhous growth. Rarely it ulcerates, forming a fungous testicle. The frequency of syphilitic involvement of the organ forms a leading factor in the diagnosis.

In *tuberculous disease* the history and associated lesions differ from those of syphilitic orchitis, and the epididymis is generally affected. Atrophied testicles may be due to congenital syphilis. In such instances typical scars, eye-affections, and the characteristic physiognomy are usually to be noted. Hydrocele may owe its origin to the same cause. Atrophy of the testes may lead to impotency and sterility. Such instances are not to be mistaken for the results of metastasis in *mumps*.

General Diagnosis of Syphilis.—Perhaps sufficient has been said regarding the importance of obtaining a correct statement with reference to the primary infection. On failure to find evidence of a genital chancre, an examination for extragenital primary sores must be instituted, and the latter will be found to be by no means rare, even among children.

The secondaries are rarely puzzling, especially when the previous history is complete. There may be complicating eruptions. In Bulkley's records of 300 cases, 23 well-recognized affections of the skin were associated with syphilis. In this connection two facts need to be emphasized: first, that a syphilitic eruption, either macular or papular, may rarely cause troublesome itching; and second, that a patient with a syphilitic eruption may experience itching due to another cause—namely, eczema or scabies.

Inherited syphilis may be diagnosticated on the appearance in a child under five months of snuffles and the characteristic skin-eruptions.

Syphilis hereditaria tarda may be recognized either from a retrospective view or from the presence of active lesions and symptoms.

Tertiary manifestations of acquired syphilis embrace these points: 1. The consideration of the fact that obscure cases in general and atypical symptom-groups are often due to the syphilitic taint. 2. Direct information or proof, as the result of careful inquiry, to show that the primary and secondary stages (either one or other, or both) have transpired. 3. The evidence presented by the patient and to be obtained by the careful objective examination of the eyes (for iritic adhesions, etc.), throat and skin (for scars), bones (for necrosis and nodes), and the testes. 4. Certain symptoms are significant, such as nocturnal pains, paralysis of the single cranial nerves, double deafness without otorrhea, etc. 5. The therapeutic test may aid in doubtful cases.

The presence of scars constitutes a most important factor in making a retrospective diagnosis. Recent scars are pigmented, and exhibit a slow, progressive clearing up, until, from four to eight years after infection, they are wholly decolorized. On the other hand, as pointed out by Hyde, eczemato-varicose scars remain stationary. These scars are apt to be found on the scalp and on the anterior surfaces of the legs. They may be single or multiple, and may exhibit certain defined shapes (semilunar, dumb-bell, etc.).

Justus's blood-test for syphilis, which consists in a sharp, transient reduction of the hemoglobin after the administration by inunction or hypodermic injections of mercury, is not pathognomonic, but an aid in diagnosis. It occurs in conditions other than syphilis (Christian and Foerster).[1]

General Differential Diagnosis.—Numerous affections and conditions—local and general—are liable to be confounded with syphilis. Mere allusion to some of these common errors of diagnosis can be made here, while others must be omitted altogether:

(*a*) *The primary sore* of the lip has been mistaken repeatedly for cancer. The history and symptoms of syphilis, together with the therapeutic test, must clear up the doubt.

(*b*) Certain *skin-eruptions* (lichen, psoriasis, papular eczema, etc.) may be mistaken for the eruption of secondary syphilis. J. V. Shoemaker[2] details the differential diagnosis in a recent article, which the reader who desires full information may consult.

(*c*) Care must be exercised lest the *specific eruptive fevers*, especially the pustular stage of small-pox, be mistaken for secondary syphilis.

(*d*) The syphilitic arthritis which may develop at the onset of the second stage must be discriminated from *rheumatic arthritis*—an easy task if only the attention be drawn to the primary lesion and the characteristic secondaries in cases of the former disease.

(*e*) Syphilis in the tertiary stage may simulate *chronic gout* or *rheumatism*, and unless there is definite evidence of syphilis on the one hand, or typical rheumatic symptoms and history on the other, the diagnosis may remain indefinitely uncertain. The therapeutic test may aid.

(*f*) *Periosteal nodes*, like those occurring in syphilis, may follow vaccination, small-pox, typhus and typhoid fevers. Here the history and associated phenomena furnish reliable data for discrimination.

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[1] *University Med. Mag.*, November, 1900.

[2] *Medical Bulletin*, November, 1893.

(*g*) *Carcinoma of the tonsil* has often been diagnosed, and the tonsils have been excised when really the seat of a syphilitic lesion.

(*h*) Janeway[1] asserts that *chronic syphilitic fever* and *tuberculosis* are not rarely confounded.

Treatment.—(*a*) **Prophylaxis.**—To prevent the transmission of hereditary syphilis infected persons should not marry within four years after the appearance of the primary sore. "Marriage should also be prevented when the patients have not been subjected to a thorough and prolonged treatment" (Porter). If at the end of the third year the patient presents a mucous patch, he must wait one year longer.

Should a healthy mother bear a syphilitic child, she must not be allowed to suckle it. This precaution, though apparently contrary to the principle laid down in Colles's law, is not superfluous, since the mother might be infected by any oozing fissures or condylomata upon the lips or in the mouth of the child if erosions of her nipple were to occur. Wet-nurses should not be employed for syphilitic children, but may be for non-syphilitic, even when the mother is affected. If syphilis appear in the mother during pregnancy, antiluetic treatment should be begun and persisted in even after apparent recovery. After the birth of the child treatment should be continued, if the child be nursed by the mother, with a view to medicating the milk.

As has already been stated, the most frequent mode of infection is irregular and illicit sexual congress, and it follows that absolute moral purity would go further toward the prevention of this widespread malady than any sanitary code or legal restrictions. Physicians cannot too strongly advocate continence. Should prostitution be regulated and controlled by the state? Experience has shown that but a slight control is exercised over the spread of syphilis in countries where systematic regulation of prostitution is attempted by the state. I am of opinion that the state should maintain some form of sanitary regulation and control, but, unfortunately, to render this efficient demands that prostitutes shall be officially registered. Such a sanitary supervision should consist in the examination of every prostitute at least twice a week, including a microscopic examination of the uterine and vaginal secretions, and the sending of every diseased prostitute to a hospital with a special department for such cases.[2] Palmer suggests that the female offender is usually not aware of the existence of a primary sore, while the male is; hence the latter should undergo inspection also. Inspection of prostitutes, however, unless rigid and careful, is absolutely valueless. Chancres are often concealed from view in the vagina or upon the lateral aspect of the os uteri. The maintenance of legal brothels, however, is not here recommended, either from a moral or hygienic standpoint.

(*b*) **Medicinal Treatment of Hereditary Syphilis.**—For syphilis of the new-born, mercury, either in the form of calomel (gr. $\frac{1}{10}$–0.0064, t. i.d.) or gray powder (gr. $\frac{1}{2}$–0.0324, t. i. d.), is to be employed. These babies must be hand-fed. The issue is almost unexceptionally bad.

When the first symptoms appear at the second or third month the above method of treatment is generally successful. Among the poorer classes no objection is made to mercurial inunctions, and these are often preferable. The ointment may be rubbed into the armpits, thighs, or sides of the abdomen, which should be covered with a flannel roller. The parts must be kept clean; and the mouth washed after nursing with a 3 per cent. solution of boric acid. Shaw prefers to treat infantile syphilis by inunctions, because of the digestive disturbances usually following the internal administration of mercury to children. *Syphilis hereditaria tarda* is best treated by the use of potassium or sodium iodid. To the iodid may be added mercuric chlorid in suitable doses, though the latter may sometimes disagree (Roberts). In addition to the specific therapy, tonic measures are usually indicated.

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[1] *American Journal of the Medical Sciences*, Sept., 1898.

[2] *The Berlin Commission on the Prevention of Syphilis*, Dec. 1, 1892.

(*c*) **Treatment of Acquired Syphilis.**—There is a specific plan of treatment which should be commenced as soon as the appearance of the secondaries has set the diagnosis of the given case at rest. This is the use of mercury, and rarely of potassium iodid also. The instances in which the latter alone is to be administered are rare. Fournier's "chronic intermittent treatment" of syphilis—which consists in continuous medication for two or three years with mercury and iodin alternately—is warmly advocated by some syphilographers; but the continuous mode is, in the opinion of most specialists, of greater advantage to the patient. Unless mercury disagree or the patient is exceedingly susceptible to its physiologic effects, I use it persistently during the secondaries, and later at intervals until the end of two years. It is a protracted course, and a protracted course only, of the specific treatment that suffices if we would obviate the dread ravages that otherwise are so apt to appear. I usually employ the protiodid (gr. $\frac{1}{8}$–$\frac{1}{3}$—0.008–0.021, three times a day), and later the biniodid (gr. $\frac{1}{30}$–$\frac{1}{24}$—0.0021–0.0027, three times a day). We should begin by giving one pill three times daily, and increasing one pill each day until the premonitory symptoms of ptyalism appear (tenderness when the teeth are knocked together, and ropy saliva); then the pills should be reduced $\frac{1}{2}$ or $\frac{1}{3}$, depending upon the number taken. By this procedure the physician is able to ascertain for each case the largest dose of mercury that can be given without harm. Hutchinson recommends the gray powder given in pill-form, combined with Dover's powder (*āā* gr. j–0.0648), this pill to be taken from four to six times daily. A well-known mixture, freely prescribed in many dispensaries, contains mercuric chlorid and potassium iodid in combination.

Inunctions of mercurial ointment (ʒss—2.0, night and morning) produce excellent results, and it is advisable in cases in which the syphilids yield unsatisfactorily to internal dosage to suspend the latter at intervals of six or eight weeks and give a course of inunctions. White advances the view that in the later stages, with the involvement of the deeper tissues, the combined use of inunctions over the affected region with potassium iodid internally often seems to have distinct advantages as compared with the administration of the "mixed treatment" by the mouth. It is necessary to omit the inunction once in seven or eight days for one day, and to take a warm bath to aid in the elimination of the mercury.

The hypodermic use of mercury in syphilis is to be adopted only when very prompt action of this agent is desired. Several preparations are used, and whether these are soluble or insoluble is a matter of little moment. The bichlorid takes first place, the dose being gr. $\frac{1}{4}$ (0.0162), in

15 to 20 drops of water, twice a week. Calomel probably holds second place (dose, gr. j—0.0648, in 15 drops of glycerin, twice a week). Among other preparations employed are the peptonate of mercury and gray oil. All injections must be made deeply into the muscles. The subcutaneous injection of sterilized serum from the blood of lambs and calves has been successfully practised by Tommasoli.

The method of fumigation has gained favor, particularly in the treatment of syphilis, in institutions on the Continent. Lane recommends that calomel (ʒiss—6.0) be put in a china bowl about half filled with water; a spirit lamp is placed under this, and the patient, "sitting above it wrapped in a cloak, has a deposit of mercury settle all over his body as the calomel is sublimed." He should remain wrapped in the cloak for one hour, take a fumigation once daily, and remain in-doors. From six weeks to three months are necessary to effect a cure.

The teeth should be cleaned thrice daily. *Hygiene* plays no mean rôle in the successful management of syphilis. The *diet* must be liberal, though green vegetables and fruits are not to be taken. Alcohol and tobacco are the two great enemies of the luetic.

Auxiliary measures, when the disease is associated with other lesions, are important. If syphilis occur in a tuberculous subject, it is of great value to add the potassium iodid to the mercurial, and, if active tuberculous lesions are present, cod-liver oil and creasote as well. Anemia and debility call for iron and a tonic plan of treatment generally. Attention should be given to the stomach, bowels, kidneys, and other internal organs, as well as to the nervous system.

In women the iodids should be suspended during menstruation if the flow of blood is excessive, but not the mercury. Says Mauriac: "During pregnancy specific treatment is well tolerated, and often requires to be pushed to a point a little short of intoxication for the good of both the mother and the child, close watch being kept upon the kidneys, suspending treatment at the first sign of albumin."

(*d*) **Treatment of Tertiary Syphilis.**—For most tertiary manifestations, including visceral syphilis, we have a therapeutic specific in potassium iodid. This should be used alone, the inunctions of mercury being added if the iodid fails to produce the desired result. I give the potassium iodid in a saturated solution, one minim being equal to ¾ grain of the salt. I use gr. x (0.648) t. i. d. at the first dose, and increase the latter 1 grain (0.0648) each day until the manifestations for which it has been prescribed disappear or iodism is induced. It is best given in milk. In cases showing cerebral symptoms it is to be cautiously used, and it is then my custom to combine the iodid with potassium bromid.

In hepatic syphilis the mercurials are usually combined with iodids from the start, and particularly calomel if there be ascites or jaundice.

In nervous syphilis, especially in the graver forms, I begin with large doses (gr. xx—1.296, three times a day), and augment as above indicated. The limit of doses depends upon the effect produced. I have often found sodium iodid to agree better with the stomach than the potassium salt. Most syphilitics tolerate the iodids to a remarkable degree; on the other hand, a few show a marked idiosyncrasy to them. Among unpleasant effects are coryza, conjunctivitis with edema of the eyelids, salivation, and certain skin-eruptions (erythema, urticaria, etc.). In this form of syphilis, as in the earlier stages, the specific treatment is made much more effective by attention to certain hygienic measures —fresh air, appropriate diet, bathing, exercise, and rest.

LEPROSY.

(*Lepra.*)

Definition.—A chronic, contagious disease, caused by the bacillus lepræ. It is distinguished by constitutional depression and, pathologically, by tuberculous masses in the muco-cutaneous surfaces, and by changes in the nerves.

Historic Note.—In 1889, Morrow stated that in India alone there were certainly not less than 150,000 lepers, while at present it is estimated that there are over 250,000. Its geographic distribution probably covers more than one-third of the entire surface of the globe. It is common in Africa, Brazil, in the East, and in Norway. In the Sandwich Islands the disease is of comparatively recent origin, and yet of great and increasing prevalence, a leper settlement having been established consisting of more than 11,000 cases. Leprosy is not unknown in America, and in Mexico it has existed ever since the time of Cortes (Morrow). Blanc states that there are 75 to 100 lepers in Louisiana alone. It was introduced into California and Oregon by the Chinese, and into Illinois, Iowa, Wisconsin, and Minnesota by Scandinavian immigrants. It has been imported from the Sandwich Islands to *Salt Lake City*, and from Normandy to Tracadie on the Gulf of the St. Lawrence, where the "disease is limited to two or three counties which are settled by French Canadians" (Osler). Sporadic cases have been met with in most of the larger American cities.

Pathology.—The bacilli grow and develop in clusters in the tuberculous nodules in the skin, residing within the epithelioid cells and leukocytes. These so-called lepra-cells are probably derived from the lymphatic vessels or capillaries, having been transformed by the bacilli. Surrounding the granulomatous masses is a layer of connective tissue. The bacilli are also found in the lymphatic glands, the spleen, and liver, but rarely in the blood. The nodular tumors form projections from the skin-surface, and, being poorly supplied with blood-vessels, they soon undergo caseation and absorption or are obliterated by dense connective tissue, which leads to the condition known as *facies leontina*. The pus-organism generally exercises an influence in causing suppuration with ulceration, which may manifest a marked destructive tendency. Similar changes occur in the internal organs or in the mucous membranes.

Nerve-lesions are induced by the presence of the bacilli within and around the nerves. Here they first set up an irritation with hyperesthesia (neuritis), which leads to atrophy, with marked degenerative changes.

Etiology.—**Bacteriology.**—In 1880, Hansen discovered the bacillus lepræ, which has since been proved to be the special agent of the disease. It strongly resembles the tubercle bacillus, but differential stains have been suggested by Unna and others. The British Leprosy Commission have shown that the bacillus can be cultivated, but inoculation experiments on animals have not as yet succeeded. Bordoni-Uffredozzi was able to cultivate a bacillus, which differed from the lepra bacillus in its morphology, although staining in a similar manner.

Predisposing Causes.—Every one is susceptible to leprosy, but the disease is most frequent between the twentieth and fortieth years, and is rare in childhood. Sex and latitude have little if any influence. Hereditary transmission probably influences about one-fortieth of the instances (Zambaco). Heredity is denied by both Hansen and Raminez. As pointed out by Bidenkap, leprosy is often rare in large cities, even though prevalent in the surrounding rural districts.

Modes of Infection.—The disease is transmitted by contact; but Widal and others, who have studied the disease as it exists in the Hawaiian Islands, think that leprosy is contagious only by inoculation. Morrow's view, that, like syphilis, leprosy is generally transferred by sexual intercourse, receives support. Hansen holds that the infection atrium is unknown; he thinks it probable, however, that the mouth and nasal cavities are the avenues of entrance.

Clinical History.—Two forms are recognized, the *tubercular* and the *anesthetic*, while a third or mixed type is described by some authors. Neither of the first two, however, runs its entire course without developing into a third or mixed form.

The *incubation* is usually long (three to five years—Hansen). This period may rarely be shorter on the one hand, or greatly retarded on the other. Vague *prodromes* are present for years (drowsiness, chilliness, irregularly recurring attacks of fever, debility).

(1) **Tubercular Form.**—In the first stage there is a patchy, cutaneous *erythema* with a slight hyperesthetic elevation of the affected areas (*macular leprosy*). These are oftenest seen on the face or upon the extensor surfaces of the arms and knees. They may vanish after a while and leave the skin pigmented and anesthetic, and later the pigment may disappear, while white spots of corresponding size remain (*lepra alba*).

When the disease progresses less favorably tuberculous *nodules* (dusky red or almost brown in color) develop in addition to anesthesia. The small ones soon disappear, while the large ones are either absorbed or break down and ulcerate—changes which, as they advance together with the slow healing process, produce marked deformities. The *skin* is greatly thickened and presents a scaly surface, and there is loss of substance in certain parts, while others are enormously enlarged (eyebrows, nostrils, lips, etc.). Among the many symptoms pointing to involvement of the mucous membrane are *ozena*, *hoarseness* or even *aphonia*, and the signs of *inhalation-pneumonia*. To the last-named disease, as well as to ulcers extending deeply into the mucosa of the pharynx and larynx, death may often be ascribed.

(2) **Anesthetic Form.**—In this variety the *local symptoms* point usually to implication of the nerves. At the onset there are *pain* and *patchy*

hyperesthesia, while minute bullæ, due to trophic changes, put in an appearance on the arms and legs. The *muscles* supplied by the branches of the affected nerve-trunk waste, and the superficial nerves feel thickened and nodular. *Bright-red patches* of vaso-motor congestion appear and soon become anesthetic, while the maculæ disappear. *Anesthesia* may proceed without the latter eruption. Dry, yellowish-white, scaly patches upon the trunk and extremities are also visible. Early their centers alone are anesthetic, but subsequently the loss of sensation spreads even to healthy portions of the skin.

Trophic alterations reach an extreme degree. Bullæ of considerable size appear, and, bursting, leave perforating or destructive ulcers, usually upon the extremities. As the result of absorption, wasting, and necrosis great deformities are produced. The hands often take on a claw-like form, and the fingers and toes may disappear (*lepra mutilans*).

Diagnosis.—The early diagnosis rests upon the presence of patchy erythema with hyperesthesia, followed by the development of anesthesia, with a disappearance of the macular eruption. Nodular neuritis is pathognomonic of anesthetic leprosy. Scraping a piece of new growth may rarely exhibit the specific bacilli. In the advanced stages of either form confusion could scarcely arise. The nodular form of *tubercular syphilis* is distinguished by the distribution of the lesions, the history, and the frequent sensory nerve-lesions. Zambaco and others have claimed that *syringomyelia* and *Morvan's disease* are in most cases but forms of leprosy; but this has been disputed by Hoffman, Schlesinger, Sahli, and others. Syringomyelia depends on lesions of the central nervous system, while leprosy has its nervous lesions in the peripheral nerves; and on this basis the two may be differentiated. The first symptoms in syringomyelia are localized usually in the upper extremities, while in leprosy they are generalized. In leprosy the tactile sense is usually lost, in syringomyelia usually not lost.

Serum-diagnosis of Leprosy.—Spronck[1] asserts that the serum of leprous patients, so far as examined, will agglutinate the bacillus in dilution of 1 to 60 or 1 to 1000. There is probably no relation between the agglutinizing power of the blood and the clinical symptoms.

Prognosis.—Leprosy runs a very chronic course, lasting sometimes two, three, or more decades. The prognosis as to the final issue is hopeless, but the patient may live in comparative comfort for many years before the ravages of the disease cause great mutilation.

Treatment.—Here may be mentioned the fact that certain diseases are supposed to exercise a retarding effect on leprosy (pleurisy, pneumonia, variola, phthisis). *Antagonistic inoculation*, however, as practised by Beaven Rake and others, has been practically negative in its effects; and the same is true of the treatment by Koch's tuberculin. The disease has thus far resisted all methods of treatment looking to a cure. Phillippo found that gurjun oil was serviceable, ulcers being rapidly cured by its application: the oil may also be applied as an ointment to the swollen parts. *Internally*, chaulmoogra oil has been employed by Berge and Phillippo with excellent results, the dose being from 1 to drams (4.0–8.0). It is sometimes administered in pearls (each containing ♏ 3 to 5 (0.199–0.333), in ascending doses, until the limit of olerance is reached. Surgical interference may become necessary. egregation of lepers has been instituted in certain localities with encouraging results. Calmette's antivenomous serum, while not a true ntidote, may produce marked temporary improvement or even cure in ncomplicated cases.

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[1] *Sem. méd.* Sept. 28, 1898.

GLANDERS.

(*Farcy*).

Definition.—An infectious disease of equine origin, and caused by the bacillus mallei. Two forms are recognized—*true glanders* and *farcy*.

Pathology.—The characteristic lesions are new growths (granulomata, according to Virchow), which are usually nodular in character, though they may be diffuse. These masses soften and form ulcers when they occur on the nasal mucosa, and abscesses when they are situated subcutaneously. Microscopically, the nodular tumors are composed of cells—lymphoid and epithelioid—together with the specific bacillus.

Etiology.—The morbid changes above described are caused by a specific organism, the *bacillus mallei*, which resembles closely the tubercle bacillus, though it is a little thicker as well as shorter. It is nonmotile. It can be readily grown, and as readily inoculated into horses, in which it produces the disease with every characteristic symptom. Perhaps the simplest method of staining the bacillus mallei "is to treat a cover-glass preparation with warm carbol-fuchsin, and then wash it off with a 2 per cent. solution of nitric acid."

Modes of Infection.—The virus is, as a rule, transferred directly from the infected animal to man, hence the disease occurs almost invariably among *males* and persons who come in contact with horses (hostlers, coachmen, soldiers, veterinarians, and farmers). Transmission from man to man has been observed, but rarely. The medium of conveyance is either the *pus* or the *nasal secretions*, which may drop or be blown from the animal's nostrils upon a wound in the skin or mucous membranes, however slight, and be absorbed.

Immunity.—The disease is rare in man, because of almost complete natural immunity. Singer has produced artificial immunity by intravenous injections of sterilized cultures of the glanders bacillus.

Clinical History.—The duration of the *incubation-period* is from three to five days, and rarely longer. Both glanders and farcy may be *acute* or *chronic* in their course.

(1) **Acute Glanders.**—At first the signs of inflammation develop at the point of infection, *lymphangitis* and swelling of the adjacent lymphatic glands being associated. *Fever* and other evidences of general disturbance soon appear, and at the end of two or more days the nasal mucosa

becomes implicated, ulcers forming, from which a fetid muco-purulent (sometimes blood-streaked) discharge takes place. *Nose-bleed* is common. Later an *eruption* comes out on the face, the trunk, and the extremities, particularly about the joints. It is papular, quickly becoming pustular, and the pustules may dry up while fresh papules are developing—a characteristic feature. The *face*, particularly the nose, now swells, and a bluish-brown tumor covered with vesicles appears. Implication of adjacent mucous membranes—conjunctivæ, pharynx, mouth, etc.—is usual, and less frequently the bronchial and gastro-intestinal mucous membranes are involved. The ulcerative processes may extend to the bones, setting up necrosis. True arthritis occurs in 10 per cent. of the cases (H. Morel).

(2) **Chronic Glanders.**—A rare disease with mild but vague general symptoms, as muscular and arthritic pains, fever at intervals, asthenia, and progressive wasting, and the local features of nasal catarrh, with a bloody muco-purulent discharge. Cough may be present.

(3) **Acute Farcy.**—In this form the virus is inoculated into the skin, which presents the chief symptoms, the nasal condition being in abeyance or absent. The primary lesion is of an aggravated type, accompanied by a large crop of cutaneous boils and abscesses, which often follow the line of the lymphatics. Their favorite seat is in the vicinity of the joints. The constitutional symptoms simulate those of acute pyemia.

(4) **Chronic Farcy.**—Granulomatous tumors, resulting in abscesses, constitute the chief clinical peculiarity. The abscesses are situated primarily in the subcutaneous tissues, and often near the joints. As a rule, they open spontaneously and discharge, first a thick, creamy pus, and later a thin, fetid material. They sometimes form distinct ulcers, which extend in depth until the bones are involved.

The *general symptoms* simulate those of chronic glanders, the fever-curve being of the hectic type. In advanced cases emaciation and prostration become extreme. The *duration* varies from ten to eighteen months, though death may result earlier from some associated disease.

Diagnosis.—The diagnosis cannot be made without a clear history of contact with an animal known to be affected with the disease. In doubtful instances pure cultures should be made, and inoculated into a rabbit or guinea-pig. Death of the animal infected usually occurs within twenty-four hours. One of the products of the bacillus mallei is so-called "mallein," which has been used by Nocard and others as a diagnostic agent in animals. Its injection into horses suffering from glanders is followed by a febrile reaction. Schindelke injected more than six hundred horses with Forth's mallein, and his results show that a reaction of 3.5° F. (2° C.) is an almost positive proof of glanders; a rise exceeding 1.85° F. (1.5° C.) affords a strong presumption; while a rise of 1.25° F. (1° C.) is suspicious.[1]

Differential Diagnosis.—Cases of acute glanders have been mistaken for *variola;* but the history of exposure, the mode of onset, nasal symptoms and the course of the eruption all differ from those of the latter disease. *Pyemia* may be eliminated by the history of exosure and inoculation experiments. The chronic forms must be distinguished from *tuberculosis* and *syphilis*.

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[1] Saunders' *Year-Book* for 1896, p. 1013.

Prognosis.—Acute glanders and acute farcy are almost invariably atal. The chronic forms, however, and particularly chronic farcy, end n recovery, under appropriate treatment, in nearly one-half the cases.

Treatment.—The primary lesion should be dealt with surgically, and thorough disinfection followed by cauterization is highly recommended. Bayard Holmes advocates the opening of fresh abscesses and the scraping out of old ones under an anesthetic. A supporting plan of treatment, by generous feeding and judicious stimulation, is to be adopted, and the symptoms are to be met as they appear. The product, "mallein," has been recommended as a specific in this disease, but even Bonomé, who reports an instance in which recovery followed its use, contends that its curative properties have not yet been demonstrated.

ACTINOMYCOSIS.

(*"Big-jaw," "Lumpy-jaw," etc.*)

Definition.—An infectious disease of cattle, less frequently of man, caused by the ray-fungus (actinomyces), which grows in the tissues, developing a mass with secondary chronic inflammation and metastatic growth, as well as a secondary pyemic infection.

Historic Note.—In 1877, Bollinger gave the first description of the ray-fungus, which he had observed in the disease known as "big-jaw" in the ox. Israel of Berlin discovered the fungus in man one year later. In 1879, Ponfick showed clearly that actinomycosis in man and cattle was one and the same disease. Murphy, who described the first case of actinomycosis hominis in America, states that up to the present date more than 500 cases have been reported.

Pathology.—A *macroscopic* mass is produced, consisting of a central fungous mass from which threads of mycelia radiate in all directions, producing the ray form of growth. Individual growths are of the size of a millet-seed, but their aggregation may result in masses as large as an orange. They are of a sulphur-yellow color and of tallowy consistence.

Microscopically, the little or single ray-like tumors show straight or wavy branching filaments (*supra*). Their development is accompanied by the growth of dense adjacent connective tissue. In addition, abscesses containing yellow granules in the pus occur, but these are secondary. The usual lesions are not the same as those described as occurring in beasts. In man the lesions are those of chronic pyemia, and consist of a metastatic abscess-formation.

Bacteriology.—The organism of the disease belongs to the cladothrix variety of fungus, and may be cultivated, though with difficulty. The finer threads may readily be stained with anilin colors. The club-shaped projections, however, do not take these stains, so that when examining sections Gram's method may be used. Rabbits and cows have been successfully inoculated with cultures of the actinomyces.

Modes of Infection.—Infection generally takes place through the mouth, teeth, and pharynx; and rarely the gate of entrance for the virus is the air-passages or the skin. It is generally introduced with the food or drink, and Bostroem, from a study of 32 cases, concludes that the poison enters the economy by means of the ingested grains of some cereal (barley). Infection cannot occur through a normal mucous membrane or skin, but a wound, however insignificant, is essential.

Clinical History.—(1) **Oral Actinomycosis.**—The patient often complains of *toothache*, *dysphagia*, and of *difficulty in opening the jaw.* The latter symptom may be owing to induration of adjacent muscles, and is a very characteristic sign (Partsch). At the angle of the jaw a swelling appears, which quickly passes into suppuration; later it opens (first externally, then into the mouth) and discharges pus containing little yellow masses. If not properly treated, extension of the process takes place in a downward direction, even to the abdominal organs.

The *upper jaw* may be the primary seat of infection, and if so the base of the skull may be perforated and the disease attack the meninges and brain. Bollinger has seen primary actinomycosis of the brain. In these instances caries of the spine may result from extension.

(2) **Pulmonary Actinomycosis.**—I am satisfied that primary pulmonary actinomycosis is comparatively rare, although Karewski and Butler have each recently reported an instance. In Butler's case the disease followed an injury by a falling board. The disease begins with *pain in the side*, and often upon the left, due to *pleurisy*. There are *cough* and a peculiar (often fetid) *expectoration*, together with general wasting. A microscopic examination of the sputum, if made with care, reveals the *actinomyces.*

In some instances the symptoms are identical with those of *disseminated tuberculosis* of the lungs (Brigidi), though generally the disease is unilateral. There is irregular fever, due chiefly to suppuration.

The **physical signs** may be those of chronic bronchitis merely; but there are, in not a few cases, extensive destructive changes of variable character (abscess, broncho-pneumonia, etc.) which modify the signs accordingly. In primary pulmonary actinomycosis an extension to adjacent organs and also metastatic growths and abscesses occur.

(3) **Intestinal Actinomycosis.**—The condition may be *primary* or *secondary.* The organism grows upon the mucosa of the intestine and excites a proliferation of the underlying connective-tissue cells, and the formation of submucous nodules. The latter ulcerate, and perforation of the serous coat of the bowel may occur, inducing peritonitis. Pericecal abscesses have been formed in like manner.

The **symptoms** point to intestinal catarrh, there being some gastric disturbance, with irregular and recurring attacks of diarrhea. The actinomyces has been detected in the stools. Secondary metastatic growths (rarely) and abscesses may arise in other organs (liver, spleen, ovaries, etc.), but it is to be recollected that the primary seat of infection may also be the spleen, liver, or other visceræ.

(4) **Cutaneous actinomycosis** rarely occurs. The skin presents chronic suppurating ulcers which show the presence of the ray-fungus, and the condition bears a close resemblance to a lupus patch.

Diagnosis.—This rests solely upon the finding of the actinomyces.

he wooden hardness of the tissues beyond the borders of the ulcers or nuses, the hardness of the neighboring muscles in oral actinomycosis, nd the yellow granules in the pus are all significant, but merely cor-oborative. To detect the actinomyces, says Warren, sections may be tained with Ziehl's carbol-fuchsin from fifteen minutes to half an hour, nd then decolorized in a 1 per cent. picric-acid solution until the whole ection has a yellow appearance. Dehydrate and mount. The fungus ppears as a brilliant red aster, while the surrounding tissues are yellow. The points mentioned above will serve to distinguish this disease from *tuberculosis*, *syphilis*, *chronic pyemia*, and *sarcoma*.

Course and Prognosis.—The course is chronic. Mild cases may recover in from six to nine months or earlier, the oral form being perhaps the most favorable. Pulmonary actinomycosis may terminate in recovery, though rarely. Death usually results from pyemia, amyloid degeneration, and wasting.

Treatment.—This is mainly surgical. The removal of the parts involved and disinfection with acid-sublimate solution are the best measures. Billroth in a case of abdominal actinomycosis communicating with the bladder effected a cure by the use of fifteen tuberculin injections. Internally, the potassium-iodid treatment, as first recommended by Thomassen in 1885, and recently emphasized by DaCosta,[1] is often attended with success when decided iodism is produced

ANTHRAX.

(*Malignant Pustule; Splenic Fever; Wool-sorter's Disease, etc.*)

Definition.—An acute, infectious disease, caused by a special bacillus and clinically accompanied by the development of a characteristic pustule (boil) and blood-poisoning (*external anthrax*). The disease likewise affects the gastro-intestinal tract and the lungs (*internal anthrax*). Both forms are derived principally from the herbivora, it being especially prevalent among sheep and cattle. The existence of anthrax in the United States, in Asia, Russia, and parts of Europe has been denied, but it occurs rarely, and Bard has described its ravages in California.

Pathology.—Post-mortem rigidity is marked. The blood is dark and thick and coagulates poorly, and in it, particularly in the spleen, as well as in the liver, kidney, and lungs, one may find the spores. Besides the local lesions of the skin (*i. e.* ulceration, gangrene, edematous infiltration), and besides the degeneration of the heart, kidneys, and liver that is common to the severe and rapid infectious diseases, the especially striking lesion is the constant and great splenic enlargement. The bowel may show hemorrhagic infiltration and gangrene, and the mesenteric and retroperitoneal glands may be enlarged and hemorrhagic.

Etiology.—**Bacteriology.**—The special agent is the *bacillus anthracis*. Gratia and Jonne give as the microscopic characteristics of anthrax, as seen in the blood, the following: (1) The anthrax bacillus has the form of a rod of a length varying from 5μ to 20μ, and in breadth from 1μ to 1.5μ. It is broken up into short articulations from 1.5μ to 2μ long, placed end to end like the sections of a tenia, the ends of each articulation being slightly swollen, giving the appearance of a bamboo cane; (2) clear spaces, appearing like a biconcave lens, exist between the ends of the articulations, and result from the slight concavity of these ends; (3) a capsule, often distinctly marked, surrounds the rod, seeming to form a protoplasmic support for the individual articulations. These threads of anthrax bacilli stain best with Löffler's blue. They grow readily on various media (agar, gelatin, potatoes, etc.) into interlacing thread-like filaments which distinctly show spore-formation, the threads assuming the appearance of strings of beads. They resist desiccation, many of the germicides, and boiling water even for a few minutes. Inoculations are followed by the production of the pustule of anthrax. Conradi[1] affirms that it is highly improbable that the anthrax bacillus produces a *toxin*.

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[1] *Proc. of the Assoc. of Amer. Physicians*, 1900.

Modes of Infection.—The *virus* (spores) gains entrance into the human body through the skin (slight wounds, abrasions, or scratches), the intestines (with food), or through the lungs (rarely). The sting of insects (mosquitoes, flies) may also transfer the poison to man.

Predisposing Causes.—*Occupation* is most influential: persons who come into direct contact with infected animals (hostlers, butchers, shepherds), and workers in factories who handle the hair or hides of such animals, being liable.

Immunity.—Pasteur's well-known protective inoculation with attenuated virus has been extensively practised in anthrax localities, with very favorable results. Peterman, however, reinvestigated the question of immunity by the albumose of anthrax, and found it without protective action, except in the case of cultures on ox-serum, which, when injected in large quantities into the veins, conferred temporary immunity.

Clinical History.—The period of incubation is from one to three days. Two leading clinical types are distinguished:

(1) **External Anthrax.**—(*a*) *Malignant Pustule.*—At the point of infection (the hand, arm, neck, or face, or other exposed part) a small *papule* first appears, and develops into a *vesicle* of considerable size with bloody contents. This vesicle breaks, leaving a characteristic dark-bluish or black *scab* (anthrax), and encircling the primary vesicle an areola of miliary vesicles may be noticed. The base of the original vesicle now becomes swollen and indurated, and this brawny edema spreads rapidly to the adjacent tissues until an extensive area is involved. The neighboring *lymph-glands* may or may not be inflamed; if so, they are apt to be connected with the pustule by red lines (lymph-vessels, veins).

Severe *general disturbances* accompany the local disorder in the course of a couple of days, and comprise fever, decided prostration, sweats, splenic enlargement, and delirium tending toward coma. If recovery occur, the edematous swelling subsides and the black scab is cast off. In unfavorable instances collapse develops, and the case ends fatally between the fourth and eighth days. In such instances intestinal symptoms (diarrhea) or nervous phenomena of aggravated type may attend.

(*b*) *Anthrax Edema.*—In a certain proportion of the cases the *sys-*

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[1] *Zeitschrift für Hyg.*, June 14, 1899.

temic infection is out of proportion to the *local disturbance*, the latter consisting of an edematous swelling without the presence of an eschar. The eyelids (commonly), lips, tongue, and upper extremities may be the seat of extensive swelling, though there is no change in the color of the skin. This is a dangerous condition, and may result in gangrene.

(2) **Internal Anthrax.**—(*a*) *Intestinal Mycosis.*—In this form certain general, indefinite symptoms are the primary features, such as headache, pains in the limbs, anorexia, languor. Soon acute gastro-intestinal features supervene, sometimes preceded by a chill. As a rule, vomiting occurs, followed by abdominal pains and diarrhea, and the stools often become bloody. Hemorrhage may also occur from other outlets. Other symptoms, as dyspnea, marked cyanosis, and restlessness, are noted, followed sometimes by stupor, general convulsions, or spasms of single muscles or groups of muscles. There is moderate fever, and the spleen is enlarged. Death is preceded by collapse.

Interesting *epidemic* outbreaks of internal anthrax have occurred, due both to drinking-water derived from infected wells and also to diseased meat. Murisier has related the history of an epidemic in which 200 persons fell ill after eating meat from a certain cow. The animal was quartered by a butcher who had previously slaughtered an ox afflicted with anthrax, and had not disinfected his instruments; four days after this 25 persons were attacked by the disease.

(*b*) *Wool-sorters' Disease.*—This occurs among the operatives in factories in which imported wool or hair, mostly from Russia and South America, is sorted, and to produce the typical affection the infection must be swallowed or inhaled in the form of dust. *Mixed cases*, or those showing both external and internal anthrax, may be met with among workers in curled-hair establishments and the like. The *onset* is sudden, with a chill that is accompanied by pains in the back and legs, prostration, and a sharp rise of temperature to 102° or 103° F. (39.4° C.). The *local* symptoms may either be chiefly pulmonary or gastro-intestinal. The former consist in dyspnea, chest-pains or feelings of constriction, cough, and rarely the physical signs of bronchitis; the latter comprise vomiting and a diarrhea that is followed by collapse, with marked lividity. Nervous symptoms, delirium, convulsions, or coma are often prominent in serious forms; but a fatal ending may occur while the mind is unclouded. The *course* ranges from one to five days.

(*c*) *Rag-pickers' Disease* ("Hadernkrankheit").—This has been identified by Eppinger as the same form of disease as "wool-sorters' anthrax." It occurs among the rag-sorters in the paper-mills near Graz. Infection occurs in the respiratory tract. The symptoms observed are high fever, followed by collapse, with depression of the body-heat, painful and paroxysmal cough, cyanosis, very weak heart, together with the signs of pleuritic effusion and consolidation of the lung.

Diagnosis.—The history (occupation, etc.) and the appearance of the malignant pustule in external anthrax leave little room for doubt. The diagnosis, however, should be confirmed by an examination of the contents of the pustule for the presence of bacilli, and if found they should be cultivated and inoculated upon a guinea-pig or rabbit.

The recognition of internal anthrax is more difficult, but the condition may be suspected if the more characteristic pulmonary or gastro-intestinal symptoms, together with those of systemic intoxication, develop in persons whose occupation entails exposure. In doubtful cases the presence of the bacilli in the blood must be shown.

Prognosis.—In external anthrax occurring in healthy persons the disease often pursues a favorable course; moreover, radical surgical measures have decreased the death-rate decidedly. Internal anthrax, however, is a deadly affection. As regards "wool-sorters' disease," those who survive for one week usually recover (Bell).

Treatment.—Prophylactic measures embrace the sterilization and destruction of the hair, hides, wool, etc., of infected animals as well as the cremation of their bodies. Subsequent disinfection of the infected premises and the prohibition of grazing in infected pastures are matters of the utmost importance. The point of infection should be destroyed by caustic or by the hot iron, and then dusted with powdered mercuric chlorid, with a view to destroying the mass. It seems to me, however, that unless the pustules be small and remain so, removal by excision should be preferred, and Klein and others have reported recoveries after removal of the primary focus of infection. In preventing extension of the brawny edema hypodermic injections, several times daily, of a solution of carbolic acid at points a little distance from the site of the pustule have given the best results. Hallopeau recommends that in order to prevent extension the neighboring structures be bathed with a 10 per cent. solution of carbolic acid (first dissolved in alcohol) in oil or glycerin, Internally, stimulants, antiseptics, and nourishing food constitute our chief reliance. In internal anthrax efforts at treatment avail nothing.

HYDROPHOBIA.

(*Rabies.*)

Definition.—A specific, infectious disease peculiar to carnivora and to a less extent to herbivora, which may be communicated to man by direct inoculation. It is characterized by slight fever, spasm of the larynx and pharynx, delirium, a short stage of paralysis, coma, and, in the great majority of cases, ends fatally.

Pathology.—The facies, pharynx, and esophagus may be congested, the latter organ being sometimes markedly edematous; pulmonary congestion has also been noticed. The mucous membrane may show here and there points of hemorrhage, and Fitz has observed blood-extravasations into the perivascular spaces of the brain. Soft thrombi may fill the cerebral vessels, especially the veins, while the blood has a dark color and its clots lack firmness.

Balzer, Benedikt, Kolesnikoff, and Schaffer made careful studies of the changes in the central nervous system. Later, Babes described the "*tubercles rabiques*," which consist of pericellular accumulations of embryonal cells, the latter finally taking the place of the destroyed cell. More recently Van Gehuchten and Nèlis discovered lesions in the cerebro-spinal and sympathetic ganglia; they "consist in the atrophy, the invasion, and the destruction of the nerve-cells brought about by new-formed cells derived from the capsule, which appears between the cell-body and its endothelial capsule. These new-formed cells increase in number, invade the protoplasm of the nerve-cell, and finally completely occupy the entire capsule." Rarely, the kidneys may show cloudy swelling. The cadaver putrefies rather early.

Etiology.—Pasteur has found the poison abundantly present in the nerve-centers, and has transferred the disease by taking bits of brain-substance or medulla derived from an infected animal and inoculating them into healthy subjects.

Bacteriology.—Spenelli, Rivolta, Foll, Ferran, and others have detected and described a bacillus which they believe to be specific. Memmo[1] seems to have established its claims as the special organism of this disease. He has successfully induced the disease in dogs, rodents, and birds, with the typical differences characteristic of each.

The usual *mode of infection* in man is through the bite of a rabid animal, the virus being contained principally in the saliva, and in an immense majority of cases (about 90 per cent.) the dog is the offending party. The cat, wolf, cow, and horse also suffer from the disease, and in rare instances they communicate the disease to man. The skunk is also liable, and its bite has often transmitted rabies, especially to persons sleeping in the open air or in tents which the animal can enter. The virus gains access to the system through the broken skin.

Susceptibility to the poison exists in about one-half the instances in which persons are bitten by rabid animals, though in some cases this apparent immunity may be owing to slight or even non-infection.

Clinical History.—The *incubation-period* lasts from six weeks to three or four months, though in young subjects and in cases in which the infection is severe the symptoms develop earlier. Certain *prodromal symptoms* are manifested, as a rule, and generally last only a day or two; I have, however, seen two instances in which melancholia, due probably to the dread of what might follow, showed itself immediately after the reception of the bite and persisted. The usual premonitory symptoms are headache, loss of appetite, sleeplessness, great depression of spirits, and sometimes darting pains that radiate from the seat of the bite. The adjacent lymph-glands may become swollen, and slight difficulty in swallowing is experienced.

Following the *invasion* are two stages: (1) **The Stage of Excitement.**—The patient wears an expression of the most intense anxiety. Hyperesthesia is present and attains to a marked degree, and the special senses exhibit the keenest vigilance, a noise or a draft of air often causing great psychic disturbance or a violent reflex spasmodic contraction of the larynx. Quite early the mere sight of water is dreaded by the patient, and forms a characteristic feature of the disease. This symptom has given the name hydrophobia to the disease, and springs from the fear of inducing a painful spasm of the larynx. The patient has thirst which he cannot assuage. There may be maniacal excitement, and the spasmodic contractions of the larynx may become so strong as to excite urgent dyspnea, with the emission of curious sounds. The muscles of the mouth may also exhibit convulsive movements, causing the patient to make snapping sounds; these, however, are secondary. There is associated great restlessness, with frequent lateral rolling of the head, and foaming saliva may be ejected from the mouth. The symptoms occur in paroxysms, and during the intervals the patient is generally free from excitement. There is fever as a rule, the temperature ranging from 100° to 102° F. (37.7°–38.8° C.) or over, but it may be absent; the pulse is moderately accelerated and is sometimes irregular, and toward the end of this stage the reflex spasms of the respiratory apparatus develop spontaneously. Mental aberrations and melancholia may ensue, and often lead to suicidal tendencies.

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[1] *Centralbl. f. Bakt.*, Abt. i., Bd. xx., 17, 18.

(2) **The Paralytic Stage.**—In the concluding stage the patient passes into actual unconsciousness or coma, without spasms. This lasts from twelve to eighteen hours, ending in death.

In man there is a *paralytic form* of rabies, but it is rare as compared with the delirious or psychic type. Thirty cases have been reported by Gamaleia, and it is apt to follow deep and multiple bites. The paralysis begins near the part bitten, and spreads until it becomes general, finally involving the respiratory centers. In rodents quiet madness ("dumb rabies"), without maniacal excitement, is the rule.

Diagnosis.—The hyperesthesia, the fear of water, the reflex spasms on attempting to swallow, accompanied by dyspnea and great mental agitation, form a very characteristic grouping of symptoms. Bits of brain-substance or medulla of the rabid animal that has inflicted a bite should be quickly obtained, and a subdural inoculation of a rabbit be made. If virulent, the paralytic form of the disease will ensue in from fifteen to twenty days. Ravenel and McCarthy,[1] following the method[2] of Van Gehuchten and Nèlis, conclude that when present the capsular and cellular changes in the intervertebral ganglia, taken in connection with the clinical manifestations, afford a rapid and trustworthy means of diagnosis of rabies in the animal. When these changes, however, are absent (as happens in early stages of the disease), rabies cannot be excluded. *Hysteria* may be misleading, but here the previous history suffices.

The name *lyssophobia* has been given to cases that simulate, but have no relation to, hydrophobia, and Mills has advanced the warning that, with however so suggestive symptoms following a dog-bite, the given case cannot be assumed to be a case of hydrophobia until other possibilities are excluded. It is highly probable that there is a form of hydrophobia which is the result of the wide publicity given to genuine and suspected cases alike. The characteristic symptoms may be present, but they are comparatively mild and the affection does not develop. This so-called *pseudo-hydrophobia* appears only in neurotic and hysteric subjects, and runs a longer course than does the disease itself. Recovery is the rule. Burr reports an interesting case of the kind that occurred in Osler's clinic, attended, however, with recovery.

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[1] *Proc. Path. Soc. Phila.*, March, 1901.

[2] This is as follows: The ganglion is put at once into absolute alcohol, in which it is left for twelve hours, the alcohol being changed once. It is transferred for one hour to a mixture of absolute alcohol and chloroform; next put for one hour into pure chloroform; then for one hour into a mixture of chloroform and paraffin, and lastly in pure paraffin for one hour. The sections are put in the oven for a few minutes, then passed through xylol, absolute alcohol, and 90 per cent. alcohol, after which they are stained for five minutes in methylene-blue according to Nissl's formula, differentiated in 90 per cent. alcohol, dehydrated in absolute alcohol, and cleared in essence of cajuput and xylol. Ravenel and McCarthy found that the capsular changes were best brought out in sections stained by hematoxylin and eosin. Since these latter changes are the most essential diagnostic features in the sections, they suggest that material unfit for the Nissl method will still show the capsular changes when stained by hematoxylin and eosin.

Prognosis.—Few if any cases of rabies in man recover if the disease be allowed to develop.

Treatment.—Prophylaxis.—Upon the reception of a bite thorough disinfection, followed by cauterization of the wound with caustic potash, etc., is a measure that can be quickly carried out. The wound is then to be kept open for a period of four or five weeks. Systematic muzzling of dogs is to be encouraged and advised.

Preventive inoculation as perfected by Pasteur is a precautionary measure of the utmost importance. He showed that the virulence of the virus which is obtained from the nervous system undergoes modification by passage through animals. Thus the potency of the virus is increased by its inoculation from rabbit to rabbit (by placing bits of spinal marrow beneath the dura mater), the period of incubation at the same time growing shorter, till at last it is but seven days. On the other hand, the virulence is decreased or attenuated as the result of similar experiments upon the monkey. Pasteur also found that if fragments of the spinal cord were suspended in a dry atmosphere they lost gradually their virulence and finally became inert. From these an emulsion is prepared which is employed in the antirabic inoculations in man. In this way he secured a virus of known and reliable strength, and with this he could readily render the dog refractory by inoculating with very weak virus; then, by increasing from day to day the virulency of the inoculations, complete immunity was established.

Protective Inoculation.—"The patients are first inoculated with a cord fourteen days old, and the inoculation is repeated daily for nine days, each time with a cord one day fresher. In winter the oldest cords used are five days old, and in summer cords that have been drying for four days are also employed" (Warren).

For patients who have been bitten on the face, hands, or bare feet, as well as for those who have been bitten long before commencing treatment, the special preventive method, the so-called "intensive treatment," is applicable. Briefly, this consists in eliminating some of the inoculations of intermediary strengths, thus lessening the number of injections, and also in administering the latter at shorter intervals than in the usual method of treatment. The success of the Pasteur method is almost universally attested. Pottevin gives the following summary of figures from the Pasteur Institute: From 1886 to 1894, 13,817 persons were bitten, with a mortality of 0.5 per cent. In the New York Pasteur Institute, 313 West Twenty-third Street, under the directorship of Paul Gibier, of 1367 cases treated during the decade ending Jan. 1, 1900, 19 died—a mortality of 0.66 per cent. The patients should be sent to the Pasteur Institute at once, since delay tends to diminish the protective power of the inoculation.

The established affection defies all known methods of treatment. Our aim should be to diminish the intensity of the painful spasms and the psychic disturbances. The patient should be isolated from sounds, light, and excitement of every sort. Food, as a rule, must consist of nutrient enemata, though by the local application of cocain the sensitiveness of the throat may be diminished sufficiently to enable the patient to take liquid nourishment (Osler). For controlling the spasms chloroform by inhalation is most effective; chloral internally and morphin hypodermically may also be tried with advantage. The patient's anxiety is best relieved by a cheerful demeanor on the part of the attendants.

TETANUS.

(*Trismus; Lockjaw.*)

Definition.—An acute, infectious disease caused by the tetanus bacillus. It is characterized by painful spasms, affecting first and chiefly the muscles of the jaw and neck (*trismus*), and secondly those of the trunk, especially the extensors of the spine and limbs (*opisthotonos*). The disease may be idiopathic, though more often it is traumatic. In certain institutions and certain localities (*e. g.* eastern end of Long Island) it occurs endemically, and among new-born children and the colored race it may prevail epidemically (*trismus neonatorum*).

Pathology.—No constant post-mortem lesions have been found. The virus acts principally upon the nervous centers of the medulla and the cord, producing inflammation (and sometimes softening) of the gray substance of the cord. According to Brown-Séquard, the characteristic lesions are consequent upon an *ascending neuritis* starting from the wound, and it is true that the nerves often present traumatic lesions with redness and swelling of the neurilemma. Tetanus neonatorum often shows inflammation of the umbilicus.

Ætiology.—**Bacteriology.**—In 1884, Nicolaier discovered the bacillus of tetanus, and in 1886, Rosenbach first found it in man. It is a long, slender rod, at one end of which appears a swelling due to the formation of a spore in that locality, thus giving the organism an appearance like that of a pin or drumstick. The bacilli are easily stained by Abbott's method, and are purely anaërobic. Pure cultures can be made, but with difficulty, since they will not grow in the presence of the smallest amount of oxygen. If pure cultures are injected into animals, typical tetanus follows. Brieger has obtained two poisons from sterilized cultures of the bacillus in the pure state, and termed them "tetanin" and "tetano-toxin"—both most virulent poisons in the minutest quantity. These alkaloidal substances produce the tonic convulsions; hence tetanus is purely toxic in nature—an intoxication. The bacilli are most probably limited to the point of infection, and here develop the *toxin*, which "is carried mostly along the nerves to the spinal cord" (Stintzing).

Modes of Infection.—In the outer world tetanus bacilli are found to be both numerous and widely distributed. They abound in the earth (garden-soil in particular), putrefying liquids, manure, in rubbish, and dust of streets and houses, etc. The fact that the bacillus of tetanus is anaërobic explains why tetanus in man is a comparatively rare disease, and also why it is most apt to follow puncture and contused wounds. It may be assumed that an injury, however slight, serves as the portal of entrance for the poison. The *locality* of the injury is almost always on the extremities, particularly on the hands and the feet; the disease is most common *in warm climates;* and as regards *age*, it is most common between ten and thirty, if we except tetanus neonatorum. Idiopathic tetanus may follow exposure to cold or sleeping on the damp earth, and in tetanus neonatorum the infection may be communicated either by careless nurses or by dirty dressings of the stump, etc. (Papienske).

Immunity.—Behring and Kitasato have rendered animals immune by the injection of cultures of the bacillus after the addition of iodin trichlorid to diminish their strength, and this serum has been successfully used to protect others against tetanus.

Clinical History.—The duration of incubation depends upon whether the given case pursues an *acute* or a *chronic* course. In acute tetanus it lasts from one to two weeks, while in chronic the first symptoms usually appear after the second week. In idiopathic tetanus the symptoms generally appear shortly after exposure to the special causes.

Symptoms of Acute Tetanus.—(1) Mild prodromal symptoms (languor, headache, etc.) may precede the more intense characteristic phenomena, which develop gradually. At first the patient complains of stiffness and tension in the muscles of mastication and back of the neck, and soon tonic spasm of the masseters renders the facial muscles more or less immobile and locks the jaws (*trismus* or *lockjaw*). The rigidity of the cervical muscles is shown by the retraction of, and by attempts at raising, the head. The physiognomy is distinctive; it is immobile, the forehead being often wrinkled and the corners of the mouth retracted, producing a peculiar smile (*sardonic grin*). Next the muscles of the body become rigid, first the trunk (*orthotonos*), and then the spine is bent or bowed and the convexity presents anteriorly (*opisthotonos*). Lateral arching of the body also occurs, though rarely (*pleurosthotonos*). The belly-muscles are hard and board-like, and their contractions may throw the body forward (*emprosthotonos*). The arms generally remain movable, but the legs may be rigidly extended. The position of the body is one of constant rigidity, but from time to time convulsive seizures of variable duration occur, causing most agonizing suffering, thoracic oppression, dyspnea, and more or less cyanosis, due to interference with the respiratory function (especially spasm of the glottis). Sharp, lancinating pains occur at the base of the chest and point to contraction of the diaphragm. "Convulsive dysphagia" (as in hydrophobia) is rarely observed. These spasms are usually reflex, due to the action of slight external irritants. The reflexes are increased. The intellect remains clear. Profuse perspiration is a significant symptom.

Fever of a moderate degree is generally present. The temperature, however, may suddenly leap to 110° or 112° F. (43.3°–44.4° C.), forming an ominous symptom, these extreme elevations of temperature being probably due to paralysis of the centers that regulate bodily heat. Conversely, fever may be absent throughout the attack, and a brief postmortem rise of temperature be seen. The *pulse* is quickened, and in the worst cases may become very rapid (140 to 160 beats per minute), small, and irregular. The *urine* may be suppressed or its passage impeded by the muscular contractions. The bowels are constipated.

(2) **Chronic Tetanus.**—The same symptoms are manifested as are seen in the acute form, but the condition does not progress so rapidly. In some instances the symptoms soon become aggravated, to be followed,

however, by periods of decided relief from the painful spasms, so that during the latter the patient's strength can be maintained by means of stimulants, etc. Intervals of partial freedom from the excruciating pains grow longer in favorable cases, until finally the period of convalescence may be reached. *Relapses*, however, are common.

(3) **Cephalic tetanus** (first described by Rose) usually follows injuries to the head (face). Its most characteristic symptoms are rigidity of the masseter muscles, spasm of the pharyngeal muscles, causing dysphagia, chronic contraction of the muscles of the neck and abdomen (rare), and paralysis of the facial nerve on the same side as the injury. The latter symptom is due to local infection by a toxin. Recovery takes place in about 25 per cent. of the instances, according to Willard's statistics.

Diagnosis.—In view of the usual history, the predominating feature—trismus—together with the early appearance of rigidity at the back of the neck, will, as a rule, render the diagnosis a simple one.

Strychnin-poisoning must be eliminated, in which the following table will assist:

TETANUS.	STRYCHNIN-POISONING.
Reception of a wound, generally followed by a period of incubation.	Ingestion of strychnin, followed immediately by the symptoms.
Begins with lockjaw; later spreads downward (the arms and hands escaping).	Begins with gastric disturbance or a tetanic contraction of the extremities. Hyperesthesia of the retina occurs and objects look green.
Reflex spasms not present at the outset.	Violent convulsions present from the onset.
Rigidity is persistent, except in the chronic form.	Intervals of complete relaxation occur.
The course is prolonged into days or weeks.	Course is brief, terminating in death or recovery.
Cultures made from the discharges of the wound show the bacillus tetani.	Examination of the gastric contents shows strychnin.

Tetany gives rise to a prolonged spasm affecting the extremities (hands in particular) and the larynx, with intermissions; it is also characterized by a peculiar posture, and occurs chiefly in the young.

Hydrophobia is discriminated from tetanus by the history of a bite from an animal, by the predominance of the reflex spasm of the respiratory apparatus, by the intensity of the psychic disturbance, and by the absence of lockjaw and opisthotonos.

Course and Prognosis.—In the acute form the course is brief, and the prognosis is most unfavorable. Death results from asthenia, heart-failure, or asphyxia (during the paroxysm). According to Richter's statistics, 88 per cent. of military cases are fatal. In idiopathic cases the mortality-rate is under 50 per cent. Chronic tetanus gives a less grave prognosis than does acute. In the new-born recovery is so rare that when it occurs the diagnosis may be called into question.

Treatment.—In traumatic cases the wound must be disinfected and thoroughly cauterized. In order to do this effectively, the agents employed must be brought in contact with every portion of the wound, so that punctured wounds must first be laid open. Excision of the wound, and even amputation, may be advisable in some cases. The fact that the deadly poison is developed at the site of infection gives to the local measures supreme importance in the treatment of tetanus.

The patient should occupy a secluded room with little light and a carefully regulated temperature. A single nurse will suffice, and all sources of external irritation should be avoided. A nourishing diet is demanded, and rectal feeding must be instituted as soon as it is found that food cannot be administered *per oram*, or the food may be introduced by means of a small stomach-tube or catheter passed through the nostril. Stimulants should not be spared when the heart's action becomes quick and feeble. In one of my own cases hypodermic injections of strychnin and digitalis probably saved the patient's life. The spasms are best controlled by chloroform-inhalations, and during the intervals the patient should be kept under the influence of morphin, administered subcutaneously. Among other capital remedies are chloral hydrate and Calabar bean. The former may be exhibited in rectal injection (gr. xl —2.59 at a dose), to be repeated at intervals of six to eight hours until the spasm is overcome. The heart, however, must be carefully guarded. Rarely, potassium bromid, curare, nitrite of amyl, belladonna, and cannabis indica are useful.

Tetanus-antitoxin has been recommended for the *cure* of the disease, and is prepared in both fluid (antitoxin serum) and dry form. A dried preparation (which does not deteriorate) is also obtainable from Merck and his agents in the form of tubes containing from 4 to 5 grams each; at the time used it may be dissolved in water or in glycerin. Of Tizzoni's dried antitoxin 2.25 grams are to be given at the first dose, and 0.6 gram at subsequent doses. As shown by recent experimentation, antitetanic serum may prevent further invasion, but it cannot cure infection that has already reached the spinal cord and brain. The dose, as recommended by Copley,[1] should be large (30 c.c. at once, to be repeated at least every six hours until improvement is seen). Stintzing states that 96 cases of tetanus have been treated to date with Behring's serum, with 35 deaths. Behring insists upon giving the serum not later than twenty-six hours after the commencement of the attack. Intracerebral injections of antitoxin (Roux and Borrel) offer no peculiar advantages.

Prophylactic injections of serum containing at least 500 antitoxic units should be used immediately upon the disinfection of the primary focus. Baccelli advises subcutaneous injections of carbolic acid.

INFECTIOUS DISEASES OF UNKNOWN ETIOLOGY.

MUSCULAR RHEUMATISM.

(*Myalgia.*)

Definition.—A common, painful disease of the muscles and of the structures to which they are attached (fasciæ and periosteum), probably due to an attenuated form of the virus of acute articular rheumatism. Leube contends—and very properly, I think—that muscular rheumatism is a general disease with local symptoms. The latter may be seated in different parts of the body, and in this way give rise to a number of leading sub-varieties, and it may either accompany acute and chronic rheumatism or it may be experienced as an independent disease. I have also met with several instances in which it followed joint-rheumatism, and Leube has seen it precede the latter. Certain authors, however, believe that the affection is a neuralgia of the sensory nerves of the muscles.

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[1] *Brit. Med. Journ.*, Feb. 11, 1899.

Pathology.—In fatal cases (these are exceedingly rare) the affected muscles show a swelling of the fibers and more or less granular change. In long-standing cases there is an atrophy of the muscles, due to trophic disturbance. Strauss describes well-circumscribed nodules in the muscles.

The changes are essentially those of myositis. In the acute form there is often an extensive round-cell infiltration of the connective tissue, with swelling and partial degeneration of the muscular fibers and the formation in them of vacuoles. In the chronic form there is a proliferation of the interfascicular connective tissue.

Etiology.—Among the disposing influences that are most important in the causation of the affection are—(1) The *rheumatic diathesis* (appropriate soil); (2) *Heredity ;* (3) *Exposure* to cold, damp, and strong air-currents, especially after heavy exercise or during free perspiration; (4) *Sex*, owing to the more frequent exposure of men while following their occupations; (5) *Age*. It is met with at all ages, but acute and subacute forms most frequently occur among children and young adults, while the chronic form most frequently affects elderly persons ; (6) *Previous attacks* increase the susceptibility to the disease. That the disease is of infectious origin scarcely admits of doubt.

Symptoms.—In the majority of instances the clinical symptoms are local. Out of 200 cases Leube found *fever* in about one-third, the temperature rarely exceeding 102° F. (38.8° C.) for two days in duration, and far more often remaining normal. In one-sixth of Leube's cases there was a cardiac murmur, that disappeared under treatment in one-half of this number. The most conspicuous local symptom is the *pain*, which is sometimes sharp, lancinating, and paroxysmal, though it may be deeply seated, dull, and constant. It is aggravated at night by contraction of the affected muscles, by weather-changes, and by pressure. In long-continued cases pressure with the broad side of the hand usually affords relief. The *duration* ranges from a few hours to several days, but may be much longer. The rheumatic nodules are common in the shoulder- and calf-muscles. The cases in which the symptoms tend to persist or recur with changes in the weather may with justification be termed *chronic*.

Leading Clinical Varieties.—(1) **Lumbago** (*Myalgia Lumbalis*).—This is the most common form, and may be taken as the type of the myalgias. The *onset* is sudden, sometimes intensely so, and the lumbar muscles are exceedingly painful and sensitive. *Motion*, such as stooping or turning the body or rising from the sitting position, causes intense exacerbations of pain. The affection occurs most frequently in laboring-men, its course being brief, as a rule, and recurrences frequent. Erben, from a study of 200 cases of lumbago, finds that the trouble is principally an affection of the lumbar vertebræ, or a neuralgia of the cutaneous nerves.

(2) **Pleurodynia.**—This term implies involvement of the intercostal muscles, and less frequently of the pectorals and the serratus magnus.

It is unilateral, and oftener affects the left than the right side, and causes untold suffering, since it is constantly aggravated by the normal respiratory excursions. The pain is also intensified by pressure, reaching, etc., and by movement of the trunk, sneezing, and coughing. Similar symptoms may be occasioned by *traumatism* in which the fibers of the thoracic muscles are lacerated, and there is also great danger of confounding pleurodynia with *costal periostitis* and with *pleurisy*.

(3) **Torticollis** (*Myalgia Cervicalis*).—Here the muscles, some or all, on one side of the neck, and at times the throat, are implicated. The head is held toward the affected side, so as to relax the group of muscles involved, and on attempting to turn it the patient rotates his entire body in a pivot-like manner. The complaint is frequent in young persons.

(4) **Cephalodynia.**—By this term is meant rheumatism of the head-muscles of the scalp and fasciæ. It may be either *general* or *local*, being sometimes limited to the frontal, temporal, or occipital muscles. The *pain* is severe and greatly increased on motion of the scalp.

(5) Other terms descriptive of localized forms of muscular rheumatism are employed: (*a*) *Omodynia* (myalgia of the deltoid); (*b*) *Dorsodynia* (involvement of the muscles of the upper part of the back, etc.); (*c*) *Abdominal rheumatism* (myalgia of the muscles of the abdomen); (*d*) *Rheumatic myositis* of the extremities.

Diagnosis.—This is assured by the etiologic influences and the presence of pain, which is greatly increased by muscular contraction. The presence of fever does not exclude the affection. It differs from *neuralgia* in that there are no painful points, and in that firm pressure with the broad hand often affords relief. *Dermato-myositis* must not be confounded with muscular rheumatism. Unverricht first distinguished the former from the latter, showing that there are present pain and swelling of the muscles, as in muscular rheumatism, but additionally redness (erythema) and hyperesthesia of the skin, while the joints usually escape. Rovere, however, reports a case occurring in the course of diabetes mellitus in which there was a joint-inflammation resembling that of rheumatism. Of general symptoms, the chief are fever and physical prostration. The spleen is enlarged, and angina and hemorrhages have been noted. The disease is obviously infectious, probably septic in nature, and occurs in fatal as well as in the mild or favorable forms. Dermato-myositis, unlike muscular rheumatism, which is more common among men, is seen more frequently in women, especially servants.

The **prognosis** is good, the disease never endangering life, though a person may be incapacitated for work by muscular rheumatism.

Treatment.—Severe and acute forms demand the use of opiates internally and anodyne and hot applications externally. When cases are seen early, morphin, administered hypodermically, may serve to relieve the pain and cut short the disease. In acute cases the salicylates and other antirheumatic remedies are to be employed. Hot fomentations give comfort, and the Turkish bath may end the attack if it can be used sufficiently early. The hot-water bag, sponging with water as hot as can be borne, or dry heat in the form of bags filled with heated salt or heated hops, will all do good service. For the dull pain which is so distressing in some cases of torticollis the affected muscles may be covered with flannel, over which a warmed flatiron may be passed for a few minutes. This is an efficient expedient. For lumbago acupuncture is highly commended. Needles of from three to four inches (7.5–10 cm.) in length (ordinary bonnet-needles, sterilized, will do) are thrust into the lumbar muscles at the seat of the pain and withdrawn after five or ten minutes (Osler). Blisters have been recommended, but I have tried them frequently without beneficial effects in any case. In subacute and obstinate cases I have recently obtained good results from the use of a 20 per cent. ointment of salicylic acid freely rubbed into the skin. Active friction with anodyne and stimulating liniments (the latter when pain is not great) is worthy of trial. Massage and electricity (the constant current in particular) are sometimes efficient, and in chronic cases potassium iodid, guaiacum, and arsenic (the latter in small doses) should be tried. The same measures of prophylaxis are to be adopted as in chronic rheumatism, and the condition of the general health must also be looked to, every endeavor being made to maintain the proper quality of blood and perfect nutrition.

CHRONIC ARTICULAR RHEUMATISM.

Definition.—An affection of the articular structures which develops slowly and gradually and may be dependent upon the same causes as the preceding forms. Rarely it is a sequence of acute or subacute attacks.

Pathology.—The joints, as a rule, do not show pronounced gross lesions, there being some degree of synovial injection and also some, though not much, effusion. Inflammatory thickening of the articular and periarticular structures (capsule, ligaments, sheaths of the tendons, etc.) with contraction, is noted, and is a change which deforms and stiffens some joints to a certain extent. Superficial erosions of the cartilages may also be witnessed, and, as stated under the Clinical History of Acute Articular Rheumatism, muscular atrophy supervenes in long-standing cases of arthritis. The probable causes of these important changes have been pointed out in connection with the latter disease. When the shoulder-joint is the seat of chronic inflammation, this muscular atrophy (affecting chiefly the deltoid) reaches its highest degree of development.

Etiology.—(*a*) *Age* predisposes to the affection. Though it may appear at any age, the greatest number of cases is furnished by the years from forty to sixty. (*b*) *Sex* exerts a slight influence, the disease being observed most frequently among females. (*c*) *External agencies*, as poverty and occupations which entail exposure to cold and dampness, act as predisposing influences. (*d*) *Heredity* may operate to favor its development.

Symptoms.—The involved joints may not present any visible evidences of disease, and perhaps the most prominent local symptom is *pain*, increased often at night as well as by approaching cold or damp weather. Both the larger and smaller joints are involved, though the former to a greater degree, and yet, though usually multiple, the disease may be limited to one joint (knee, hip, shoulder, etc.). The joints are somewhat *swollen*, as a rule, at times slightly reddened, tender upon pressure, and their mobility is generally restricted. Pain and *stiffness* e most marked in the morning hours (after rest), and often largely isappear with each returning evening (after use). All the local sympms are subject to exacerbations and remissions. A peculiar *crepitation* may be elicited on applying the hand over the affected joints durng motion, and eventually ankylosis, with some degree (usually slight) f distortion of the joints, may occur.

The *general features* are usually conspicuous by their absence. No ever is present, and, in most instances, there is no serious impairment f the general health. On the other hand, as the result of constant suffering, a wretched general condition with marked anemia and debility may finally be reached, such patients often passing sleepless nights and suffering severely from dyspepsia. Chronic endocarditis may develop along with the chronic articular changes—a not uncommon association, though frequently the history of a previous attack of acute rheumatism is also obtainable, to which the endocarditis may be attributed (for the differential diagnosis of this disease *vide* Arthritis Deformans).

Prognosis.—Full recovery is, with but few exceptions, out of the question. A cure may rarely be effected if the case come under appropriate treatment in the incipient stage. The disease, however, rarely shortens the duration of life, though it may do so by interfering with the nutritive processes, the latter effect resulting from loss of sleep (due to pain) and inability to take active exercise.

Treatment.—(*a*) **The local measures** hold first place. The affected joints should be enveloped in flannel at all times, and underneath the latter may be applied cold cloths, and the whole covered with oiled silk. On the other hand, sponging the joints frequently with hot water also furnishes good results, relieving decidedly the pain and stiffness. Bier, A. Graham Reed, and others, employed a hot-air treatment with good effects. Blisters have been employed. In removing effusions they are most efficacious. In the absence of synovial effusion the thermo-cautery is to be preferred to blisters, and for the swelling and stiffness massage with passive movement affords excellent results. Massage is also valuable when atrophy of the adjacent muscles exists; and in these so-called "rheumatic paralyses" electricity is an important help. Iodin and stimulating liniments are more or less serviceable.

(*b*) **Hygienic Measures.**—The diet should be abundant and nourishing; it may embrace milk, eggs, the lighter forms of meat, fats, farinaceous articles, and cruciferous vegetables; wines and alcohol may be permitted. Dietetic abuses, however, tend to aggravate the arthritic condition. The patient should adopt and continue moderately active exercise until compelled to omit it on account of the advancing joint-lesions. Cold spongings of the skin-surface, followed by active friction, have a good effect in that they lessen cutaneous sensitiveness.

(*c*) **Internal remedies** do not control the morbid process directly, although arsenic, iodin, potassium iodid, guaiacol, and other agents are much used for this purpose, but their effects are usually limited, and never brilliant. It should be our aim to maintain the general health at a maximum level by the employment not only of the sanitary means before alluded to, but also by tonics (iron, quinin, strychnin, etc.). I have found a course of cod-liver oil, continued for a long period of time, the most serviceable form of internal medication.

(*d*) In general terms **hydrotherapy** is an important adjuvant to the treatment.

The thermal springs whose waters are alkaline or contain sulphur, and of which the hot springs of Arkansas and Virginia, and the Richfield Springs, New York, furnish good examples, have been strongly advocated, and sometimes prove curative in their effects. I have seen excellent results from the methodic use of hot-water baths at a constant temperature (100° to 105° F.—37.7° to 40.5° C.), combined with passive motion and careful manipulation of the affected parts. If the latter be adopted, every precaution must be used to avoid exposure to cold or draft during and after the baths, which should not be prolonged beyond ten minutes.

WEIL'S DISEASE.

(*Acute Febrile Jaundice; Fiedler's Disease.*)

Definition.—An acute febrile disease, probably specific in origin, and characterized by jaundice, remittent fever, and muscular pains. It usually runs a definite course and terminates by lysis.

Pathology.—During the comparatively recent studies of the post-mortem lesions occurring in this disease very little has been noted. The liver and spleen are sometimes the seat of an active hyperemia, and occasionally some gastro-intestinal irritation is present. The cortical substance of the kidneys is swollen and mottled, and the epithelium of the tubules and glomeruli shows cloudy swelling.

Etiology.—The special organism of the disease is unknown; indeed, it may be an acute febrile jaundice of varied etiology. Jaeger claims that it is due to infection by the *bacillus proteus fluorescens.*

Predisposing Causes.—Among these may be mentioned the following:

(*a*) *Age.*—The age of the patient usually varies from twenty to forty years. A. Holz records a case in a woman fifty-one years old.

(*b*) *Occupation.*—*Butchers* are most commonly affected.

(*c*) *Sex and Season.*—Most of the recorded cases occurred in males and during the summer months.

(*d*) *Locality.*—The cases have appeared in groups, in both rural and urban localities.

Symptoms.—The disease is usually ushered in by a *chill,* followed by *fever, headache,* and *pain* in the muscles, joints, and epigastrium. *Jaundice* usually appears on the second day, and may either be slight or very intense; if it be due to obstruction, the stools are gray-colored, showing the absence of bile. The fever is of the remittent type, running from ten to fourteen days and terminating by lysis. Nausea, vomiting, and diarrhea may rarely occur. The *liver* and *spleen* are often enlarged, the latter being tender on pressure. The *urine* is febrile, high-colored, and often shows the presence of albumin, with tube-casts and sometimes blood (hemoglobinuria). In grave (but rare) cases *cerebral symptoms,* such as delirium, convulsions, and coma, may occur and prove fatal.

The **diagnosis** rests on the acute onset, fever, pains in the muscles ints, and epigastrium, nephritis, and icterus. *Schlammfieber*, which evailed mainly among young persons who had worked in the recently oded districts near Breslau during the summer of 1891, and assumed pidemic proportions, has not been satisfactorily classified. Müller hows its resemblance in many respects to Weil's disease, which may ccur at times without jaundice (?).

Prognosis.—The prognosis, both as to life and recovery, is good. W. E. Hughes, notwithstanding, records two cases that proved fatal within forty-eight hours of the onset.

The **treatment** is purely symptomatic.

MALTA FEVER.

(*Mediterranean Fever; Rock Fever; Undulant Fever.*)

Definition.—A protracted infectious disease, caused by the *micrococcus melitensis*, and characterized clinically by irregular fever, copious sweats, rheumatoid pains, and frequent relapses.

History.—It is *endemic* in Malta, and from time to time is encountered there, and at other Mediterranean ports, in *epidemic* form. Owing to observations made by Wright on the serum reaction, this disease has been shown to exist in India, Hong Kong, the United States, the West Indies, and Brazil. Kinyoun first suspected the presence of Malta fever on this side of the Atlantic, along the coast and in the islands of the Gulf of Mexico. Recently, Musser and Sailer[1] recognized the affection in Philadelphia in a soldier who had come from Porto Rico. No essential *pathologic lesions* have been identified with the disease. Hughes[2] noted an enlargement of the spleen and of the mesenteric glands, and grave cases exhibited bronchitis or broncho-pneumonia.

Etiology.—**Bacteriology.**—The *micrococcus melitensis* (Bruce) has been found in certain tissues (the spleen in all fatal cases), and is readily recognized morphologically and by culture. Bruce, in two cases, and Hughes, in four, reproduced the disease in monkeys by the inoculation of pure cultures of the organism. Young, healthy adults are principally affected, and antihygienic conditions increase morbidity.

The **incubation-period** lasts from a few days to twenty or thirty.

Symptoms.—The disease is of *slow* and *gradual* development, and the features simulate those of beginning typhoid fever. Headache, anorexia, malaise, and slight fever (often preceded by shiverings); the face may be congested, and epistaxis may be present. The bowels are constipated, and the stools may be blood-streaked. The spleen is always enlarged, and frequently painful, particularly on pressure. There is *fever* of a remittent type, with undulating course, lasting one, two, or three weeks; this, after an apyrexial period of two or three days, is followed by a relapse, with rigors, higher fever, delirium, and sometimes by diarrhea and increased prostration.

The *relapse* frequently lasts from five to six weeks, and then, after a week or two, a second relapse may ensue; symptoms somewhat similar to the first—rigors, intermittent form of fever, extreme prostration, and general rheumatoid symptoms. The latter may be so well marked as to prohibit muscular movements of any kind. The case now either terminates in recovery or, after the lapse of one or even two months, there may be a repetition of the whole symptom-complex. In *grave cases* the temperature is continuous, and death may occur in hyperpyrexia (Hughes). Certain *complications*, as touches of pleurisy and pneumonia, rarely appear.

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[1] *Philada. Med. Journ.*, Dec. 31, 1898.

[2] *Annales de l'Institut Pasteur.*

Diagnosis.—From the use of pure cultures of the special organism the blood of Malta-fever patients gives a typical agglutination. Thus the affection is with ease and certainty distinguished from typhoid fever and erratic forms of malaria. In no other manner can it be discriminated from typhoid fever in the earlier stages. If malaria is suspected, a microscopic examination of the blood should be made.

Duration and Prognosis.—Soldiers show an average stay in the hospital of ninety days (Bruce); the duration in obstinate cases, however, may exceed six months. The death-rate is low—about 2 per cent.

Treatment.—This should be sustentative or supportive, in view of the uncertain, protracted course. Nourishing liquids and usually stimulants are required during the febrile stage. Fever is to be combated by the application of cold (cold bath, wet pack, or sponging). Fitzgerald and Ewart[1] successfully treated a case with Malta fever antitoxin. Tonics, coupled with a change of climate, favor convalescence.

FEBRICULA.

(*Simple Continued Fever; Ephemeral Fever.*)

Definition.—A brief febrile attack, unattended with definite local lesions; and of varied, often indeterminate etiology. A true ephemeral fever is one that lasts about twenty-four hours, while the term simple continued fever or febricula is given to cases lasting a longer period.

The cases are diversified with reference to their **etiology** and clinical relations, but may be roughly grouped under several heads:

(*a*) A large group of cases in which a *gastro-intestinal disturbance* is the only assignable cause. The latter may be due to cold or more often to errors in diet (particularly the use of tainted food-stuffs), accompanied by absorption of ptomaines, or it may assume the form of gastro-intestinal catarrh met with in young children.

(*b*) Undeveloped or abortive forms of the *infectious diseases* (typhoid, typhus, rheumatism). These affections, particularly during times of epidemic prevalence, may run a brief course without manifesting any of their distinctive characters. This is particularly true of the abortive types of typhoid, and other acute infections. Again, diseases that ordinarily manifest a characteristic eruption (*e. g.*, scarlet fever, measles, erysipelas) may run their course without doing so, or the eruption may escape observation.

(*c*) It may follow *exposure* to the summer sun or *excessive heat* (?), or *exhaustion of the nervous system*.

(*d*) It is not infrequently the result of a slight and unnoticed *localized inflammation* (tonsillitis, bronchitis, lymphadenitis, etc.).

(*e*) The *inhalation of sewer-gas or other noxious vapors* (such as emanations from decomposing organic matter) may produce an aberrant form of the fever (*vide* Septicemia).

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[1] *The Lancet*, April 15, 1899.

Symptoms.—It is to be remembered at the outset that a single symptom, peculiar to all cases, is the *fever.* The *onset* is generally sudden, and especially in ephemeral fever, but it may be gradual; if sudden, there is rarely either a chill or vomiting, while in neurotic children a convulsion may occur. The temperature ascends quickly to 102°–103° F. (39.4° C.) or over, pursues the continued type, and at the end of one, two, or more days subsides abruptly by crisis. There are accompanying symptoms, many of which are due to the fever, such as headache, hebetude, mild delirium, flushed countenance, a full, rapid pulse, anorexia, constipation, scanty, high-colored urine, and, not rarely, herpes labialis. Defervescence may be attended with critical sweats, diarrhea, or a copious flow of urine. *Special types* (*e. g.*, cerebral, gastric, gastro-intestinal) may be observed, due to the predominance of the symptoms presented by individual organs or systems.

In another class of cases the access of simple fever may be less sudden, the maximum level attained being somewhat lower and the attending phenomena less acute and pronounced. Da Costa[1] has described cases belonging to this category. The *course* is more protracted, though rarely exceeding a week or ten days, and the defervescence is not so abrupt. So-called *thermic fever* is at the present writing believed by Guitéras, who first described it, to be due to a special, though as yet unknown, organism.

The **diagnosis** necessitates the exclusion of other acute fevers. The affections from which it is most difficult to distinguish febricula are *typhoid fever*, *remittent fever*, *scarlet fever*, *incipient tuberculosis*, *larval pneumonia*, and *meningitis* (in children). In febricula, however, there is an absence of local manifestations and of physical signs pointing to consolidation of the lungs; characteristic skin-eruptions are also absent. Tyson points out that in cases in which there is splenic enlargement (rare) the resemblance to typhoid is close, and the diagnosis may have to remain in doubt until settled by the Widal test or by time. The cases must also be discriminated from the fever which sometimes attends chlorosis and certain nervous disorders.

The **prognosis** is good.

Treatment.—Few cases require treatment other than rest in bed and liquid nourishment for several days. Cooling drafts internally, and mild forms of hydrotherapy (spongings, ice-caps) externally, are indicated. If traceable to gastro-intestinal disturbance, a laxative usually proves beneficial and effective. It should be followed by intestinal antiseptics. Unless it is clear that the given case is non-infectious and non-contagious, isolation of the patient should be ensured.

MILK-SICKNESS.

Definition.—A peculiar infectious disease, occurring both in man and in the lower animals, when it is known as "trembles." The disease is unknown east of the Alleghany Mountains, but throughout many of the Western and South-western States it formerly prevailed very extensively, with fatal effect. It has, however, been almost exterminated as the result of denudation of the forests and the advancing cultivation of the virgin soil. It still prevails in parts of North Carolina (Osler), and until very recent times has been seen in certain parts of Illinois.

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[1] *Transactions of the Association of American Physicians*, vol. xi., 1896.

No peculiar pathologic lesions have been described.

Etiology.—It is believed to be due to a special poison derived from the earth, but as yet we are ignorant of its exact nature. Phillips claims to have found a spirillum in the blood.

Modes of Infection.—The disease attacks cattle most frequently (especially unweaned calves), horses, sheep, goats, and less often many undomesticated animals; wherever trembles prevails among cattle, milk-sickness is met with in man. It is thought that the poison is communicated to man in the milk, butter, and cheese, or in the flesh of infected animals.

Among disposing factors are the *seasons*, the disease being most frequent in the late summer and autumn. It is most common in adult life.

Symptoms.—The period of *incubation* may be short or long in duration, and *prodromata*, such as headache, anorexia, languor, and oncoming fatigue, may be noted. These symptoms increase in severity, and are soon eclipsed by the more characteristic features—nausea and vomiting, a hot pain in the stomach, and a peculiar fetor of the breath. There is an unquenchable thirst, a swollen, tremulous tongue, and absolute constipation. Fever is present, but it is slight, and the surface-temperature is often below the normal. The nervous symptoms include restlessness, merging into mental dulness with marked indifference, and the latter condition passing in grave cases into a stupor that may deepen into actual coma. Convulsions may arise or the patient may drop into a fatal typhoid state.

The **diagnosis** rests chiefly upon the history (particularly upon the coexistence of "trembles" in cattle) and the exclusion of other acute intoxications.

The **prognosis** is generally favorable, though a fatal termination due to asthenia may occur within a few days of the time of the onset.

Treatment.—*Prophylaxis* consists in the avoidance of those foods that act as bearers of the disease. Apart from the use of supporting measures (appropriate diet and stimulants), we can attend only to the symptomatic indications. Medicated enemata should not be omitted.

MILIARY FEVER.

(*Sweating Sickness.*)

Definition.—An infectious disease, characterized by copious sweats and a vesicular (miliary) eruption. In certain countries it has prevailed epidemically (France, England, Italy, Germany), and in 1887 a severe epidemic occurred in France. Schaffer[1] reports the occurrence of a recent epidemic in an Austrian province in the spring of 1893, lasting for nearly three months. Out of 5079 persons (the total population of the district), 159 suffered, as follows: 17 men, 14 women, and 128 children. At the present day it seems to be met with only in Picardy, in a few other French provinces, and throughout a limited area in Italy.

Neither have definite *pathologic lesions* nor the *specific exciting* cause been found. Among *predisposing influences* the following have been noted: (*a*) Most epidemics occur in spring and summer; (*b*) It is more common among women than men, and most frequent during the middle period of life. A large percentage of the entire population of an invaded district (usually limited in area) is attacked.

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[1] *Weiner med. Blätter*, 1893, No. 32.

The **symptoms** that characterize miliary fever are *fever* with its usual accompaniments, irritation of the skin, epigastric oppression, copious and persistent sweating, followed, on the third or fourth day of the disease, by an *eruption* (due to profuse sweatings) of miliary vesicles.

The vesicles burst, and within forty-eight hours scaly desquamation is generally completed. In severe types the nervous phenomena (delirium, etc.) are grave in character; hemorrhages may occur, and at times fatal collapse may follow. *Relapses* are not uncommon.

The **prognosis** is affected largely by the character of the epidemic, the average death-rate being 8 or 9 per cent.

Quinin has met with almost universal favor as a remedy, but the expectant plan of **treatment** is the most appropriate, the indications being fulfilled as they arise. The sweating may demand atropin.

FOOT-AND-MOUTH DISEASE.

(*Epidemic Stomatitis; Aphthous Fever.*)

Definition.—An acute infection of certain lower animals (cattle, sheep, pigs, goats), caused by a micro-organism as yet undiscovered, although Klein has described a micrococcus. It is characterized by fever, by the appearance of vesicles and ulcers in the mucosa of the mouth, in the furrows about the feet and on the udder, and by the rapid development of asthenia and marked emaciation. Though a disease of mild character, its territorial range is so vast as to entail untold loss to European countries. Young animals or sucklings perish in great numbers on account of the deteriorated quality of the milk, which assumes a yellowish-white appearance and has a bitter, nauseating taste.

During epidemics of foot-and-mouth disease the poison may be transferred to man, in whom the disease is known as *epidemic stomatitis*, the poison generally being transferred by means of milk. Boiling the latter destroys the virus, but rarely the infection may be transmitted through butter and cheese made from the milk of infected cattle. Communication by inoculation (while milking) may also occur. The disease does not seem to be transmissible through the meat of diseased animals.[1] In America a few instances only of transference from animals to man are recorded.

Symptoms.—The *incubation-period* lasts from three to five days. A rigor may mark the onset or merely slight shiverings, followed by fever and malaise, and soon vesicles, such as are described under Aphthous Stomatitis, appear upon the tongue and inner surface of the lips. The mouth is hot, the mucosa reddened and swollen, and salivation is present. A form of miliary eruption that may become pustular may also appear on the skin-surface, and particularly on the fingers and hands. Hemorrhages have been observed in severe epidemics.

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[1] Zuell's translation of Friedberger and Fröhner's *Pathology and Therapeutics of the Domestic Animals.*

The **diagnosis** is made with ease if the disease be prevailing at the same time among lower animals. The peculiar coincidence of the eruption in the mouth and extremities, sparing the rest of the body, has not been noticed in any other eruptive disease (Whittaker).

Course and Prognosis.—The course is mild and ends in about one week, the disease being very rarely fatal.

Treatment.—*Prophylaxis* requires the use of milk from healthy animals (cows or goats), together with measures looking to the care of the stables and isolation of diseased cattle. A reliable method of immunization against foot-and-mouth disease has not as yet been discovered.[1] For *treatment* the reader is referred to the article on Aphthous Stomatitis.

GLANDULAR FEVER.

Definition.—By this term is meant an acute infectious disease of children, characterized by adenitis affecting the lymph-glands of the neck, especially the anterior cervical.

History.—A detailed description of glandular fever was first given by E. Pfeiffer, in 1889, under the name of *Drüsenfieber*, but it had probably been previously described by Filatow, of Moscow. Donkin, Fischer and Dawson Williams, in England, and J. Park West have given excellent descriptions of the disease.

Pathology.—The anterior cervical lymphatic glands are involved first, and it is "probable that the infection finds its point of entrance through either the tonsils or the pharyngeal mucous membrane" (Williams). The adenitis may also affect the inguinal and axillary glands.

Etiology.—The special micro-organism of the disease is unknown. The complaint occurs usually in the form of house-epidemics. West, of Ohio, however, has described the most widespread epidemic hitherto recorded. There were 96 cases in 43 families, and rarely did a child exposed to the infection escape. The disease usually occurs during childhood; the ages of West's cases ranging from seven months to thirteen years. A. E. Roussel has reported four cases, one occurring in an adult. Most cases occur between the months of October and May, inclusive. According to Hand, the weight of clinical evidence tends to variation in the etiology in different cases (*e. g.*, it is often one of the protean manifestations of influenza).

The incubation-period lasts usually from five to eight days.

Symptoms.—The *onset* is *sudden*. The child holds the neck stiffly, since movement causes pain; there are anorexia, nausea, and less commonly vomiting, the bowels are constipated, and often there is abdominal pain. The child may complain of pain and swelling; an examination of the pharynx may show some chronic enlargement of the tonsils, and in some cases injection of the pharyngeal mucosa, actual pharyngitis being rare. The temperature oscillates from 101° to 103°. Nervous symptoms (delirium, hebetude) are rarely observed.

The *glandular enlargement* becomes obvious on the second or third day, and in most cases is observed first on the left side, then, after a few ays, on the other side of the neck also. The glands vary in size from bean to a hen's egg, and are painful on palpation. They rarely suppurate. Other groups of glands (axillary, inguinal) may be successively involved. Cough and dyspnea may point to involvement of the bronchial and tracheal glands. The mesentery glands were enlarged in 38.5 per cent. of West's cases. Splenic enlargement occurs in 50 per cent. of the cases, while the liver is voluminous in almost all cases. The average *duration* is sixteen days (West). Among *complications* may be mentioned hemorrhagic nephritis, bronchitis, and otitis media.

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[1] Siegel, "Experiments in Immunization against the Poison of Bites and Scratches." Quoted in the *Philadelphia Med. Jour.*, January 28, 1899.

Diagnosis.—The recognition of glandular fever embraces the exclusion of such affections as *tonsillitis*, *pharyngitis*, and *influenza*, in the course of which adenitis might arise. Griffith[1] has reported cases resembling glandular fever in which influenza was probably the sole disease present.

Prognosis.—Recovery is the rule.

Treatment.—The course of the disease is probably uninfluenced by treatment. Locally, cold compresses and fomentations are useful. Internally, West advises castor-oil in the early stage, followed by minute doses of calomel (gr. $\frac{1}{12}$ to $\frac{1}{10}$) twice or thrice a day.

[1] *Univ. Med. Magazine*, October, 1900.

PART II.

CONSTITUTIONAL DISEASES.

DIABETES.

(*Diabetes Mellitus.*)

Definition.—A nutritional affection, attended by an abnormal amount of sugar in the blood, and characterized clinically by persistent glycosuria, by polyuria, and by a progressive loss of flesh and strength.

Pathogenesis.—This is still undetermined. Post-mortem lesions of different organs and structures of the body have been met with in diabetes—a fact that has given rise to a variety of views.

(1) That it is dependent upon *organic disease of the pancreas*, especially granular atrophy, or upon marked functional disturbance of this organ. It has been shown experimentally that extirpation of the pancreas is followed by diabetes, and yet, according to Minkowski and Lépine, if a small portion remains glycosuria does not result. On the other hand, Sandmeyer extirpàted the pancreas of two dogs, leaving from one-ninth to one-fifth of the organ. The animals became diabetic—one four and the other thirteen months after the operation—and the first dog succumbed two months and the other eight months later.

It may safely be assumed that total loss of function always, and partial loss sometimes, leads to diabetes. Again, from the observations of Hansemann in the Berlin Pathological Institute and the Augusta Hospital, it would seem certain that the coincidence of pancreatic disease and diabetes occurs oftener than either diabetes or pancreatic disease alone, and, in truth, oftener than both these separate affections combined. Lépine and Martz have been able to produce a glycolitic ferment by treating the pancreas after their own special method, which need not be detailed here. This ferment is identical with that which is contained in the blood, and in the presence of which glycogen is assimilated; pancreatic diabetes occurs, therefore, when through organic disease or functional disturbance the formation of this ferment is wholly or even partly arrested. Another source of the glycolitic ferment is the salivary secretion.

(2) If the glycogenic function of *the liver* be interfered with materially, diabetes follows. This may result from organic hepatic disease or the fault may lie solely with the nervous system. Puncture of the floor of the fourth ventricle will also cause glycosuria, and section of the pneumogastric nerve is followed by vaso-motor paralysis of the hepatic vessels, disappearance of glycogen from the liver, and the appear-

nce of sugar in the urine. This view explains how the cord and entral lesions and disturbance of the sympathetic system produce diabetes.

(3) The so-called *alimentary glycosuria* has frequently been induced experimentally by Miura and others. It results from the ingestion of more carbohydrates and peptone than can be stored in the liver as glycogen, so that some of the latter finds its way into the hepatic vessels with consecutive glycosuria.

(4) The administration of *phloridzin* produces glycosuria both in animals and man. There are two views as to the cause of phloridzin diabetes: (*a*) that the kidneys, owing to the action of the phloridzin on the renal epithelium, eliminate the sugar from the organism; (*b*) that an excessive formation of glucose occurs. The condition of the blood in phloridzin glycosuria testifies to the decomposition of proteids rather than to the mere elimination of sugar (Lépine).

(5) *The Microbic Theory.*—Paul Ernst and others have observed all forms of fungi in diabetes, thus showing that the disease is favorable to the development of various micro-organisms, but as yet none have been shown to sustain an etiologic relationship.

(6) Pavy's view regarding diabetes, recently advanced, is that the carbohydrates of the food are converted into fat by the protoplasmic action of cells in the intestinal villi, and enter the system in the same way as do fats taken as such. The surplus carbohydrates that escape the action of the cells of the villi are transmuted into glycogen in the liver. The glycogen stored in the liver obviously forms fat also, since this organ has some fat-forming function, and there are thus two barriers preventing the carbohydrate matter from entering into the blood, and if either is deranged hyperglykemia with the consequent glycosuria results.

No single view explains all cases of diabetes. The influence of the nervous system, however, is undoubted. In light cases the carbohydrates are not warehoused in the liver and muscles, and the excess of glycogen not burned up in the tissues, so that from the storage reservoirs (liver and muscles) the blood is supplied with an excess of grape sugar. In severe cases the carbohydrate moiety of the proteids of the food or tissues furnishes the sugar excreted in the urine (Stengel). Lastly, the precise form of disturbance of metabolism that prevents a normal disposal of carbohydrates is unknown.

Pathology.—The *pancreas* in more than one-half the instances shows morbid changes. The most frequent lesion is the granular atrophy of Hansemann, occurring in 36 out of 40 cases of pancreatic diabetes. Bard and Pic found 150 cases of primary cancer of the pancreas on record, and in only 17 was glycosuria noted. Fibroid induration of the pancreas is sometimes observed in diabetics, and is due to syphilis. Diffuse cancer of the organ may lead to diabetes, but not readily, Hansemann having observed 2 cases, and I also 2, without diabetes. Calculus, with atrophy, may or may not be associated with diabetes. Occlusion of the pancreatic duct, atrophy from pressure, and cystic degeneration may be met in this disease. Of 100 cases of acute necrosis of the pancreas collected by Fitz and Seitz, only 2 had diabetes.

The *liver* is often enlarged and fatty, particularly the zones corresponding to the distribution of the hepatic artery. According to French writers, there is a diabetic cirrhosis of the organ (*cirrhose pigmentaire*), the pigment being derived from destroyed blood-cells. I observed an instance associated with syphilitic hepatitis. *Microscopically*, the liver-cells are found to be enlarged, nucleated, and globular in outline. Rindfleisch holds that these changes are most striking in the peripheral portion of the lobule, while in the central zone they are slight.

The Kidneys.—A well-marked chronic interstitial nephritis, with fatty degeneration, is often present. The tubal epithelium and the vessels of the Malpighian bodies may show a hyaline change. More commonly the appearances are those of an ordinary catarrhal nephritis consecutive to persistent hyperemia.

Nervous System.—In rare instances organic disease of the medulla (tumors, sclerosis, etc.) is found. Changes in the posterior columns of the cord have been noted, and a peripheral neuritis, simple or multiple, is commonly though not constantly seen. The so-called *diabetic tabes* is generally supposed to be due to multiple neuritis. Sclerosis and enlargement of the ganglia of the sympathetic system have been noted in a few cases.

The Lungs.—The commonest lesions in the lungs are gangrene following pneumonia and the so-called diabetic phthisis. Fatty emboli are found in the pulmonary vessels.

The Heart.—Arterio-sclerosis with cardiac hypertrophy is often met with, but does not constitute a peculiar lesion.

The Skin.—Cutaneous pigmentation (diabetic bronze of the French), more or less uniform, has been reported in 9 cases (Hanot and Chauffard). It is associated with hypertrophic cirrhosis of the liver.

The Stomach.—Dilatation and, according to Jacobson, marked catarrhal changes are common in the early stage.

The Blood.—The normal proportion of sugar in the blood (0.15 per cent.) is usually increased, though there is no immediate connection between the percentage of sugar in the blood and in the urine in diabetes. Both in experimental and pathologic diabetes hyperglykemia may be marked, with moderate or slight glycosuria, and Lépine has shown that diuretics diminish hyperglykemia by increasing the glycosuria. The blood-plasma contains much fat. It is probable that the albuminoid matters in the blood may produce glucose. Glycogen probably exists in the blood-corpuscles, and not in the plasma, "where it would be destroyed by the diastasic ferment" (Dastre), and it is a normal element of the blood, apparently belonging to the leukocytes (Huppert and Czerny). The corpuscles show no special alterations.

General Etiology.—(*a*) *Heredity* is generally believed to exert a predisposing influence, since cases are observed to succeed one another in the same family. (*b*) *Season* also exerts an influence, diabetes appearing more frequently in the months of March, April, July, and November (Davis). (*c*) *The male sex* suffers much more frequently than the female. Wegeli, however, found in 107 cases that children of both sexes were affected in an equal proportion. (*d*) *Age.*—Most cases occur between thirty-five and sixty years of age. Infantile diabetes is rare, and occurs most frequently about the age of five, though it has been met with under one year. (*e*) *The Hebrew race* is especially susceptible. The colored race rarely suffers, although of a series of 77 cases 8, or 10.8 per cent., were in negroes (Futcher). (*f*) *The better classes* of society furnish most instances, and particularly that large element composed of neurotic subjects. (*g*) *A nervous shock* or strain or prolonged mental anxiety acts as a predisposing cause. (*h*) *Occupation.*—The urine of 607 individuals engaged in manual labor that required great muscular and respiratory activity showed no sugar in any case; while the urine of 100 individuals engaged in intellectual work of a more or less fatiguing character, but always intense and sedentary, showed sugar in 10 of the cases (Worms). (*i*) *Obesity* predisposes, somewhat, particularly to the lipogenic form. (*j*) *Certain chronic diseases—e. g.* syphilis, malaria, gout—predispose. (*k*) *Pregnancy* has a slight though decisive influence. (*l*) It sometimes follows *acute infectious diseases.* (*m*) *Locality.*—Diabetes mellitus is, comparatively speaking, rare in America, although Hare's statistics indicate that diabetes is becoming more prevalent. In certain other countries (Normandy, India, France) diabetics appear to be constantly increasing in number, the mortality in Paris having more than doubled from 1883 to 1892, inclusive. The disease is much more frequent in cities than in rural districts. *Contagion.*—Among 770 cases observed by Senator there were 9 instances of man and wife suffering from the disease. In a series of 5,000 cases ·1.8 per cent. of conjugal diabetes occurred (Schram).

Special Etiology.—Under this head may be arranged the following groups of cases: (1) *Diabetes* due to *pancreatic disease.* (2) Cases occasioned by *hepatic disease* (*organic and functional*). (3) Those comparatively rare instances caused by *disease of the brain* (*tumors, sclerosis, or irritative lesions of the diabetic center*) *and spinal cord.* (4) Diabetes follows *traumatism*, and especially injuries to the head. Not infrequently it occurs after injuries to other parts of the body, such as the spine, sacral region, abdomen, etc. In 212 cases of traumatism of the head Higgins and Ogden found 20 cases of glycosuria, though only a small proportion of the cases (2) exhibited a permanent glycosuria from the date of injury. Ebstein,[1] after an exhaustive study of 6 of his own cases and of 44 gathered from literature, concludes that there can be no question of the direct causal relation of traumatic neurosis and diabetes.

Clinical History.—For the sake of accuracy and convenience of description, the cases will be divided into the acute and chronic forms.

1. **Acute Diabetes Mellitus.**—The instances are few and the course is, as a rule, rather subacute than acute, manifesting a predilection for the young and middle-aged. The *onset* is more abrupt than in the chronic form, but the characteristic features do not differ from those of the latter. Many of the cases due to pancreatic disease are of this class. Exceptionally, acute diabetes occurs in the aged.

2. **Chronic Diabetes.**—The symptoms are evolved *slowly* and *gradually*, as a rule, and prominent among prodromal conditions is dyspepsia or chronic gastric catarrh. We may also note certain nervous disorders, such as headache, mental irritability, moroseness, and insomnia, with or without gastro-intestinal symptoms. In some cases the patient suffers merely from general debility and malaise, and either frequent micturi-

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[1] *Deutsche Arch. f. klin. Med.*, April, 1895.

tion, polyuria, or unnatural thirst is apt to be noticed. Rarely, diabetes has an *abrupt onset*, as after an injury, a sudden nervous shock, or a chill. With the development of the affection the polyuria becomes marked, as a rule, the thirst great, the appetite keen, and glycosuria appears. In spite of the enormous quantities of food taken, progressive emaciation and debility attend.

Leading Symptoms and Complications in Detail.—(1) *The Urinary Symptoms.*—The daily amount of urine varies from four or five pints to as many gallons. In very mild cases and in intercurrent febrile attacks it may be slightly, if at all, increased in quantity. Its color is pale, and its specific gravity ranges from 1020 to 1050, rarely being as low as 1015; it has an acid reaction, a sweetish, aromatic odor, and a distinctly sweetish taste. Sugar is present, the amount varying from ½ of 1 per cent. to 2 per cent. in mild cases, to 5 or even 10 per cent. in severe attacks. The total amount eliminated in the twenty-four hours varies from five or ten ounces to a pound or more.

Other forms of sugar than glucose (inosite and levulose) may be contained in the urine, and glycogen has rarely been found. The urine may also contain fermentation-products (acetone, diacetic acid, beta-oxybutyric acid). Acetone strikes a Burgundy-red color on the addition of the chlorid of iron. Hirschfeld's studies upon the excretion of acetone in diabetics show that in severe forms an increased amount is excreted, while other writers regard it as being benign. Diacetic acid is probably of graver significance than acetone, whilst the presence of β-oxybutyric acid is a danger-signal of diabetic coma (*vide infra*).

The *urea* is greatly increased, Kaufman finding the quantity in the blood of diabetic dogs to be doubled. *Uric acid* is either normal in quantity or slightly diminished, but a large amount of ammonium is present, indicating an increase of organic acids. The phosphates may also be present in greatly increased proportion (Ralfe), and in such cases the glycosuria may be more or less intermittent. This has been described as a special variety—phosphatic diabetes. Lipuria may be present, and creatinin is increased.

Slight albuminuria, often with an intermittent tendency, is common even in the early stages, and is not of grave significance. Well-marked nephritis with its characteristic phenomena may develop, though usually in advanced diabetes; and if albuminuria be marked, the amount of sugar excreted may be considerably diminished. The development of chronic interstitial nephritis, however, is not a favorable complication, as some have supposed. With or without nephritis, arterio-sclerosis may be observed, and pyelo-nephritis (rarely) and cystitis (not rarely) may appear as complications. As the result of fermentative processes in the bladder gases may form (*pneumaturia*).

(2) *Digestive Symptoms.*—Although a general feature, *thirst* may be discussed under this head. This symptom may be most distressing, necessitating the drinking of large quantities of water at frequent intervals both by night and by day. The amount of water taken stands in direct relation to the amount eliminated. Notwithstanding the fact that the increased amount of water is needed to dissolve the sugar, cases of confirmed diabetes are met with in which thirst is not marked. Cases are also encountered in which the amount of urine is large and the percent-

age of sugar excreted very low. The cause of the unusual thirst is not quite clear, though it is probable that the chief factor is the increased systemic demand for liquids.

The *appetite* is abnormally large and sometimes almost insatiable (bulimia), and there may be an intense craving for carbohydrates. I have, however, met with two instances of well-developed diabetes in which the appetite was not inordinate. Considering the quantity of food consumed, the digestion is often surprisingly good, but the association of dyspepsia and diabetes is by no means an uncommon one. The stomach may be found enormously dilated at times, yet functionating normally. As a rule, there is constipation, though brief intervening attacks of diarrhea may occur.

The *tongue* is generally dry, large, often presenting a rough and fissured surface, and it may either be coated or red and glazed. The gums sometimes swell, and may ooze blood. The saliva is scanty and its reaction persistently acid, while the salivary secretion may show sugar on testing. The teeth decay, and aphthous stomatitis or thrush may attack the oral cavity.

The *liver* is frequently somewhat enlarged, though the biliary secretion usually is not disturbed; jaundice may, however, arise as a complication. Marie has given a description of *pigmentary* "*hypertrophic cirrhosis* with diabetes mellitus," of which only 9 undoubted cases have been published. It appears late in adult life, and, in addition to the symptoms of diabetes mellitus, slight ascites, considerable hypertrophy of the liver and spleen, with brown or even gray-black cutaneous pigmentation, are among the chief features noted. There is no true icterus as a rule, but the urine is highly colored and contains bile-pigments.

(3) *Cutaneous Manifestations.*—Diabetic urine, on account of the sugar it contains, has irritant properties, and often produces in the female *pruritus vulvæ*, a most troublesome symptom and one that should always excite suspicion of this disease. In the male, *balanitis* often occurs, due to the effect of the decomposing urine, and from the same cause the genitals and adjacent cutaneous surfaces may be the seat of *eczema*. This seems to be more common in women than in men. The skin is usually harsh and dry, though rarely copious perspiration may be observed, and particularly if phthisis be a complication. The hair often falls off, and in one of my cases *onychia* with shedding of the nails occurred. Among the commonest of the early cutaneous symptoms are *furuncles* and *boils*. Later large *carbuncles* often appear. *Gangrene* (especially of the feet) due to arterio-sclerosis is not infrequent, and *edema*, arising independently of nephritis, is not uncommon.

(4) *Nervous Symptoms.*—Diabetic coma is the most important symptom, marking a fatal termination in more than half the cases. It is of most frequent occurrence in instances showing rapid wasting and in the young, and is heralded by a fruity odor in the exhaled breath and in the urine. The polyuria and glycosuria lessen, while acetonuria increases as a rule. The tolerance for the carbo-hydrates is increased (Hirschfeld). The cases may be arranged into the following clinical groups:

Group 1. To this belong abortive forms that terminate in quick recovery. This process may be repeated several times at intervals, and at last a fatal coma may supervene.

Group 2. Perhaps the largest group, in which the diabetic coma follows some form of exhausting exercise. It may end fatally in a few hours or, though less frequently, in three or four days.

Group 3. This is a comparatively small class, and is characterized by collapse of the circulation (small, rapid, feeble pulse, cyanosis, etc.), leading to coma. It is induced either by over-exercise or by intoxication. I have seen 2 typical instances, but feel that it may be questioned whether most of these cases should be classed as diabetic coma.

Group 4. Without previous dyspnea or distress there appear such symptoms as headache and signs of intoxication, and these are followed quickly by deep and fatal coma (Frerichs).

Group 5. Here diabetic coma is preluded by symptoms of some localized disorder, such as gastro-enteritis, pharyngitis, pneumonia, gangrene, or carbuncle. The attack sets in with headache, delirium, distress, and dyspnea both inspiratory and expiratory. Cyanosis may develop early, and, if so, cardiac failure precedes the coma. The duration is from one to five days. This group, which was first described by Frerichs, may have a different onset, and I have seen two fatal cases, one attended by carbuncle, the other with gastric symptoms.

Group 6. Hirschfeld has recently described a class of cases in which we find, in old persons, a moderate glycosuria and coma supervening under the influence of gangrene or carbuncle.

The *causes* of diabetic coma are still obscure. Hirschfeld points to insufficient nutrition from an exclusive meat diet as a factor. Kussmaul believed diabetes to be due to acetone. Kulz, Stadelman, and others have more recently found β-oxybutyric acid in the urine, and this is now generally held to be the immediate excitant of diabetic coma (which is an acid-intoxication). This acid results from an increased destruction of the proteids. Lastly, coma must sometimes be of uremic origin, and Herrick[1] emphasizes the fact that casts are very common. Cases that follow suppuration and gangrene may be septic in nature.

Peripheral neuritis is common. The most frequent form is *diabetic tabes*, indicated by an absence of the knee-jerks, darting pains, paresis of the extensors of the foot, and by the peculiar gait (steppage). Other symptoms pointing to neuritis may be numbness, tingling, and certain trophic disturbances—shedding of the nails and perforating ulcer of the foot. R. T. Williamson found the knee-jerk absent in 25 of 50 cases recorded; and in 18 of 21 cases of diabetic coma. Schupfer[2] attributed absence of patellar reflex to toxic effects in most cases. Neuralgia may be a troublesome symptom, particularly when it is of the symmetrical sciatic type, and it points to neuritis. The same is true of paraplegia, a condition that may be met. Herpes zoster was observed in one of my cases.

Psychopathia (*e. g.* irritability of temper, hypochondriasis) may sometimes be present, and temporary hemiplegia has been noted.

(5) *Special-sense Symptoms.*—Not infrequently cataract develops, leading to blindness. Its cause is not clear. Transient ptosis and strabismus may also appear, and among other ocular conditions are optic-nerve atrophy, retinitis (often due to associated nephritis), and

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[1] *Journ. Amer. Med. Assoc.*, January 26, 1901.

[2] *Soc. Lancisiana Roma*, Jan., 24, 1898.

hemorrhage. Amaurosis is rarely observed. Among the aural symptoms I would mention otalgia, otitis media, and mastoid disease.

(6) *Muscular Symptoms.*—In diabetics there is a tendency to cramps, especially in the calf of the leg, that appears during the night and on waking in the morning. Unschuld found it present in 33 out of 109 cases. Another variety of cramps that may appear at any hour of the day may occur with the so-called "gastric crisis." In these attacks colicky pain in the epigastrium, with vomiting, and febrile reaction attend.

(7) *Respiratory System.*—Serious pulmonary complications may appear in the advanced stages. The most frequent is pulmonary tuberculosis, which has the customary termination, and does not differ from the usual form of the disease. A second, quite frequent complication is gangrene (circumscribed or general). The peculiar offensive odor of the expectoration may be wanting here. A serious form of *pneumonia* (lobar or lobular) sometimes occurs, and may terminate in gangrene.

(8) *Circulatory System.*—The *pulse* may be of natural frequency and tension. In other cases it is somewhat slow, and the tension may be increased. This is often due to an associated arterio-sclerosis. The heart is sometimes quite weak. As to frequency, the pulse varies greatly: it may be slow (brachycardia), not exceeding 40 or 50 beats per minute, or it may be accelerated. Dyspnea, a tendency to syncope, and gastric disturbance may be seen in combination.

(9) *Sexual Symptoms.*—Impotence may be an early symptom; it is often of great diagnostic significance. Diabetes may be acquired during pregnancy: *per contra*, the diabetic may conceive, though rarely, and bear a healthy child; but death of the fœtus occurs in about one-half of the cases. Premature delivery occurs in most cases (26). After delivery the condition is generally aggravated.

(10) *Constitutional Symptoms.*—Usually there is a constantly increasing loss of flesh and strength. In the mildest types, however, good bodily nutrition and a fair degree of strength may be maintained. When emaciation is progressive the polyuria is apt to be proportional. The *temperature* is at first normal, later usually subnormal, though intercurrent febrile attacks, due to complications, are often witnessed.

Clinical Varieties.—(*a*) **Infantile Diabetes.**—Heredity, traumatism, and convalescence from severe acute infectious disease are the most potent causes. The type is severer and the course shorter than in adults. On the other hand, a comparatively mild chronic form is rarely met in children.

(*b*) **Pancreatic Diabetes.**—This is a grave variety, and may present evidences of pancreatic involvement. There may be epigastric pain; the fats are poorly assimilated; and the physical signs may rarely point to pancreatic growth. Fitz[1] states that out of 166 cases treated in the Massachusetts General Hospital fatty stools were not recorded in any instance. Marked polyuria and great thirst may be absent, and albuminuria is rare.

(*c*) **Alimentary or Lipogenic Glycosuria.**—This is caused by dietetic errors, and especially by excesses in eating and drinking, combined with physical inactivity. Block experimented on 50 patients, and found that the amount of grape-sugar that could be given before glycosuria appeared differed widely in different diseases. Frequently the smallest quantity was required in *nervous diseases*, and particularly in cerebral affections. Von Jaksch induced alimentary glycosuria in cases of hysteria and in cases of phosphorus-poisoning with fatty degeneration of the liver. This form of the disease is often a temporary affair. The percentage of sugar in the urine is usually small.

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[1] *Yale Med. Journ.*, March, 1898.

Prognosis.—In acute diabetes the *duration* varies from a few days to eight or ten weeks, while in chronic diabetes the course ranges from one or two to five or even ten years. When the disease commences in the declining period of life, the course is longer still. The severe forms are generally fatal, and occur, as a rule, at an early period of life and in persons with an hereditary taint. The mild types and those that occur later in life offer a more hopeful prognosis, and in certain cases the withdrawal of all carbohydrates from the diet will cause the sugar to disappear from the urine. Of the special varieties, *alimentary glycosuria* is altogether favorable in its course, *traumatic diabetes* somewhat less so, while the prognosis of the *pancreatic form* is quite unfavorable.

Stout persons bear saccharine diabetes better than lean. Pre-existing affections may render the prospect quite gloomy, and certain complications indicate grave danger (coma, phthisis, gangrene, pneumonia, cardiac weakness, nephritis). Of 108 such cases, 64 per cent. terminated fatally (Wegeli), and between the ages of four and five years 20 out of 29 cases perished. Stern's recent statistics show that the mortality-rate from diabetes mellitus in New York per 1000 population has been on the increase since 1894.

Diagnosis.—Diabetes is distinguishable by means of (1) its causal influences and its pathologic antecedents and relations; (2) its gradual onset, often marked by certain suspicious symptoms (*e. g.* debility, impotence, symmetrical sciatica, cataract, furunculosis); (3) the persistent (rarely intermittent) presence of glycosuria, polyuria, and, later, acetonuria and albuminuria; (4) the inordinate thirst and appetite; (5) cutaneous boils, carbuncles, gangrene, pruritus vulvæ in the female, balanitis in the male; (6) neuritis (especially double sciatica), diabetic tabes, and coma; (7) muscular cramps; (8) special complications; and (9) the long course with slowly progressive asthenia and wasting.

In suspicious cases, even before the discovery of sugar in the urine, grape-sugar may be administered for diagnostic purposes. If glycosuria result, the cases are to be treated just as in pure diabetes. Transient glycosuria, however, is not the genuine affection. Grape-sugar must be eliminated for weeks, months, or years (von Noorden).

Blood-test.—Bremer has found that the red corpuscles in a drop of diabetic blood cannot be stained with anilin colors as can those of normal blood. In this respect the behavior of the corpuscles of leukemic and diabetic bloods are identical. Williamson's blood-test, which rests upon the power of diabetic blood to decolorize a weak solution of methylene-blue to a yellow-green or yellow color has confirmatory value.

Treatment.—1. **A properly regulated diet** is of the first importance. Such food-articles as contain starch or sugar (honey, sugar, ordinary flour or bread, biscuits, rusks, toast, arrow-root, oatmeal, cracked wheat, potatoes, tapioca, sago, peas, beans, turnips, carrots, parsnips, asparagus, artichokes, squashes, beets, corn, rice, hominy, the stalks and white parts of cabbage, lettuce, broccoli, figs, grapes, prunes, apples, pears, bananas, jams, syrups, sweet pickles, chocolate, cocoa, liquors, and especially sweet wines) are either to be altogether prohibited or restricted to definite quantities, as will be pointed out below. Among articles to be forbidden are also the livers of animals, mollusks (oysters, etc.), and the inside meat of crabs and lobsters. The chief diet must be animal, since the non-nitrogenous substances are to a very limited extent, and in some instances not at all, assimilated. My own plan is to first note the effect of a rigid dietary as follows:

(*a*) *Animal food:* Fresh meats, poultry, game, bacon, ham, fish of all kinds, including crabs and lobsters (except the inside meat of the latter). Fatty substances in large quantities (℥viij—256.0—daily), with a view to restricting nitrogenous destruction, are highly commended by Klemperer, and the free use of butter is also urged, eggs, cream-cheese, curds, and buttermilk being allowed.

(*b*) *Vegetables:* Saur-kraut, lettuce, sorrel, mushrooms, water-cresses, spinach, chicory, celery, cucumbers, mustard-cress, and pickles of various sorts (except sweet).

(*c*) *Bread:* The crust of a French roll, first recommended by Flint. Ebstein has recently very highly recommended aleuronat bread; it contains a large proportion of vegetable albumins. The so-called No. 1 gluten biscuit[1] is the only form of gluten bread made in this country that does not contain nearly as much starch as the white flours (Tyson).

(*d*) *Fruits:* Lemons, oranges, and nuts (except chestnuts).

(*e*) *Beverages:* Milk enough for cooking purposes; tea and coffee, sweetened with glycerin or saccharin; alkaline mineral waters (Saratoga-Vichy, Seltzer-water), simple water with some brandy, and acidulated drinks; Bass's ale, in which all the sugar is converted into carbonic acid and alcohol, and certain acid wines (claret, Rhine).

This strict diet usually causes the sugar to diminish greatly in amount, and in many cases to disappear entirely. If the patient keeps well-nourished and strong, carbohydrates should not be added, since there is no toleration for the latter. On the other hand, the weight falls if there be toleration for carbohydrates. Nothing is gained by relieving the glycosuria and polyuria if it be accomplished at the expense of the general strength of the patient. Von Noorden claims that a non-carbohydrate diet improves tissue-metabolism, thus increasing the system's power of warehousing carbohydrates, and recommends a rigid albuminoid diet at intervals of a few months. The abuse of albuminoids may increase the sugar in the urine (Naunyn), and in two cases under my observation this ended in diabetic coma. S. Solis Cohen, after repeated trials, recommends levulose as a form of sugar that can be assimilated without augmenting the excretion of glucose. With lean patients he uses ℥j (32.0) per day; with stout persons, only enough to act as a sweetening agent. Lactose has been found to give similar results. Hale also strongly urges levulose, after considerable personal experience. As a substitute for the latter agents a small amount of ordinary bread (which contains 55 per cent. of starch) or potatoes, as the patient prefers, may be allowed. The effects upon the general condition of the patient (body-weight), as well as upon the glycosuria (ascertained by a daily quantitative estimation of the sugar in the urine), are to be carefully noted, and the proportion of carbohydrates may be increased gradually until the limit of the system's ability to assimilate them is found. A more generous dietary is allowable only after the sugar has been absent from the urine for a couple of months and then it is to be adopted in a gradual manner.. A skimmed-milk diet has been recommended by Donkin, Tyson, and others.

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[1] This is made by the Battle Creek Sanitarium Co., of Battle Creek, Mich.

2. Next to an appropriate diet stand certain directions as to **proper hygienic living**: (*a*) All forms of mental excitement and worry must be avoided; (*b*) moderate and regular physical exercise aids metabolism, and is thus directly useful; massage may be substituted for active exercise when the latter is prohibited on account of weakness; (*c*) the diabetic requires a temperate and equable climate; (*d*) a daily tepid bath if the patient be feeble, and a cold bath if he be strong, are to be commended; (*e*) flannels should be worn next to the skin all the year round; (*f*) the living and sleeping apartments must be thoroughly ventilated; (*g*) the teeth must receive careful attention in order to prevent caries.

3. The **medicinal measures** deserve only third place in the treatment of diabetes, and of these *opium* is still the chief. It is not necessary to employ it in all cases, but it may be tried if the dietetic and hygienic treatment before recommended cannot be carried forward or fails to effect a cure. Opium seems not only to exert an influence over the polyuria and the excretion of sugar, but it almost invariably lessens the intense thirst and conduces to refreshing sleep. The drug is well tolerated by diabetics. The commencing dose may be gr. j (0.0648) three times daily, and later increased to gr. v (0.324) or even to gr. x (0.648) three times daily. If morphin be employed, we may begin with gr. ¼ (0.0162) and increase the dose to gr. j (0.0648) or more three times daily. Pavy warmly advocates the use of codein (gr. ½–iij—0.0324–0.1944, three times a day). My own best results have been obtained from the use of the latter remedy in the form of the sulphate, in ascending doses, commencing with gr. ¼ (0.0162) three times a day, and augmenting the dose by gr. ¼ (0.0162) every second day until gr. ij—0.129 (rarely more) are taken thrice daily. Codein possesses the advantage of being less constipating and less likely to disturb the digestive function than either opium or morphin. In patients of a full habit the alkaline waters exercise a valuable influence; Bethesda, Carlsbad, and Vichy of France have long had a reputation. For the foreign water our native alkaline waters may be substituted, especially the Saratoga-Vichy. While these are valuable adjuncts, they are, however, without the curative and specific effect that is claimed for them by certain authorities.

Among other therapeutic agents that have been employed are the following: the solution of the bromid of arsenic, ♏iij–v (0.199–0.333) three times a day, after meals—in some cases a useful adjuvant to the treatment above outlined; potassium bromid, gr. xx (1.296), three times a day, approximating in efficacy the latter remedy; guaiacol, ♏v–x (0.333–0.666), three times a day in a tablespoonful of milk or cod-liver oil, has given excellent results (Clemens); antipyrin (gr. x—0.648, three times a day); sodium salicylate, gr. xv (0.972), three times daily, lessen the formation of sugar; and strychnin, gr. $\frac{1}{30}$ (0.0021), three time daily, is an almost invariably useful remedy. Of the numerous remedies in whose favor convincing evidence is wanting, but which ar mployed by different clinicians, the following may merely be enumerated: Fowler's solution, potassium iodid, iodoform, lactic acid, glycerin, nitroglycerin, creasote, quinin, jambul, lithium, and methylen-blue.

The treatment of diabetes by *fresh pancreas* or by dry or glycerin extracts has been generally unsuccessful. Fitz, however, mentions a case in which remarkable improvement followed the exhibition of raw calf-pancreas. These preparations have been employed to supply the ferment (internal secretion) essential to the assimilation of sugar. R. Lépine has obtained from the fresh pancreas, from saliva, and from the diastase of malt a glycolitic ferment by a method which, he tells us, still requires to be perfected. This agent he has used in 4 cases of diabetes with a fair degree of success. Williams tried grafting sheep's pancreas in diabetics in two cases, but the results were unsatisfactory. Gilbert and Carnot found that *extract of liver*, administered by rectal injection, caused a marked decrease in the excretion of sugar.

Thyroid extract, in small doses, was followed by immediate improvement in two of my cases; it is indicated in alimentary glycosuria.

4. **Symptomatic Treatment.**—Most symptoms demanding therapeutic interference the competent physician is prepared to meet by following general rules. The management of diabetic coma, however, will be briefly discussed. Believing it to be due to an acid-intoxication, Klemperer gave large amounts of alkalies in 9 cases by intravenous injection, without preventing a fatal issue. To counteract the acids of the organism, sodium bicarbonate (drams 5, daily) is advisable (Robin).

The normal saline infusion, used by hypodermoclysis, has given favorable results in some cases.

Klemperer urges the use of fatty substances in large quantities as the best means of restricting nitrogenous destruction, and thus preventing the condition to which diabetics so frequently succumb. When a disgust develops for fats a substitution-method of treatment consists in administering alcohol (℥iss—48.0—per day). Like carbohydrates, alcohol in *small* quantity checks waste and may lead to an accumulation of flesh (Hirschfeld). Strychnin, digitalis, or ether may be tried hypodermically during the attack. Prolonged tepid baths with occasional douching have seemed to produce beneficial results in some cases, and are worthy of a trial in all. Elimination from the bowels is to be increased, and oxygen should be inhaled.

DIABETES INSIPIDUS.

Definition.—A chronic nervous affection, characterized by constant thirst and an excessive flow of urine, which, however, is free from sugar and albumin and is of low specific gravity.

Pathology.—No definite or characteristic lesions have been noted, though some degree of enlargement of the kidneys, together with sacculation, due to pressure backward upon the renal structure by the enormous quantities of urine in the bladder and ureters, has been observed. The ureters and pelves of the kidneys may be dilated, and the bladder, owing to constant over-distention, may be hypertrophied. The nervous lesions are diversified, but are not peculiar to simple polyuria. Most important, perhaps, are the tuberculous and other tumors about the floor of the fourth ventricle.

Etiology.—(*a*) Diabetes insipidus is often induced by *nervous influences*—shock, fright, etc.—and may also be of traumatic origin. In the majority of the latter cases it follows injuries to the head, but also, more rarely, it may be traced back to injuries of other parts of the body. Tuberculous and other lesions in the vicinity of the floor of the fourth ventricle may produce polyuria. It has also been caused by paralysis of the sixth nerve, with or without meningitis. (*b*) It may occur during convalescence from *acute infectious diseases*. I have seen 2 instances after influenza in young subjects. (*c*) *Intemperance*, especially the consumption of inordinate quantities of malt liquors, proves a cause. In several of my own cases the amount of urine passed was out of all proportion to the quantity of fluid ingested. One of these patients consumed three pints of beer daily, while the urine excreted amounted to eight quarts. (*d*) *Heredity.*—Weil found in four generations of a certain family consisting of 91 members, that 23 exhibited continuous polyuria —all, however, remaining in good health. (*e*) *Age.*—The disease is relatively more frequent in childhood and early adolescence than is diabetes mellitus. Of 70 cases collected, 22 were under ten years of age, and 13 between ten and twenty (Roberts). Diabetes insipidus may be *congenital.* (*f*) The great proportion of cases occur in males as compared with females.

Nature of the Affection.—The specific cause of the disease, if it have one, is as yet undiscovered. We are totally ignorant of its true nature, though the facts discovered by Bernard, that either a puncture at a certain spot in the floor of the fourth ventricle or section of the vagus causes polyuria, go to show that it is of nervous origin. It is true that the disease may come on in persons apparently in robust health without discernible causative agencies. In many instances, such as organic affections of the brain or abdominal tumors, the condition is purely symptomatic, and these are probably not to be classed as cases of genuine diabetes insipidus, which is a vaso-motor neurosis, usually of central, though sometimes of reflex, origin.

Clinical Symptoms.—The *onset* is *gradual*, as a rule, but when it follows a fright or traumatism it may develop quickly. There are two main symptoms—the passage of an enormous quantity of limpid urine, and the constant thirst. The daily *amount* of urine varies from 20 to 60 pints (10–30 liters); it is transparent, and the specific gravity is low (1001 to 1005). While the percentage of solids is lessened, the total is usually about normal, and may even be increased. *Albumin* and *sugar* are rare, but in a few cases inosite has been detected. The act of micturition is of very frequent occurrence, and the quantity of urine passed at each sitting surprisingly large. The persistent *thirst* necessitates frequent drinking, but the voracious appetite seen in diabetes mellitus does not mark this disease, in which the appetite is only slightly increased. As a result of the polyuria the skin and mucous membranes are abnormally dry, as in genuine diabetes. But, unlike the latter affection, a fair degree of bodily nutrition is maintained as a rule.

The saliva and other digestive secretions are scanty, and this, together with the good appetite, is a fact which explains the disturbances of digestion sometimes met with. The tolerance of the system to alcohol is often phenomenal. Associated nervous phenomena are frequently observed, such as neurasthenic symptoms, insomnia, and chorea.

Prognosis.—The majority of instances proceed to recovery sooner or later, while others pursue an almost endless course—forty or even fifty years in duration—and the patient meanwhile retains his general good health. There is a small group of grave cases that are due to organic diseases either of the brain or abdominal organs (tuberculosis). Death may also be occasioned by some intercurrent affection.

Diagnosis.—The clinical recognition of diabetes insipidus rests upon—(*a*) the enormous amount of urine passed; (*b*) its low specific gravity; and (*c*) the absence of sugar and albumin.

Differential Diagnosis.—Among affections that must be differentiated are *diabetes mellitus*, which has a single point of resemblance—namely, the polyuria; *hysteric polyuria*, which is transient and accompanied by other hysterical manifestations; and *chronic interstitial nephritis*, which generally distinguishes itself by the presence of albumin and hyaline casts in the urine, arterio-sclerosis, and cardiac hypertrophy.

Treatment.—The amount of drinking-water is to be moderated in a gradual, cautious manner, and the patient should be warned not to exceed his actual necessities. I find also that methodic physical exercise and other hygienic means act beneficially. Galvanism has its advocates; it is most useful when cord lesions exist.

Of medicines, nervines, especially valerian and its preparations, are useful in the idiopathic variety of the complaint, and may be given in the form of the ammoniated elixir (ʒj–ij—4.0–8.0) three or four times daily. The valerianate of zinc, quinin, and iron may be variously combined, according to the indications presented by special cases. Ergot and gallic acid have long enjoyed a high reputation in this disease. The commencing doses should be moderate, and then be increased until full physiological doses are employed, this method often bringing about admirable results. Antipyrin, acetanilid, the bromids, and arsenic have been extensively employed and lauded by different writers in the treatment of this affection. My own best results have been attained by the use of ergot. The potassium iodide is much vaunted. Next to this agent the bromids and acetanilid, given alternately at intervals of a couple of weeks, have been found to be most useful. If a primary disease exists, it must be met on intelligent general therapeutic principles.

ARTHRITIS DEFORMANS.

(*Rheumatoid Arthritis; Rheumatic Gout.*)

Definition.—A chronic disease, characterized by progressive changes in the arthritic structures (cartilages, synovial membranes, etc.) and by osseous periarticular formations, producing great deformity. The affection may rarely be acute in its course.

Pathology.—Among early gross changes there may be an effusion into the affected joints, but this disappears later. The cartilages are absorbed, the process beginning centrally, where there are both the maximum amount of friction between the opposed cartilaginous surfaces and the minimum blood-supply. Disappearance of the cartilages is naturally followed by contact of the ends of the bones, the latter becoming polished and resembling ivory as the result (eburnated). The friction between the bony extremities may lead to absorption of the latter.

At the periphery, where pressure is slight or even absent, the cartilages become greatly thickened in consequence of persistent irritation, and later become ossified, forming osteophytes which overlie the articular surfaces. These may lock the joints. Bony nodules may also be formed from the periosteum of the bony shafts.

Almost simultaneously the *synovial membranes* become inflamed, a proliferation of their cells taking place, and this exudate may undergo organization and rarely ossification. Later the *capsule* and the *ligaments* are thickened, causing a restriction of movement of the affected joints and producing pseudo-ankylosis. Less frequently they soften and weaken to such an extent that often partial, and sometimes complete, dislocation of the joints ensues; but displacement of the ends of the bones, amounting even to complete luxation, may also be due to absorption. This is often observed in the head of the femur, producing the so-called *morbus coxæ senilis*. Muscular wasting occurs and may be profound. Neuritis has been noted.

The *histo-pathologic* changes consist in cell-proliferation, with fibrillation and softening of the matrix of the cartilages, followed by absorption due to pressure. At the margin, however, proliferation of the cells leads to massive nodulation.

Etiology.—The *nature* of the disease is still dubious, though the old view, that it is *closely* connected with rheumatism on the one hand or gout on the other, should be abandoned. J. K. Mitchell long since maintained that rheumatoid arthritis is of neurotrophic *origin*, being especially dependent upon affections of the spinal cord, and without stopping to adduce all of the facts that tend to support this theory, the following deserve mention: (1) Diseases of the cord (locomotor ataxia, etc.) are known to cause arthritic conditions; (2) The character of certain causal factors, such as nervous shocks, griefs, etc.; (3) The symmetry of the joint-deformities; (4) The time of occurrence; and (5) Noticeable trophic disturbances that are frequently associated. Falli[1] autopsied 4 cases, 2 of which were typical, and in the latter lesions were found in the anterior horns of the spinal cord, atrophic in the first case, but degenerative as well as atrophic in the second. According to Falli, not all cases of arthritis deformans are to be interpreted as instances of nervous disease. Another theory of the disease is the *microbic*, which has received some measure of support.

Bacteriology.—Dor claims to have succeeded in finding a definite organism. He also claims to have reproduced the disease by injecting cultures directly into the blood of rabbits, and considers the germ an "attenuated culture" of the *staphylococcus pyogenes aureus*. v. Dun-

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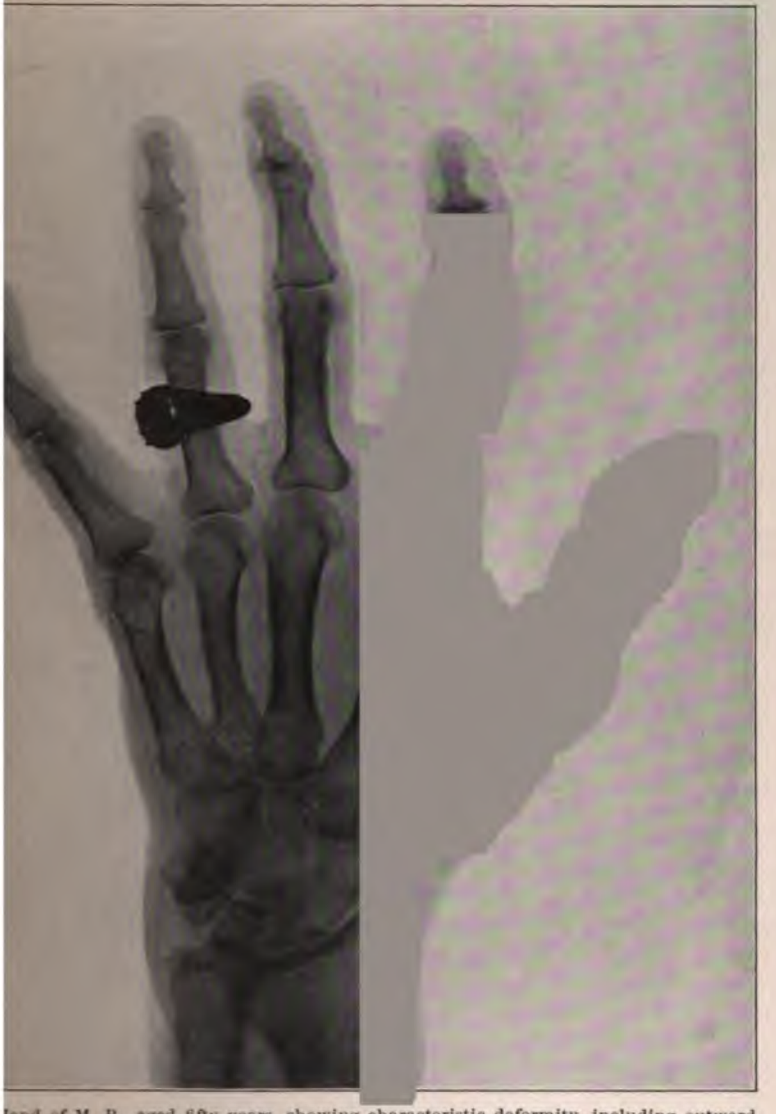
[1] *Il Policlinico*, Dec., 1894.



Hand of M R., aged fifty years, showing characteristic deformity, including outward deflection of fingers, in advanced arthritis deformans.

gern and Schneider isolated after death from the mucus of the gall-bladder, and also from the exudate in the joints, small diplococci that did not resemble the organisms previously described by Blaxall and Schüller. Injections of the cultures into the knee-joint of rabbits resulted in the production of lesions similar to those observed in the patient.

Predisposing Causes.—(*a*) *Nervous shocks*, mental worry, and deep grief. (*b*) *Females* are more frequently victims than are males, the proportion, according to the statistics of Garrod, being about one to five in favor of the former sex. To account in part for its greater frequency in women is the fact that sterility and certain ovarian and uterine complaints seem to exert a strong etiologic influence. (*c*) *Age* exerts a decided influence. It is most frequently contracted in the third decade of life, though it has been noted as late as the end of the fifth. It occurs also in children, though rarely. Out of 307 cases treated in the Devonshire Hospital during 1892, only 2 per cent. manifested the disease before the age of ten. (*d*) *Heredity* has been traced in some instances, and in many a *family tendency* to joint-affectien. (*e*) Though it occurs in all classes of society, the *poor* or those exposed especially to debilitating influences are more liable than the *rich*. (*f*) Ewart[1] recognizes some mixed conditions in which rheumatoid arthritis may supervene on the gouty diathesis.

(1) Symptoms of the Chronic Form.—At first one joint, usually of the hand, is slowly involved; soon the corresponding joint on the opposite side is attacked. These may recover apparently, but are soon reinvaded and grow progressively worse. The affected joints slowly enlarge, and are moderately painful, particularly on movement. *Pain*, however, may either be slight or even absent, or severe (rarely agonizing) in character. There is neither redness nor tenderness, as a rule, but on palpation an effusion, variable in extent, is generally detectable. The *course* during the early stage is often marked by periods of improvement, alternating with exacerbations in the local symptoms, and especially in the swelling and pain. While, as intimated, one or two joints only are affected at the start, gradually those of the feet, arms, legs, and trunk are invaded symmetrically, until, in the worst cases, every joint is deformed.

The most characteristic symptom is the *deformity*, which manifests itself earliest in the hands. The fingers are generally pointed toward the ulna, rarely toward the radius, and the presence of the osteophytes and the immensely thickened capsular ligaments, together with the retracted muscles, all tend to alter entirely the shape of the joints. The fingers, for example, are flexed and extended upon the hand, and sometimes overlie one another. With the progress of the deformity a partial, and less often a complete, luxation of the joints occurs (see Fig. 29). The joints may become finally either quite fixed, owing to the presence of the periarticular osteophytes, or a limited degree of movement may remain.

Palpation and *auscultation* of the involved joints reveal crepitation during movement. Strangely enough, the thumb remains intact, compensating for the loss of the functional movement of the fingers to a remarkable extent. In addition, the hand is sometimes less affected than the rest of the joints—a fact which enables the patient to perform a great variety of delicate movements, or even to engage in useful and surprisingly skilful handicraft. The adjacent muscles become wasted and are the seat of contractures, causing flexion of the limbs, especially of the thigh upon the abdomen and the leg upon the thigh. Other trophic changes, such as paresthesia and pigmentation or glossy areas of the skin, may be observed. In 3 of my cases onychia was present. In extreme instances the decubitus is lateral and the patient utterly helpless.

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[1] *International Medical Magazine*, April, 1899.

The *course* of the disease throughout the more advanced stages is exceedingly variable. Its advance may be arrested and the general health remain unimpaired, and this may take place after implication of but a few joints, so that the entire affection may be confined to a comparatively small part of the body, either in the upper or lower extremities. In progressive cases more or less gastro-intestinal disorder arises; the symptoms of indigestion appear, the appetite is impaired, and anemia develops. The patient's sufferings make him irritable. Hypochondriasis may be a concomitant. In established cases the pulse is persistently rapid and the skin inclined to free perspiration.

Clinical Varieties.—(1) Of the **chronic form** there are certain subvarieties. The disease may be limited to a single joint (*monarticular*), this form most commonly affecting the hip-joint, when it is known as *morbus coxæ senilis*. It is seen generally in old men, and often follows an injury. Its features—pathologic and clinical—including the muscular wasting, are the same in kind as those of the *polyarticular* variety. Monarticular arthritis deformans may also be confined to the shoulder-joint or the knee, and, as in the preceding form, men who have passed the middle period of life are mainly affected.

A special variety, which is generally not monarticular, involves only the vertebræ (*spondylitis deformans*). This may be combined with *morbus coxæ senilis*, or the condition may be confined to the cervical spine, as in a recent case of my own, thus preventing flexion of the head. A fair degree of rotation usually remains, but it sometimes happens that the entire spinal column is involved and held in a perfectly rigid position.

Still another form in which the distal joints of the fingers become knobbed (*Heberden's nodes*) demands separate description. Heberden's nodosities occur chiefly in women between the thirtieth and fortieth years, though I have seen one case which began after the fiftieth year. According to Heberden, who first described them, the nodes have no intimate association with gout, and this view coincides with my observations. At first the affected joints become *swollen*, *tender*, *slightly red*, and *painful*, and then seemingly undergo great improvement. The condition however, is progressive, advancement occurring in the form of fresh exacerbations, which are only rarely traceable to errors in diet, and are separated by periods of remission. The morbid process is the same as in rheumatoid arthritis, and the destructive changes in the joints proceed until distinct hard nodules are formed. These are usually most marked at the sides of the extensor surfaces of the second phalanges. The disease does not spread to any of the larger joints, and, although incurable, it is free from danger to life.

(2) **The Acute Form.**—This is comparatively rare, and occurs com-

monly between the ages of twenty and thirty. It occurs in children, and is more common in women than in men. Among its common antecedents in women are pregnancy, delivery, excessive lactation, and the menopause. *Multiple arthritis*, affecting both the large and small joints, sets in acutely, and there are pain and either a slight redness or a considerable swelling, due chiefly to an effusion which is intra- rather than periarticular. There are only a slight tendency to migration from joint to joint, and a slight febrile disturbance.

Still described a form of chronic joint-disease in children which he thinks presents differences sufficiently marked to suggest a distinct clinical and pathologic entity, and differing from the rheumatoid arthritis of adults. It is defined as a progressive enlargement of the joints associated with general enlargement of the glands and enlargement of the spleen. He has studied 22 cases, 19 of which came under his personal observation. It occurs before the second dentition. Stiffness, general thickening of the tissues around the joints without redness or tenderness, except in very acute cases, with limitation of movement and more or less rigid flexion of the joints, characterize the arthritic disturbance. The most distinct feature of the disease is the enlargement of the lymphatic glands, those in relation to the involved joint being primarily affected. The glandular swelling is general and constant. Enlargement of the spleen is also a striking feature. Cardiac complications are absent. The *course* of the disease is slow.

Differential Diagnosis.—The diagnosis between the chronic form of the disease and *chronic rheumatism* is often extremely difficult. In the latter, however, a few of the larger joints only are involved, while there is an absence of the peculiar deformity and marked fixity of the articulations. On the other hand, cardiac complications are absent in chronic rheumatoid arthritis, and the course is progressive. A *monarticular arthritis* which differs in its morbid process from rheumatoid arthritis sometimes affects the shoulder-joint. It is not uncommon, and is "characterized by pain, thickening of the capsule and of the ligaments, wasting of the shoulder-girdle muscles, and sometimes by neuritis" (Osler). I have met with 5 instances of this sort, in all of which pain was intense and the course subacute. All ended in recovery.

The acute form is frequently confounded with *acute articular rheumatism*, from which it is to be discriminated by the special etiologic factors, the less severe pain, the less marked redness, the slight tendency to migration from joint to joint, the slighter febrile disturbance, and by the practical freedom from cardiac complications. *Gout* will be distinguished in the description of that disease.

Prognosis.—Though incurable, rheumatoid arthritis is not immediately dangerous to life, and in a certain proportion of the cases improvement, and in a smaller proportion arrested progress of the disease, may be expected.

Treatment.—This especially involves measures that are directed toward the improvement of bodily nutrition—a generous dietary, systematic warm bathing, and an abundance of fresh air, with properly-regulated physical exercise. Tonics may be necessary to invigorate the economy, and iron to overcome the anemia. The prolonged use of cod-liver oil in conjunction with other remedies has given me excellent results. Of special agents, the most satisfactory in their effects if administered early are iodin and arsenic. An eligible form of the latter is arsenious acid, given in granules (gr. $\frac{1}{36}$—0.0018, after food); the former may be administered in the form of a saturated solution of sodium iodid, of which ten to fifteen drops may be given in milk one hour after food. The patient may be sent to a warm climate in winter and to a cooler one, preferably a mountain-resort, in summer. These patients also do well at certain mineral springs, such as the sulphur springs of Virginia, the hot springs of Arkansas or Töplitz, at Baden in Switzerland, and the warm sodium chlorid baths in Wiesbaden. Hot mineral spas should only be resorted to in the early period of the affection. Strümpell has seen excellent results follow the employment of hot sand-baths, which can be used at home. Stewart advocates the Tallerman method of treatment—*i. e.* of superheated dry air. Short employs the apparatus ordinarily used in hospitals for the administration of hot-air baths, with a view to producing diaphoresis. Fibrous anchylosis may be treated surgically.

Eliminative Treatment.—Guaiacol carbonate may be given in doses of from 5 to 15 grains (0.3–1.0 gm.) daily and rapidly increased. This acts by combining with the bacterial toxins, to be eliminated as guaiacol sulphate (Bannatyne).

The local means are of the highest value. If the joints be inflamed, cold compresses, covered with oiled silk, to which some narcotic agent may be added, will afford relief. This should be followed by thorough and systematic massage, which is our best measure for the reduction of the swelling (by promoting absorption of the inflammatory exudate) and for lessening joint-rigidity. It also restores the atrophied muscles and assists the general health. Swedish movements are useful in maintaining mobility and often in restoring that which has been lost.

GOUT.

(*Podagra.*)

Definition.—A form of perverted nutrition due to an auto-infection, accompanied by the formation of a variable (usually increased) amount of uric acid, and characterized clinically by attacks of acute arthritis, with or without uratic deposits in and around the joints.

Nature of the Affection.—The numerous theories that prevail at present in regard to the disease are irreconcilable, but it seems certain that there is (*a*) an excessive absorption of nutritive substances, both solid and liquid: (*b*) a disordered metabolism growing out of the effects of imperfect physical development, combined with too little muscular exercise; (*c*) a defective elimination of waste-products, although in some cases a normal elimination of waste-products exists.

There are a number of *uric-acid theories*, some of which may be briefly mentioned: 1. Garrod contends that an acute attack of gout is invariably produced by an excess of uric acid in the blood, due to increased formation and greatly decreased elimination; also, that inflammation is aused by the deposition in the joints of sodium urate. 2. Haig holds hat there is a diminished alkalinity of the blood, and that the latter annot therefore hold the uric acid in solution, and not an excessive production of uric acid. 3. Ebstein thinks it probable that there exist an excessive production and accumulation in the blood of uric acid. The surcharged blood excites local inflammation, followed by necrosis, and uric acid deposits. 4. Sir William Roberts believes that acute attacks of gout are dependent upon the precipitation of the crystalline biurate of sodium; that the urate is transformed into the less soluble biurate in the blood. 5. V. Noorden concludes that the essential process is a tissue-necrosis attributable to the presence of a hypothetic ferment, and that the uric acid, which is without etiologic effect, is deposited at the necrotic focus. 6. Klemperer[1] has shown as the result of observations made in cases of gout, that as long as the function of the kidneys is not materially interfered with the presence of considerable amounts of uric acid in the blood must be attributed to increased formation. But the presence of an equivalent of uric acid in the blood in certain affections other than gout (*e. g.* leukemia) shows that this factor is not the sole cause of gout. 7. Morhorst states that in any alkaline liquid the basic substances combine with uric acid, if this be present, to form a urate. These uratic precipitations are met in non-vascular tissues only, the alkalinity of which is less than that of the blood, and that they are the essential cause of the symptoms. 8. Kolisch maintains that when the kidneys are healthy the alloxuric bodies are, in great part, excreted as uric acid; but when they are diseased the xanthin bases are increased at the expense of the uric acid. Chittenden and others, however, hold that the xanthin bases are practically free from toxic effects. 9. Luff thinks that uric acid is formed in the *kidneys* from a combination of urea and glycocin, an increasd amount of the latter substance being formed in the liver.

Sir Dyce Duckworth[2] ascribes *gouty* urichemia to an inherent morbid tissue metabolism and a *neurotrophic* disturbance.

The recent view that failure of the renal function precedes the development of gouty manifestations, although not established, and the older view, that an increased proportion of uric acid is found in the blood in gouty subjects, seem to be the most widely accepted.

Pathology.—The post-mortem history of gout is concerned principally with the arthritic changes, including the deposits of the urate of sodium in the cartilages, the ligaments, and the synovial membranes. These are fluid in their earliest state and contain numerous small crystalline masses; they soon inspissate and later become hard and dry (tophi). The latter excite secondary inflammatory changes that may lead to fibrous overgrowths, distortion, and fixation of the joints. Gouty tophi may be absorbed or they may finally be discharged through the skin in consequence of an ulcerative process. The chalky concretions have been found also in the cartilages of the ears, less frequently of the nose, eyelids, and larynx. They have also been described in the periosteum and along the tendons of the palms of the hands, where they produce a characteristic form of contraction of one or more fingers (Dupuytren's contraction). Charcot has found them in the penis. If death occur in the *acute* attack, hyperemia and swelling of the capsule, ligaments, and synovial membrane are found, together with an inflammatory exudation into the joint.

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[1] *Deutsche medicinishe Wochenschrift*, 1895, No. 40, p. 653.

[2] *Proc. Thirteenth Internat. Med. Congress*, Paris, Aug. 2–9, 1900.

The *kidneys* are usually involved, the changes being similar in character to those observed in the joints, and innumerable areas of necrosis, followed by uratic deposits, are seen throughout the organs, though chiefly in the papillæ. Osler says that "the presence of these uratic concretions at the apices of the pyramids is not a positive indication of gout." N. S. Davis, Jr., points out that the kidneys are affected in spots, with intermissions in the degenerative changes, which are microscopical in size, until finally large areas are involved. Granular contracted kidney (chronic interstitial nephritis), with or without arteriosclerosis, is sometimes caused by the gouty condition (*vide* Interstitial Nephritis).

The *heart* and *blood-vessels* always present changes. Gout induces arterio-sclerosis, and the latter in turn causes cardiac hypertrophy, particularly of the left ventricle. In chronic cases fatty degeneration of the heart-muscle sometimes occurs, and chronic valvulitis, with deposits of urate of sodium in the valves, has been noted. Chronic bronchitis, asthma, and emphysema are among the more common changes connected with the *respiratory tract*, acute conditions being rare.

Etiology.—The following are the principal contributing causes:

(*a*) *Heredity.*—Garrod's dictum, "that more than one-half of all gouty subjects can distinctly trace their ailment to an hereditary taint," is doubtless correct, heredity from the grandparents, which is not of infrequent occurrence, being included in this estimate. If the better class of society alone be considered, the percentage will probably be still larger. It must not be forgotten, however, that patients out of pride represent other articular affections as gout. (*b*) *Age.*—Primary attacks are most frequent in middle life. They are rare before puberty, though exceptionally seen even in suckling infants; but after the age of puberty they become more frequent. After the fiftieth year they decrease rapidly in frequency, and are very rare in quite advanced life. The cases that develop quite early in life often show a striking hereditary taint. (*c*) *Sex.*—The arthritic form is less frequent in women than in men, while the former are disposed to the irregular type of chronic gout quite as strongly as the latter. (*d*) *Diet.*—Over-indulgence in the pleasures of the table, together with defective muscular exercise, constitutes a potent factor, and this even in persons who are endowed with exceptional powers of digestion. (*e*) *Alcohol*, and particularly the fermented liquors, are among the chief favoring influences. The fact explains the relatively greater frequency of gout in certain countries (*e. g.* England and Germany), in which the heavier beers and ales are freely used, than in America, where lighter fermented drinks are more popular. The cases, however, are on the increase in this country. (*f*) *Social State.*—Most cases occur among the upper class of society, but there is also a well-defined form of "poor-man's gout" due to an excessive use of malt beverages. (*g*) *Lead.*—Workers in lead furnish numerous typical examples of gout. Garrod found that in 30 per cent. of the hospital cases the patients had been painters or workers in lead. He also showed that the administration of lead salts to gouty persons almost invariably determined a gouty paroxysm. Whether lead produces gout by arresting the excretory processes, and by thus inducing a fibroid change in the kidney and liver, as is held by Oliver of New Castle, is not definitely settled. Poore points out that gout produced by lead or chronic kidney trouble is constantly associated with anemia and emaciation, and forms a distinct clinical entity. We may presume the existence of a primary renal gout. (*h*) Cornillon and others detail cases in which injuries were followed by the first appearance of the disease.

Clinical History.—1. **Acute Gout.**—The earliest manifestations of the disease are apt to take the form of a more or less typical attack of *acute arthritic gout*. The latter is usually preceded by certain *prodromal symptoms*, which vary in different cases, but are almost constantly similar for the paroxysms of individual cases. The patient may complain either of slight muscular cramps and articular pains, or of dyspeptic disorder, or of an asthmatic seizure; or he may exhibit mental disturbance —irritability of disposition, broken, restless sleep, and depression of spirits. In a small percentage of instances, just prior to the attack the patient feels better than ordinarily. It has been observed that immediately before and also during the early part of a paroxysm the daily amount of uric and phosphoric acids found in the urine is diminished; but Klemperer has shown that no relation exists between the amount of uric acid present in the urine and the character of the disease.

The attack generally develops in the very early morning hours. The patient awakens suffering from pains in the metatarso-phalangeal joint of the great toe, that soon become excruciating, while the joint feels as if it were tightly compressed in a vise. The *local signs* of inflammation—heat, redness, swelling, and excessive sensitiveness—quickly supervene. The skin pits on pressure and becomes shiny. The *body-temperature* rises to 102° or 103° F. (39.4° C.), and the patient manifests intense irritability.

At the end of an hour or two the sufferings abate, the fever often declines, with free perspiration, and the patient may be able to pursue his avocation. During the next day some degree of enlargement and inflammatory edema remains, and on the following night the symptoms are usually repeated in all their violence. The condition usually progresses in this manner from four to seven or eight days, though after a few days the intensity of the paroxysms is apt to lessen. After the attack the swelling subsides and there is a slight desquamation of the skin, which resumes its normal color, and the general health is often unusually good. These so-called fits of gout usually recur from time to time, the duration of the intervals depending largely upon the patient's habits or routine of life. On the whole, the first interval is apt to be the longest, while later the intermissions may not exceed two or three months. With subsequent attacks the affection is apt to spread to other articulations. There is no tendency to suppuration.

2. **Retrocedent Gout.**—This term implies the sudden transmission of the arthritic process to some internal organ. During a paroxysm the joint-inflammation may quickly disappear with an equally sudden development of intense pain in the region of the stomach, vomiting, diarrhea, faintness, and a rapid, feeble pulse. Suppressive gout may attack the heart and produce precordial pain, dyspnea, cardiac palpitation, and much anxiety of mind. It may also excite pericarditis with a fatal result. Transmission to the head, with the development of intense

cerebral symptoms (maniacal excitement, coma, and apoplexy), also occurs. Nervous phenomena, however, are more commonly due to uremic poison.

3. **Symptoms of Chronic Gout.**—Chronic gout follows the acute variety. The transition is gradual, the intervals between attacks shorter, while the attacks themselves grow milder and longer. At last the local inflammation does not appear. The condition extends to other joints: first, to the corresponding joint on the opposite side, then to the other toes and the ankles. Later, the fingers and wrists may be invaded, but almost never the largest joints (hip, shoulder). With the progress of the affection the chalk deposits slowly and gradually increase until the characteristic deformity is produced. The skin covering the tophi may ulcerate, exposing the chalk-stones, an unmistakable picture. When the fingers are affected we note a deflection at the second or third joint, constituting a peculiar habitus.

Among important *associated conditions* are chronic gastric catarrh, arterio-sclerosis, cardiac hypertrophy with considerable functional disturbance of the heart, and "contracted kidney," forming a much complicated yet easily recognized clinical picture. If in cases of this sort the *urine* of a gouty person is carefully examined, and is found to contain a small percentage of albumin and tube-casts, the whole train of events becomes easy of interpretation. The cases may be divided into two classes: (*a*) those in which the complexien is florid and the general health vigorous; (*b*) those with pale, sallow facies, emaciation, and enfeeblement. These groups are chiefly dependent upon the differences in the etiologic factors. Gouty subjects often manifest unusual mental vigor.

The *course* of chronic gout is liable to be interrupted by acute exacerbations with fever, during which dangerous complications may arise—*e. g.* uremia, pericarditis, pleurisy, pneumonia.

4. **Irregular Gout.**—Says Sir Dyce Duckworth: "Gout manifesting itself anywhere but in a joint is to be considered irregular or incomplete." Such cases are confined chiefly to persons of gouty heritage, though I feel confident that the diathesis may be also acquired. But though the etiologic factors that produce lithemia also in time produce gout, these two conditions should be discriminated; for, while in both we usually note an excess of uric acid in the blood, in lithemia there are no tophi present, and hence no necrotic foci in the joints or elsewhere. Irregular gout, then, rarely occurs in persons who have had previous typical attacks, but should any of the conditions described below as being dependent upon the gouty diathesis be associated, or should they alternate, with acute gout, they may be properly ascribed to the latter. On the other hand, when these conditions occur in persons who are free from hereditary taint, and who are not addicted to the intemperate use of alcoholic beverages, or excessive indulgence in the pleasures of the table, and are not possessed of luxury- and rest-loving temperament, the diagnosis of irregular gout is to be made with extreme caution. It is perfectly justifiable to apply a therapeutic test when other means of diagnosis fail.

The features of irregular gout are exceedingly diversified; the following are the more important:

(*a*) *Joint- and Muscle-pains.*—The muscular pains may be anywhere, and "flying" in nature, but the muscles of the back of the neck, the lumbar region, the abductors of the thigh, and the gastrocnemii are especially liable (Tyson). These pains are most severe in the early morning hours and subside as the day grows. Articular pains attended with some degree of swelling and deformity of the joints (the latter, however, not due to uratic deposits) may be of gouty origin; and, according to Paget and Garrod, Heberden's nodosities (previously described under Rheumatoid Arthritis) may present vesicular eminences due to gout.

(*b*) *Gastro-intestinal Disturbances.*—The symptoms referable to the intestines are identical with those presented by lithemia. In one of my cases intestinal colic followed by diarrhea put in an appearance at long intervals. Tonsillitis, pharyngitis, pericarditis, and even parotitis, may also be manifestations.

(*c*) *Cardio-vascular Symptoms.*—Just as in pure lithemia, so in atypical gout, the increased amount of uric acid usually present in the blood, by increasing the blood-tension, excites arterio-sclerosis and chronic interstitial nephritis—affections which are fully described in appropriate sections of this work.

(*d*) *Nervous Manifestations.*—The different varieties of headache, including migraine, are common. Sciatica and other forms of neuralgia, tingling, itching, burning sensations, and even pain in the palms of the hands and soles of the feet, are of frequent occurrence. Hot and itching eyeballs are, according to Hutchinson, among frequent manifestations; apoplexy may arise, secondary to atheroma induced by gout; and rarely meningitis (basilar) is among the gouty morbid states. The latter also include certain psychopathia—insomnia, irritability of temper, and melancholia. The possibility of gouty neuritis is to be remembered.

(*e*) *Urinary Symptoms.*—The urine is highly colored, of high specific gravity, often scanty, and the standing specimen deposits lithic acid. This is not peculiar to gout, however. In many cases uric acid is in excess only at intervals, giving rise to so-called uric-acid showers, while at other times it is diminished in quantity. Gouty persons are liable to gravel: I agree with Tyson, however, in thinking that the two conditions more frequently alternate than coexist. Intermittent glycosuria is also common in gouty subjects, and may lead to true diabetes mellitus; this glycosuria may alternate with uric-acid showers. With these affections—intermittent glycosuria and gout—obesity is not uncommonly associated. Oxaluria has been noted. Grandmaison believes the association of albuminuria with gout to be a frequent one, and that the early albuminuria is often intermittent. Among grave *secondary affections* chronic interstitial nephritis, with its characteristic features (slight albuminuria and later casts), very commonly develops, sooner or later, and cystitis (with gouty hemorrhage into the bladder), urethritis, prostatitis, and orchitis, all may be dependent upon gout.

(*f*) *Pulmonary Disturbances.*—Chronic bronchitis, to which asthma and emphysema are frequently secondary, is often the asthma of podagra.

(*g*) *Cutaneous Eruptions.*—Eczema is frequently associated with the gouty diathesis, and I have often observed eczematous eruptions in gouty subjects alternating with the symptoms of bronchitis or gastric catarrh.

(*h*) *Ocular Disorders.*—The chief eye-symptoms are conjunctivitis and keratitis (with tophi in the cornea and eyelids), iritis, hemorrhagic retinitis, and glaucoma. Gouty involvement of the ear (external canal and the auricle particularly) occurs oftenest late in life, though hereditary gout may rarely cause ear symptoms shortly after birth.

Differential Diagnosis.—The distinction between typical acute gout and *acute articular rheumatism* is a simple matter. But when, as is rarely the case, the former manifests itself as a polyarthritis, the discrimination is sometimes difficult. W. H. Thompson has pointed out that in gouty polyarthritis, when the knees, elbows, and phalangeal finger-joints are affected, the points of greatest tenderness on transverse pressure are over the condyles. On the other hand, in acute rheumatism the cutaneous tenderness is greater, while the points of maximum tenderness correspond with the tendons anterior and posterior to the joints. Moreover, gout distinguishes itself by its previous history (heredity, alcoholism, gluttony), by the tophi, which may be first detected in the ears or conjunctivæ, by the development of contracted kidneys, and the less marked fever. After repeated attacks deformities of the joints ensue. In a doubtful case the blood-serum may respond to the uric-acid test, as follows: Add 5–6 minims (0.399) of acetic acid to 2 drams (8.0) of blood-serum in a watch-glass; then place a linen thread in the solution and after twelve to twenty-four hours this will be incrusted with crystals of uric acid. The result is not, however, obtained exclusively from the blood of gouty subjects.

Chronic rheumatism is distinguished from gout by the fact that the latter disease involves chiefly the small, and chronic rheumatism chiefly the large, joints. Moreover, chronic interstitial nephritis and arteriosclerosis, with their varied and often serious consequences, are frequently attendant upon gout, but not upon chronic rheumatism.

To differentiate chronic gout and *rheumatoid arthritis* is sometimes a hard problem, but the following table will indicate the main points of difference:

GOUT.	ARTHRITIS DEFORMANS.
Frequently hereditary.	Not so.
Causes are chiefly dietetic.	Causes chiefly nervous.
Affects males and the better classes most frequently.	Affects females and lower classes most frequently.
Begins in the big toe and extends to other toes; it is unilateral.	Begins in the fingers, which point to the ulnar side; develops in symmetric order.
Attacks are periodic.	More steadily progressive.
Deformity due to tophaceous deposits.	Deformity due to exostosis and ankylosis, and more marked.
Uric acid usually in excess.	Not so.
Complications (nephritis, arterio-sclerosis).	Very rare.

Treatment.—(1) **Prophylaxis.**—In order to prevent the development of gout, especially in persons who have inherited or acquired a strong predisposition to the disease, temperate and even rigid habits of living should be adopted. Alcohol, particularly the heavier wines (Madeira, port, sherry, champagne, etc.) and heavier malt liquors, must be eschewed, and the patient must eat sparingly of concentrated meat (particularly red meat). A residence in the country with active out-of-door exercise is of paramount importance, but straining efforts, both mental and physical, are to be avoided. The climate should be temperate and moderately dry. The sleeping apartments should be capacious, well ventilated, and free from draught, and the action of the skin is to be favored by cleanliness, and if the patient be strong by a cold bath in the morning with friction. For the robust, Turkish baths at intervals of two or three weeks constitute an excellent measure. In the class of patients that are pale and debilitated warm baths on retiring are preferable, and the chilling of the skin-surface is to be carefully guarded against. The patient should wear flannels next to the skin in all seasons.

(2) **Active Treatment.**—(*a*) *Dietetic.*—"There is no diet for gout, but there is a diet for the patient" (H. C. Wood). The amount of food must be lessened as a rule, and taken at regular intervals. On the other hand, spare gouty subjects are met with, and in such I have found a rather generous diet, including fat-producing foods, of great service. Broadly speaking, the dietary should be constituted as follows: *succulent vegetables* (cabbage, salads, string-beans); *fruits* (except bananas, tomatoes, and strawberries); *farinacea*, as rice, hominy and the like (oatmeal to be avoided); *meats* should be restricted; beef and mutton may be allowed in corpulent subjects, but are otherwise to be interdicted, particularly in well-marked cases of gout; oysters and fish (except those that contain too much protein, salmon, smoked herring, canned sardines, mackerel, halibut, salt codfish, flounder), and fowl, particularly the white meat of chicken, are permissible; *fats* in the form of good butter may be taken freely—from 2½ to 3½ ounces (70.0–100.0) per diem, according to Ebstein; *milk* is entirely unobjectionable, and should be used in large quantities. If whole milk does not agree, it may be mixed with an equal part of Vichy. According to Kolisch, eggs are not objectionable, as the neucleins contained do not form alloxins. *Stale* breads may be used. I have observed that occasionally patients do best on albuminoids, while, on the other hand, with about equal frequency they improve on a vegetable diet; but I am convinced that a mixed diet, such as has just been indicated, is best adapted to the vast majority of the cases. Among articles to be avoided are pastry, tea and coffee, hot bread and cakes, sweet puddings, cheese, dried meats, and all highly seasoned dishes.

Beverages.—Alcohol is ordinarily to be interdicted. Rarely it becomes necessary to administer it, particularly in cases of suppressed gout, and when needed whiskey or gin (diluted) is to be preferred. Champagne, Tokay, Port, and malted liquors are particularly injurious in their effects, but clarets, Rhine, and Moselle wines can be generally taken without unfavorable results.

Mineral waters, particularly the alkaline, are highly advantageous, and sometimes are even curative. Their value, like that of the warm baths and systematic exercise, is dependent upon their power to increase renal elimination. Whether they promote solubility of the uric acid in the blood is questionable; moreover, according to the observations of Klemperer, this is not a rational indication. The carbonate and citrate of lithium are efficient diuretics, but have no other claim to virtue in this disease. Among natural waters of special value abroad are Vichy, Carlsbad, Homburg, Ems, Kissingen, Aix, Buxton, and Bath, and in this country Saratoga and Bedford. These waters are to be taken in large quantities and when the stomach is empty. It is highly probable that the environment, rigid system of hygiene, including exercise and an appropriately modified dietary, play the principal rôle in producing the favorable results obtained at these noted springs.

(*b*) *Medicinal Treatment.*—During an acute attack the pain, if excruciating, is to be relieved by a hypodermic injection of morphin, which is to be followed by a purgative dose of some mercurial. Colchicum is the specific remedy, and must be administered, in the form either of the wine or the tincture, in doses of ♏xx–xxx (1.333–1.999) every four hours. It alleviates the inflammation and promptly relieves the pain, but its effects during the attack should be carefully noted. After the paroxysm it should be continued, though in small doses, combined with the citrate or bicarbonate of potassium or lithium. The limb should be raised and the affected joint or joints wrapped in flannel or cotton-wool. Warm alkaline solutions or hot fomentations often afford relief in the worst cases, and anodynes may be tried locally. The diet should consist chiefly of milk, animal broths, and egg-white during the attack, later rice, eggs, fish, and other light forms of meat may be added, the more liberal dietary previously indicated being slowly resumed.

In the *intervals* between the acute attacks the prophylactic and dietetic measures previously mentioned are to be resorted to, in order that recurring paroxysms may be prevented, and in addition the alkaline diuretics and saline laxatives, together with warm bathing, will be found of the utmost value.

In *chronic* and *irregular forms* of gout medicines are of subsidiary importance, and are in no wise comparable in their beneficial effects to the previous recommendations. Two agents deserve prominent mention, however. They are piperazin and the extract of *thymus* gland. The ingestion of the latter, as obtained from the calf, is followed by an increased excretion of uric acid. Piperazin has been warmly advocated in all forms of gout for its supposed effect as a solvent of uric acid, and clinicians are almost unanimous in reporting its favorable results. Its beneficial effects are probably due to its diuretic action. The dose is gr. v–x (0.324–0.648) thrice daily, freely diluted with water. Some authors highly recommend the salicylates for acute attacks of gout, both primary and intercurrent, in the course of the chronic form. In my own experience they have been less effective in this disease than colchicum, though ammonium salicylate or salicin may be tried if there be present marked gastric disturbance, since they are better borne under these circumstances than colchicum. Luff has demonstrated by experimentation the negative value of the alkalies and salicylates in the treatment of gout. If nephritis or a failure of compensation be present, even the former remedies should be administered with extreme caution.

For *chronic gout* potassium iodid has been much used, though with slight advantage to the patient, I think. The bitter tonics, combined with a vegetable salt of iron, should be resorted to in the anemic, debilitated class of gouty patients, and a change of climate often serves to improve bodily vigor in the same category of cases.

LITHEMIA.

Definition.—A condition due to a disturbed cellular metabolism. It is characterized chemically by an excess of uric acid in the blood, and clinically by various digestive, circulatory, genito-urinary, and nervous phenomena. My purpose in describing lithemia is that the common error may be avoided of attributing its symptoms to other causes.

Pathogenesis and Etiology.—Lithemia is comparatively a latent condition. There is an *excess of uric acid*, which may be for a time eliminated through the natural channels (kidneys, lungs, skin, etc.) without the occurrence of symptoms. On the other hand, when, as the result of too little exercise, impaired elimination, luxurious living, the use of sweet wines, combined with the neurotic temperament, uric acid is allowed to collect in different parts of the organism, marked disturbances—nervous, gastro-hepatic, etc.—follow. Da Costa defines lithemia as a condition in which "the income of nutriment is in excess of the output of waste." C. G. Stockton holds that lithemia is a gastro-intestinal auto-intoxication. Among agencies that *predispose* are alcoholism, heredity, climate (temperate or cold climates favor diminished action of the skin), and the male sex.

Symptoms.—The nervous, circulatory, respiratory, integumentary, and genito-urinary symptoms are practically the same as those described under Irregular Gout; but I would here emphasize the broad clinical fact that the urethral and genital mucous membranes often become inflamed on slight provocation, producing urethritis, cystitis, orchitis, epididymitis, vaginitis, endometritis. These conditions resist treatment.

Gastro-intestinal Symptoms.—The appetite is variable, sometimes voracious, and at other times it is impaired or perverted. The tongue is coated, and a metallic taste is often complained of, while various forms of indigestion attend. There may be a delay in the conversion of the albuminoids, causing *pyrosis*, *gastric oppression*, *fulness*, and sometimes nausea and vomiting soon after food. These symptoms, together with *marked flatulence*, are manifested at a later period after meals if there be failure in the digestion of the carbohydrates. The bowels work irregularly, and there may be diarrhea attended by colicky pain, with frothy and ill-smelling discharges. Hemorrhoids are usual, and melena may occur independently of the hemorrhoids. The liver is somewhat enlarged and often tender. A few prominent **cardio-vascular symptoms** should be mentioned, such as *palpitation*, particularly after eating. More rarely it occurs while the patient is at rest or even lying abed. *Increased arterial tension* develops early, but may not be constant, and is due probably to the action of the uric acid in the blood upon the vaso-motor nerves, exciting universal contraction of the arteries. This condition may be present for a long time before actual arterio-sclerosis is in evidence. The latter complaint invariably follows, however, and sooner or later the well-known group—chronic gout, arterio-sclerosis, and granular kidney—will be presented.

Treatment.—(1) **Prophylaxis.**—The patient should be taught the lesson of thorough mastication, and robust, plethoric persons should exercise with method in the open air, with a view to consuming the fats in the body. For this purpose cycling, horseback-riding, rowing, and walking are all excellent. Nervous persons, however, demand rest (Gray). The constant use of lithia-water, more particularly in the spring of the year, is warmly advocated by Wilcox.

(2) **Diet.**—As in gout, so in the preliminary stages of lithemia, no single dietary suits all cases, though I agree with those who contend that a

diet consisting chiefly of albuminoids is proper in most cases. The lighter forms of albuminous articles of diet are to be preferred, and, if well borne, fruits and green vegetables may be added; but fried meats of all sorts and made-over dishes are to be eschewed. Assuming that certain cases are dependent upon an auto-intoxication from the gastro-intestinal tract, the object should be to limit fermentation by the use of a nitrogenous diet; and I have found large amounts of water very beneficial in such instances. There are cases in which the gastric digestion is feeble, and in such the carbohydrates are better borne than the albuminoids. Cream and good butter are the only forms of fat to be allowed. Alcohol should be interdicted.

(3) **Medicinal Treatment.**—If the patient be robust, it is well to begin with a saline laxative, such as Carlsbad Sprüdel salt (ʒj–ij—4.0–8.0). moderately diluted and taken before breakfast. If necessary, the hepatic function may be stimulated still further by a mild mercurial or by podophyllin. On the other hand, the neurasthenic, delicate sufferer must use a milder form of laxative, such as Rochelle salt in the same dose, or sodium phosphate in the morning, or a rhubarb pill at night. This class of lithemics also requires nerve-sedatives (sodium bromid, etc), and diuretics to aid in the excretion of uric acid. If it be true, as some claim, that the sodium phosphate is for the greater part excreted by the urine, and that it holds in solution more uric acid than any other salt, it is one of the foremost remedies in the treatment of the affection. Personally, I have found it to be a most useful agent. To reduce acidemia and to stimulate gently hepatic activity the salts of lithium, highly diluted, may also be tried. To aid in the digestion of the albuminoids hydrochloric acid may be needful, and if the appetite be impaired it may be combined with a simple bitter or with nux vomica (♏x–xv—0.666–0.999) thrice daily.

RACHITIS.

(*Rickets.*)

Definition.—A constitutional disorder of childhood, exhibiting developmental anomalies, chiefly in the bones and cartilages, causing deformities.

Pathology.—A mere summary of the anatomic characters can be given here. There is a derangement of the nutritive processes which retards and otherwise modifies the growth of the bony skeleton, particularly of the skull, the ends of the ribs and of the long bones. The latter soften or remain unduly flexible as the result either of the absorption of ossified structures or of the greatly diminished deposition of lime-salts. Longitudinal section of the long bones shows the seat of the chief changes to be at the junction of the epiphysis with the shaft. In health we note at this point two thin layers, an outer (next to the epiphyseal cartilage) proliferative zone, and an inner layer (of ossification). In rachitis both zones, though more particularly the proliferative, are greatly thickened, much softened, and their margins irregularly notched. The periosteum is thickened and easily separable from the shaft.

A *microscopic* examination shows an increased rate of proliferation of the cartilage-cells with a scanty, fibroid matrix, while the ossific layer presents disseminated and imperfectly calcified areas. Similarly, the deep (osteoblastic) layer of the periosteum is thickened, and remains spongoid. It is highly probable that absorption of true bone-tissue rarely occurs, and that the most characteristic pathogenic change is a lack of development of the normal structures. The morbid changes probably arise, as Kassowitz contends, from the presence of hyperemia of the cartilage, marrow, and periosteum—a process that interferes with the deposition of lime salts.

The cranial bones present areas of the so-called craniotabes, and yield to the pressing finger in consequence of delayed ossification. This may lead to a disappearance of the cranium in certain areas, causing depressions, while flattened protuberances may develop over the arterio-lateral regions. When cases terminate in recovery the bones become hard and ossify, although the deformities persist. The chemist has shown us that rachitic bones may contain less than half the normal percentage of lime-salts. The liver and spleen are moderately enlarged, and rarely the mesenteric glands are increased in size.

Etiology.—(1) Rachitis may occur in the *new-born*. Schwartz states that among 500 new-born children in Vienna, 75.8 per cent. show distinct signs of rachitis. Doubtless this estimate is too high, and entirely at variance with the experience of clinicians in general; but I believe that congenital rickets is by no means a rare condition. Many of the cases are still-born, and those that outlive childhood become peculiarly dwarfed (*micromania*). (2) *Heredity.*—The instances in which rachitis develops at an early period of life, due to ante-partum causes, are not rare, but it must not be forgotten that it is extremely hard to estimate the influence of heredity where both parent and child are exposed to similar unfavorable hygienic and dietetic conditions. Ill-health, malnutrition, close confinement, lactation, and syphilis may all act as predisposing factors during pregnancy. Setting aside syphilis, and perhaps phthisis, the state of the health of the father has little if any effect in the causation of rachitis in his offspring. (3) *Geographical Distribution.*—The disease is more common by far in large cities than in rural districts, and in European countries—Russia, Germany, Great Britain, and Italy more especially—the disease prevails more extensively than in America. (4) *Race.*—The colored race furnishes a preponderance of rachitic subjects. The reason for this may be a racial need of warmth that is not supplied by the temperature of more northerly latitudes, their native habitat being in a more southerly climate. The Italian race also suffers inordinately. (5) *Station.*—It is especially among the ranks of the poor children, whose environment is highly unfavorable, in large cities that rachitis is seen. Joukownski, from personal observations in over 3000 poor children in St. Petersburg examined for rachitis, found that from the working-classes come the greatest number of cases. Like scurvy, rickets may be found in the families of the wealthy under perfect hygienic conditions (Osler). The quarters of the cities in which the poorer classes live are densely crowded, the dwellings are insufficiently ventilated, and there is a great lack of sunlight. (6) *Diet.*—The disease is dependent largely upon unsuitable or insufficient food; and among hand-fed children, especially if the milk is sterilized, the disease is much more common than among those at the breast. It also occurs in breast-fed infants when the mother's milk is poor in quality as the result of previous ill-health or too long-continued lactation. The view was at one time widely held that rickets was produced by a farinaceous diet, and that the active agent was lactic acid, produced by the fermentative processes set up by the starch. Even granting, however, that the lactic acid forms a soluble salt by union with the lime of the bone, thus removing it from the system, this does not explain the productive lesions described under Pathology. According to another view, which is supported by experimental proof, rachitis is apt to develop when the amount of both proteids and fats is low. Certain forms of diet predispose to rickets, principally for the reason that they do not supply certain necessary articles in adequate proportion. (7) *Age.*—Of 903 cases, more than 75 per cent. occurred before the end of the second year; but of these only 99 commenced during the first half year (Bruennische, Von Rittershain, Ritsche). It may occur as late as the tenth year. (8) *Sex* is without effect. (9) *Syphilis.*—Divers views are entertained regarding the rôle played by syphilis as a cause of this disease. Doubtless the two affections are sometimes associated, and it cannot be denied that syphilis brings about a marked impairment of nutrition both in the mother and the child, so that the disease may engender a predisposition to rickets.

Bacteriology.—Mircoli contends that it is produced by the action of ordinary pyogenic organisms upon the osseous and nervous systems. He adduces clinical and pathological evidence in support of this position.

Symptoms.—The onset is *slow*, and the symptoms of gastro-intestinal catarrh, with their usual effect upon the general nutrition, may precede or accompany the true rachitis symptoms. At the beginning the infant is restless, irritable, and sleeps poorly, and slight fever is present in some cases. About the head and neck the child perspires freely, especially when asleep, wetting his pillow while the rest of the bed is dry. It is also annoyed by the bed-clothes, which it continually throws off, lying exposed even in a cool temperature. Among the *earlier symptoms* is a tenderness both over the bony surfaces and the soft parts, so that the patient wishes to keep still and dreads to be handled. The child is languid and disinclined to move his limbs or to walk or play, even if he have done so previously.

The symptoms are progressive in their development, rachitis being ordinarily a chronic disease, so that after many months more pronounced features, including various bone-deformities, appear. Owing to the impairment of nutrition of the muscles the use of the limbs may become impossible, and these cases have been spoken of by writers as "rachitic paralysis;" this, however, is a misnomer. Cases have been reported by Berg and others that resembled spastic paralysis, pseudo-hypertrophic paralysis. Urinary phenomena are neither constant nor characteristic. Secondary anemia of mild grade supervenes, the hemoglobin often being comparatively low, and there may be a leukocytosis.

The first rachitic *osteal changes* are presented by the cranial bones, the ribs, the radius, and the ulna. The cranium appears enlarged, though this enlargement is more apparent than real, being due to the diminished size of the facial bones. The sutures remain open, the fontanels are large, and their closure is delayed, sometimes until the fifth or even the eighth year. *Craniotabes* is most frequently seen in infants under one year of age. This soft, thin condition of the bones is due to pressure oth from within and without; it occurs on the surfaces on which the ead of the child rests while lying. To detect the presence of craniobes light pressure with the fingers is to be made in a direction away rom the sutures. It is to be recollected that craniotabes is often a yphilitic manifestation. *Per contra*, increased hardness of certain bones ay be observed (cranio-sclerosis). A *rachitic head* generally aproaches a square in outline, or it may present marked angularities, ith an increase in the antero-posterior diameter and a flattened top. Hyperostosis may cause prominence of the parietal and frontal emiences, giving the forehead a square, broad outline. A short, round head (brachycephaly) may rarely be met (Bonnifay). The veins of the scalp are enlarged, and the hairy growth is usually scanty, being often removed from the back of the head by rubbing. Drs. Whitney and Fisher first called attention to the fact that the ear placed over the anterior fontanel often detects a systolic murmur. A considerable patency of the anterior fontanel both in health and disease allows of detection of this murmur, however, and hence its diagnostic value is slight. A prominent feature of the disease is delayed teething, the teeth that appear being deficient in enamel, ill-shaped, although not prone to decay.

The *ribs* early become beaded. Anteriorly, where they join the costal cartilages, swellings occur, causing the "rachitic rosary." This is composed of nodules corresponding with the costo-chondral articulations, and these can generally be seen and always felt under the skin. They rarely outlast the fourth or fifth year. The ribs present two short curves —one at the junction of the dorsal and lateral parts of the thorax, and the other in front, where they turn sharply inward toward the sternum. This deformity is the result of the atmospheric pressure upon the softened bones, a shallow groove usually being produced in the line of the costochondral articulations or obliquely from the second or third rib downward and outward. These changes lessen the transverse diameter of the thorax in front and interfere with the lung-expansion in the antero-lateral portions of the chest. They also produce bulging of the sternum, resulting in the so-called *pigeon* or *chicken breast*. On both sides, from a point corresponding to the anterior end of the eighth or ninth rib, there passes outward toward the axilla a furrow (Harrison's groove) which is caused by an eversion of the lower part of the thorax, and is heightened by atmospheric pressure, particularly during inspiration. This thoracic deformity is not peculiar to rickets, but is met with in all cases in which there is moderate obstruction to the ingress of air into the lungs.

Among the first indications of rickets is an enlargement of the lower end (junction of the shaft and epiphysis) of the *radius*. The radius and ulna are sometimes twisted and deflected outward, owing to the fact that some of the body-weight is supported by the hands when sitting or crawling. The clavicle may be thickened and curved near either end, and occasionally the scapulæ may be enlarged, but deformities of the upper extremities are rare as compared with those of the lower. Occasionally the vertebræ and intervening cartilages soften, with a resulting spinal curvature, and in such instances there is usually an antero-posterior curvature with which lateral deflexion may be associated.

Pelvic deformities are not uncommon, and are of no little importance in female children as bearing upon the questions of marriage and subsequent labor. The femora may be curved, often forward and more rarely outward; swelling of the lower end of the tibia is, however, the first change to be observed in the lower extremities. In some well-advanced cases the heads of the bones forming the knee-joints are also enlarged, and outward curvature of the femora and tibia is common, especially under the age of one year (see Fig. 30). After the child begins to walk a forward bowing of these bones, due to the weight of the body and to muscular action, occurs. Knock-knee is sometimes observed. Those who have suffered from rickets in infancy usually fall short of the average stature on reaching adolescence, giving rise to disproportion between head and height.

These skeletal changes sustain a causal relation to many, and some serious, affections, chiefly *nervous*. Thus, craniotabes is supposed to induce laryngismus stridulus, though this condition may also arise in the rachitic without cranial softening. In like manner, rickets predisposes to tetany, which affects most commonly the upper extremities. Convulsions are also prone to occur in this disease. The reflex nervous excitability is unquestionably exaggerated in rickets, and another exciting cause for the eclampsia so often met with is the associated gastro-intestinal catarrh. The abdomen becomes greatly enlarged, chiefly by flatulence, though to a less extent also by the swelling of the *liver* and *spleen*. *Chest-complications* are common. Most of them are due primarily to a mechanical interference with the cardio-pulmonary circulation, and with the respiration. Among these are atelectasis, bronchial catarrh, broncho-pneumonia, and emphysema. *Green-stick fracture* of the bones often occurs in the rachitic subject.

Diagnosis.—Says Holt: "The most important early symptoms for diagnosis are sweating of the head, cranio-tabes, great restlessness at night, delayed dentition, and enlarged fontanel. All these, taken separately, may mean something else, but collectively they can mean nothing but rickets." At a later period the beading of the ribs and other characteristic deformities are usually present.

Prognosis.—The evolution of rickets is a long process; hence most patients become weak, anemic, and emaciated. The so-called "fat rickets," however, is not rare. Innately, the disease tends to spontaneous cure, which is attained from the end of the second to the fifth year; but its course may be abridged to a few months by appropriate treatment. When death occurs it is usually occasioned by one or other of the complications before mentioned (laryngismus stridulus, pneumonia).

Treatment.—**Prophylaxis.**—Simple means directed to the antepartum causal factors in the mother may in a large proportion of the cases be preventive of rickets. Prophylaxis also embraces appropriate feeding and other agencies that tend to maintain the normal nutrition of infants.

Hygienic Management.—*Proper feeding* is an important factor, and if the child cannot be satisfactorily nursed by its mother and if it is under the age of six months, a wet-nurse should be procured. Should this not be practicable, it must be hand-fed, and the best artificial food is cows' milk, if properly prepared. It is diluted to suit the age, and I have found that barley-water, when made in the manner recommended by J. Lewis Smith, may be added to milk, replacing the water most advantageously. A heaping teaspoonful of barley-flour is poured

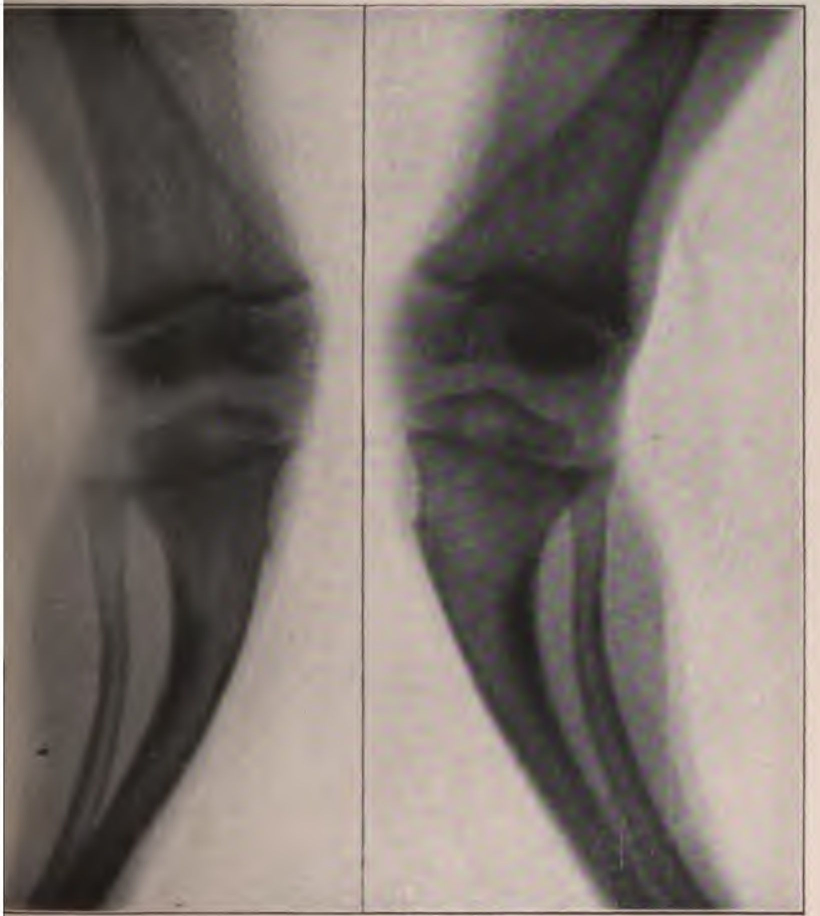

FIG. 30—Outward curvature of tibia and fibula (Willard).

into 25 teaspoonsful (℥iij—96.0) of water, and when the mixture is lukewarm 10 or 15 drops of diastase (Forbes) are added to it, the gruel in a few minutes becoming much thinner from the digestion of the starch. The physician must regulate with much precision the frequency of the feeding, and the amount of food taken according to the age of the child. The stools are also to be inspected. If they are green or if curds appear, either digestion is imperfect or the child is being over-fed. Older children may be given the lighter meats, freely, green vegetables, and fruits, but these must be carefully selected.

Other *hygienic details* are of little less importance than a proper diet. The decubitus of the child must be changed frequently, so as to prevent bony deformities; moreover, the rickety child should not be allowed to walk, and to prevent his doing so splints extending beyond the feet have been recommended. A tepid bath, warm clothing, and a prolonged daily stay in the open air are measures that should not be neglected.

Of *medicines*, those that rank highest are phosphorus, the hypophosphites, iron, and cod-liver oil. The officinal oleum phosphoratum (gr. $\frac{1}{150}$—0.0021) is used by Jacobi. Phosphorus is highly spoken of by many writers. It may be given either pure (gr. $\frac{1}{200}$ to $\frac{1}{100}$—0.0003 to 0.0006) or preferably in the form of an emulsion with sweet oil or cod-liver oil:

℞. Phosphori, gr. $\frac{1}{10}$ (0.00648);
Olei olivæ, ℥ij (64.0);
M. et ft. emulsio.

Sig. ʒj three times a day, after meals, for a child under the age of one year.

Kissel states there is no evidence in favor of the use of phosphorus in rickets. Baginsky, Leray, Weiss, and others have found from its extensive employment that it is of doubtful value in the majority of cases. Kassowitz, Swetchen, and others, however, observed cases with cure, hence the remedy deserves a trial.

When it is desired to administer cod-liver oil and it is not tolerated by the stomach, it may be rubbed gently into the skin of the thighs and trunk. Arsenic in small doses has proved to be a capital remedy in selected cases; and iron, particularly in combination with arsenic, is indicated if anemia be pronounced.

The numerous *complications* to which rachitic subjects are liable present special indications which are to be met by the same measures as when they arise under other circumstances. The condition of the digestive organs must be kept constantly in mind; and no remedy, however promising, that is designed to assist the general condition should be continued if it tends to aggravate the digestive disturbance or to embarrass the stomach. The treatment of the rachitic deformities belongs to the domain of the orthopedic surgeon.

SCORBUTUS.

(*Scurvy.*)

Definition.—A constitutional disorder, dependent upon dietetic errors, and characterized by anemia, excessive weakness, spongy gums, a tendency to muco-cutaneous hemorrhages, and a brawny induration affecting chiefly the muscles of the calves and the flexor muscles of the thighs. The fact is to be emphasized that while scorbutus and rickets are distinct affections, they may and often do coexist.

Pathology.—We know nothing concerning the pathogenesis of scurvy. Evidences of profound anemia are found upon microscopic examination of the blood, which is thin and dark, but there is no leukocytosis. The skin may show spots of subcutaneous hemorrhage (ecchymoses), but the most characteristic hemorrhage is that under the periosteum of the femora. Bleedings into the articulations and muscles may also at times be noted, and occasionally the serous membranes are the seat of hemorrhages, as well as the internal organs. Submucous hemorrhages are extremely common. The intestinal mucosa may also present ulcers. The gums are swollen, spongy, dark in color, and sometimes ulcerated, and the teeth may be loose or missing. The epiphyses, particularly of the lower end of the femora, may be congested, and rarely they are detached. The spleen is soft and swollen. The heart, liver, and kidneys sometimes show fatty and usually parenchymatous degeneration.

Etiology.—**Incidence.**—In former times scurvy was very prevalent among sailors at sea and soldiers in the field, and epidemics were common. Doubtless, however, it has declined in importance as a disease incident both to sea-life and to armies; but, as pointed out by Wise, it would seem that changing physiologic and economic conditions may cause it to be dreaded on the land as it has hitherto been on the sea. Osler states that the disease is not infrequent among Hungarians, Bohemians, and Italian miners in Pennsylvania. It is rarely *epidemic* at the present day. F. A. McGrew records an epidemic (with a total of 42 cases) in Chicago, in 1894. *Endemic* appearances of scurvy are still common, particularly in portions of Russia (Hoffman) and elsewhere also, sweeping through prisons, barracks, almshouses, and other institutions of like kind. While the majority of cases met with are sporadic, the above facts point to the infectious character of scurvy.

Bacteriology.—Testi and Beri have isolated a micro-organism which has been cultivated and inoculated into guinea-pigs and rabbits, producing in the latter pathologic lesions and symptoms simulating closely those of scurvy. The microbe is perfectly round and is a diplococcus. These experiments require confirmation.

Predisposing Causes.—The chief factor is an unsuitable dietary long continued. The absence of the organic (potassium) salts present in fresh vegetables disturbs normal histogenesis. Albertoni has recently shown that in scurvy of a protracted course free hydrochloric acid is absent from the gastric juice, and that the total acidity is much reduced, but this is neither so in every case nor at all stages of the disease. He found no deficiency of chlorids in the body. Peptonization is feeble.

Debilitating influences, as unhygienic surroundings, excessive muscular exercise, humidity, and cold, often play no mean role in causing scurvy. Mental anxiety and depression seem to have etiologic significance. The old are very susceptible, and all ages are liable to the dis-se. *Sex* has no special influence upon scorbutus. Starvation does not redispose to the disease.

Symptoms.—Scurvy has a *slow* onset. The earliest symptoms are enerally a swelling around the eyes, over which the skin has the color f a bruise, and a pale face, which looks bloated and wears an apathetic xpression. There is noticeable almost from the start a gradually in-reasing debility, emaciation, an inability to perform mental or physical abor, and despondency. The patient experiences arthritic and muscular rheumatoid pains and dyspnea on slight exertion.

With rare exceptions the *gums* swell, sometimes enormously, and become spongy, bleeding most readily. They may become ulcerated, and may be, though rarely, fungoid in appearance. The *teeth* often become loose, and in rare cases drop out. The *breath* emits an offensive odor, that is sometimes due to necrosis of the jaw. The *tongue* swells, though it is usually clean and often pale. In the mouth may be observed submucous hemorrhages in many cases. There is loss of appetite, but the digestion is usually good; there may, however, be constipation or diarrhea, more frequently the former. *Scorbutic dysentery* has been described by certain writers. The *skin* is dry and of a muddy color, blended occasionally with a greenish or greenish-yellow tinge. At the end of a week or ten days *petechiæ* and *ecchymoses* appear upon the legs, arranging themselves about the hair-follicles. These may also come out later on the trunk and upper extremities. Submucous hemorrhages may give rise to circumscribed swellings, and *subperiosteal hemorrhages* may occur and engender node-like protuberances. There may be frequently noticed a peculiar brawny induration, due to extensive hemorrhagic infiltration of the muscles and subcutaneous tissues, most marked in the hams and calves. The condition is not without considerable *pain*, particularly if the parts be touched, and in severe cases bullæ and vibices may be seen, as in a recent case of my own. Hemorrhages from the mucous channels of the body occur, and epistaxis is frequent. In bad cases hematuria, also melena and rarely hematemesis, may be observed. Blood may be effused into the serous membranes, accompanied sometimes by inflammatory changes in the latter; also into the lungs, which are rarely the seat of secondary pneumonia. Pulmonary infarction occurs, but is a rare event. Hemoptysis may be a symptom of the lung-complications or may occur as an independent phenomenon.

The *heart* may present symptoms, such as palpitations, feeble impulse, arrhythmia, and sometimes a basic blood-murmur, but these are without diagnostic importance. The pulse is soft, small, and on exertion much accelerated. The *temperature* is sometimes subnormal, and the presence of fever is a certain indication of the existence of some complication.

The *nervous symptoms*, aside from the profound mental depression, are not prominent. Insomnia may be a distressing symptom. Delirium (late) is sometimes witnessed. Meningeal hemorrhage may supervene. Both night-blindness and day-blindness are among the rarer and extraordinary ocular features.

The *urinary symptoms* vary in different cases. Albuminuria is, however, common. The specific gravity of the urine is increased, the color high, and the solid constituents diminished, except the phosphates, which are abundant. Albertoni found the proportion of chlorids less than the normal, while other investigators claim that the percentage is high. *Nephritis* may occur as a complication. The bones in long-standing cases may be congested and sometimes necrotic, and the epiphyses may separate from the shafts. In one of my cases an old cicatrix reopened.

Diagnosis.—This rests upon the following points: the history, the peculiar facies, the spongy and swollen gums, the gingival and deep-seated cutaneous hemorrhages, the progressive loss of strength and energy, great mental depression, and the speedy recovery after an appropriate regimen. Scurvy will be distinguished from *purpura* under the description of the latter disease.

Prognosis.—Unless far advanced, the prognosis generally becomes good upon the institution of correct dietetic principles. If the disease have made extensive inroads, the danger to life is considerable. The gravity of the internal symptoms (particularly pulmonary) is far greater than of the external, and, indeed, the presence of the latter is a favorable omen. Certain complications augur a serious termination, such as pneumonia, hemorrhagic infarctions of the lung, pleurisy with bloody effusion, dysentery, acute nephritis, etc.

Treatment.—**Prophylaxis.**—By carrying out the known means of prevention the disease has been diminished more than 90 per cent. among mariners and soldiers. This change has been brought about by the enforcement of governmental regulations which demand that an adequate supply of antiscorbutic articles of food must be provided for military campaigns and for long sea-voyages. Fresh fruits and vegetables can be readily transported in hermetically sealed jars or cans.

Treatment of the Attack.—The chief indication is to be met by the use of fruits and fresh vegetables. Of the former, two or three lemons daily or oranges and other fruits suffice to work a surprising degree of improvement in a short space of time. Antiscorbutic vegetables (potatoes, water-cresses, raw cabbage, lettuce, saur-kraut) in liberal quantity should also be given. Meats, eggs, milk, and farinaceous dishes are not to be prohibited, since the patients require all forms of food to invigorate the system and to render normal the constitution of the blood; but if the digestive power be feeble, it is advisable to begin with the juice of oranges or lemons, conjoined with meat-juice, egg-white, milk, and light farinaceous articles, adding the stronger forms of animal food and fresh vegetables when improvement is noted. We may assist the digestive function in bad cases by the use of simple bitters, strychnin, and hydrochloric acid (after meals); hematinics are sometimes indicated.

Special symptoms may call for appropriate measures. Constipation requires simply an enema. On the other hand, diarrhea presents an indication for intestinal antiseptic and astringent remedies. The oral condition varies, hence the measures to relieve it vary also; but if ulcers be present, the solution of potassium chlorate is best. For swelling of the gums the application by means of a cotton swab of tannic acid (2 per cent.) or a solution of silver nitrate (2–5 per cent.) is serviceable. A combination of boric and carbolic acids in a solution of suitable strength may be used as a mouth-wash. If copious hemorrhages occur, hemostatics are eminently useful. The various complications must be met by the usual measures, according to their nature.

INFANTILE SCORBUTUS.

Definition.—A constitutional disease, characterized by the same mptoms as scurvy in adults, except that in many instances undoubted idences of rachitis are associated.

Pathology.—The bones are thickened and excessively sensitive, wing to a marked subperiosteal hemorrhage, with more or less maceraion, and want of firmness between the epiphysis and shaft. The muscles ay also be the seat of effusion. The characteristic lesions of rickets are ften associated.

The *nature* of the affection is unsettled. Originally looked upon by most observers as acute rickets, it was subsequently described by Cheadle (from the clinical side) and Barlow (from the anatomo-pathologic side) as infantile scurvy. On the other hand, Ashby of Manchester, Fürst and other German writers, are inclined to the view that the affection should be considered a hemorrhagic form of rachitis. The belief that rickets predisposes to scurvy, but that the two diseases have not the same pathogenesis, is probably the correct one.

Etiology.—Scurvy is almost confined to *hand-fed infants*, especially those reared upon the numerous infant-foods which have been foisted upon the market, including condensed milk, etc. Louis Starr, Jacobi, and others have shown that it sometimes follows the prolonged use of sterilized milk, although the etiologic importance of the latter food has been too much emphasized. An investigation by a committee of the American Pediatric Society [1] showed that of 379 cases the majority occurred between the ages of 7 and 14 months, inclusive, and that the disease has a greater tendency to occur among the rich or well-to-do. This committe's report also embraces the following among other justifiable conclusions: "The farther a food is removed in character from the natural food of a child the more likely its use is to be followed by the development of scurvy."

Symptoms.—The *skin* presents the muddy color peculiar to the disease in adults. The patient may be well nourished, but more often there is a tendency to *wasting*, and other symptoms of impaired nutrition appear, particularly irritability and disinclination to exertion. The more characteristic features appear after one or two months, and the child cries when handled, especially on touching the lower limbs. About the same time there is an irregularly cylindrical swelling of one of the thighs, due to subperiosteal effusion. Soon the other limb is similarly involved, though not always to a like degree. At first the legs are flexed, but later they become straightened and slightly everted on account of the progressive hemorrhage or separation of the epiphyses. The bones in other portions of the body may be involved secondarily in more or less rapid succession, but the swellings are less marked than in the lower limbs. Later, if teeth be present, the gums may swell and become spongy. Ecchymoses in the form of petechiæ appear upon the skin-surface, and particularly about the eyes. Barlow describes a remarkable ocular phenomenon: "There develops a rather sudden swelling of one eyebrow, with puffiness and very slight staining of the upper lid. Within a day or two the other lid presents similar appearances, though they may be of less severity. The ocular conjunctivæ may show a little ecchymosis or may be quite free."

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[1] *Medical Record*, July 2, 1898.

Hemorrhages from the mucous surfaces may finally put in an appearance.

Diagnosis.—To distinguish *rickets* from infantile scurvy Barlow's brief though clear aggregation of the characteristics of the latter disease may be quoted: "(1) Predominance of lower-limb affection, in which there is immobility going on to pseudo-paralysis; excessive tenderness; general swelling of the lower limbs; skin shiny and tense, but seldom pitting, and not characterized by undue local heat; on subsidence revealing a deep thickening of the shafts, also liability to fracture near the epiphysis. (2) Swelling of the gums about erupted teeth only, varying from definite sponginess to a minute, transient ecchymosis."

In incipient and anomalous cases there is danger of diagnosticating rheumatism when scurvy is really the condition present (Griffith).

Prognosis.—Favorable, even in well-established instances, if brought under the proper regimen.

Treatment.—An antiscorbutic dietary—mother's milk or fresh cows' milk, meat-juice, and orange- or lemon-juice—successfully meets the main indication. If there be systemic exhaustion—a condition that is not infrequent—gentle stimulation with brandy (highly diluted) and an abundance of fresh air are pre-eminent among the measures to be employed. Iron, arsenic, and cod-liver oil may be needful to complete the cure, but usually the simple means already mentioned will prove effective. The limbs, especially the lower, may claim attention. Local treatment, however, is rarely necessary, except there be separation of the epiphyses, when suitable splints are to be applied.

PURPURA.

Two main groups are to be distinguished: (1) **Secondary purpura**, which occurs from a great variety of causes and in numerous affections, in which its clinical significance has been pointed out in appropriate sections of this work. It seems pertinent, however, to enumerate the chief among the diseases and conditions under which it may arise, as follows: (*a*) scurvy; (*b*) acute, infectious diseases (cerebro-spinal meningitis, variola, measles, septicemia, ulcerative endocarditis); (*c*) hemophilia; (*d*) numerous chronic affections, as nephritis, leukemia, pernicious anemia, jaundice, Hodgkin's disease, and tuberculosis; (*e*) malignant sarcomata; (*f*) nervous affections, as locomotor ataxia, acute and transverse myelitis, and hysteria; (*g*) mechanical causes, straining efforts, intense paroxysms of whooping-cough, and violent convulsions; (*h*) certain drugs may produce a petechial eruption—quinin, copaiba, belladonna, ergot, mercury, and the iodids; (*i*) snake-poisons produce rapid and extensive hemorrhagic extravasations, as shown by the careful studies of S. Weir Mitchell.

(2) **Primary** or **idiopathic purpura** forms the second group. It is divisible into (*a*) simple purpura (*purpura simplex*); (*b*) arthritic purpura, of which two varieties may be recognized: (1) *peliosis rheumatica*, and (2) *Henoch's purpura*; (*c*) hemorrhagic purpura (*purpura hæmorrhagica*).

(*a*) **Simple Purpura.**—The cause is unknown. Among predisposing

influences, however, is *age*, the condition being most common in children about the time of puberty. It may be a sequel of the acute, infectious diseases, and in not a few cases develops in seemingly healthy subjects.

Symptoms.—This is the mildest variety of primary purpura. The *hemorrhages* into the skin take the form of petechiæ, vibices, or ecchymoses. The first are extravasations of blood in the form of minute points, that appear, as a rule, in the hair-follicles, and, unlike the erythemas, do not disappear upon pressure. The vibices receive their name from the fact that the hemorrhages occur as streaks, while the ecchymoses are larger, but similar in nature and behavior to the petechiæ. They may exceed in size that of a split pea, and their hue ranges from a deep red to a livid bluish tint. As they fade away they assume at first a yellowish-brown, then a yellow color, and finally disappear. The eruption appears in a series of crops, and its seat of election, often favored by the erect posture, is the legs. Bloody serum may be effused into bullæ or large blebs. Shepherd and others have reported cases in which the purpuric eruption ended in gangrene, though in Shepherd's case the gangrene was believed to be due to the use of sodium salicylate.

(*b*) **Arthritic Purpura.**—(1) *Peliosis Rheumatica* (*Schönlein's Disease*). —The *cause* of this remarkable disease is unknown. Formerly many writers inclined to the view that it is of rheumatic origin, and since endocarditis and pericarditis are occasionally observed in association with peliosis rheumatica, considerable coloring is given to this belief. On the other hand, the fact that the cardiac complications are rare in arthritic purpura shows that not all cases of the latter disease are genuinely rheumatic. It occurs chiefly in males from the twentieth to the thirtieth year of age. Among the *prodromata* are angina, slight articular pains, headache, loss of appetite, and fever ranging from 100° to 102° F. (37.7°–38.8° C.). The affection is especially characterized, however, by *polyarthritis*, the joints being swollen, painful, and very tender; also by purpura, associated or not with urticarial wheals or erythema exudativum; and by subcutaneous edema. The purpuric *eruption* is the only symptom that has pathognomonic significance, and in this affection it shows a strong preference, as regards distribution, for the affected joints and the legs. The eruption, as already intimated, does not display constant characteristics. It may not differ from that of simple purpura, and the rash consists of petechiæ, ecchymoses, streaks, and rarely of bullæ (*pemphigoid purpura*); or it may be made up of wheals of urticaria, attended with intense itching; and, finally, it may be identical with erythema nodosum. These forms of eruptions may be variously combined.

Hemorrhages from the mucous surfaces rarely occur, though epistaxis is the most common. The extent of the *edema* varies greatly, in rare cases being quite extensive and overshadowing all other symptoms (febrile purpuric edema). *Albuminuria* may be noted, and accompanying the purpuric eruption there will be a mild febrile movement. Convalescence is usually protracted (even into years), and is often interrupted by recurrence of the characteristic features.

The *diagnosis* is made from the presence of three characteristic symptoms—polyarthritis, a purpuric rash, and edema. The combination of purpura and urticaria is one of the chief distinguishing features. It is not always possible to eliminate *rheumatism*, but the non-rheumatic character of some of the cases may be clearly shown by the therapeutic test, as happened in one of my own patients.

Prognosis.—This type of the disease is generally benign, death being very rare. Complications, however, may prove serious, especially the cardiac. The throat-condition may outlast the attack, and terminate in gangrene of the uvula or tonsils.

(2) *Henoch's Purpura*—Henoch and Couty have described a form of rheumatic purpura occurring chiefly in children, and characterized by painful and sometimes swollen joints; by a purpuric eruption, plus erythema multiforme; by vomiting, diarrhea, and intestinal pain; by localized edema of the skin; and by hemorrhages from the mucous membranes and sometimes into the kidneys.

The *diagnosis* is difficult in proportion to the scanty development of the purpuric symptoms, some of which are often wanting.

The *prognosis* is favorable, though complications of more or less serious import may arise. One of Osler's cases proved fatal with the symptoms of acute hemorrhagic Bright's disease.

(3) *Factitious Purpura.*—Bruce and Galloway[1] report a case in which any irritation of the skin, such as might be caused by drawing the blunt end of a pencil over it, produced a white line, which presently became pink and then intensely purpuric. In this way letters, figures, and the like could be shown as hemorrhagic outlines.

(*c*) **Purpura Hæmorrhagica** (*Morbus Werlhofii*).—This is the severest form of purpura, and its apparent etiologic connection with certain infectious diseases, particularly rheumatism, malaria, etc., is interesting, but not well understood. The disease is perhaps most common in young females, particularly if they have fallen into general ill-health; but all persons are liable, and post-mortem anatomo-pathologic pictures of the disease leave little room for doubt that it is an infectious complaint.

Symptoms.—*Prodromal symptoms*, (malaise, headache, depression, anorexia) may appear, and last one or two days. The invasion is moderately abrupt, with fever, and soon cutaneous ecchymoses appear upon the skin, quickly increasing in size and numbers. Slight *hemorrhages* from the mucous membranes into the internal organs occur. Epistaxis generally comes first; it tends to persist and to recur, and the same peculiarities pertain to bleedings from other points. *Prostration* now becomes rather marked, the patient complaining of pains in the limbs, loins, abdomen, and chest, and the latter often presage a fresh hemorrhage. There is moderate *fever*, as a rule, the temperature during the height of the attack ranging from 101° to 103° F. (38.3°–39.4° C.), or it may reach 104° to 105° F. (40.5° C.), though rarely. The *pulse* is accelerated (120 to 130 per minute), but full and regular, though in the worst cases it becomes small and very rapid. The mind is usually clear. *Hematuria* followed by *nephritis* may occur.

There is *anemia* varying in intensity with the extent of the hemorrhage and the severity of the type, and showing the characteristics of symptomatic anemia. The face may be exceedingly pale and anxious. The *course* is run in from seven to ten days in mild cases, while the severer attacks pursue a longer course. It is to be recollected, however, that the malignant form (*purpura fulminans*) has a speedily fatal termination.

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[1] *British Jour. of Dermatology*, Jan., 1898.

The *diagnosis* of purpura hæmorrhagica rarely presents any difficulty. *curvy* may simulate it in some particulars, but is distinguished by its hief etiologic factor—a diet deficient in fresh vegetables and fruits—by he spongy, swollen condition of the gums, the loosened teeth, and brawny nduration of the limbs. Moreover, in purpura hæmorrhagica the hair-ollicles do not occupy the centers of the ecchymotic spots, and the hemor-hages from the mucous membranes are more copious than in scurvy. Malignant types of the eruptive fevers distinguish themselves by the his-tory of the prevailing epidemic, by the characteristic prodromes and in-vasion, and by the high temperature. It must be remembered, however, that variola purpura often pursues an afebrile course.

Prognosis.—Grave, except in mild cases. In the malignant type death may come before hemorrhages from the mucosa appear. Certain complications may prove fatal—cerebral hemorrhage, inundation of the lungs with blood, Bright's disease, and shock from rapid, profuse bleedings. Death may also be the result of exhaustion due to protracted bleedings.

Treatment.—(*a*) The management of *secondary purpura* is embraced, in other portions of this volume, in connection with the treatment of the diseases and conditions which it accompanies.

(*b*) *Simple purpura* demands arsenic, first in moderate doses, and then increased until slight toxic effects are noticeable. Legroux speaks in warm terms of the iron compounds, and especially of iron perchlorid in doses of ʒss–j (2.0–4.0) daily, and if the child is somewhat anemic, the inhalation of oxygen will promote hematosis. The disease also requires fresh air in abundance and a generous diet.

(*c*) *In peliosis rheumatica*, in addition to the measures recommended in purpura simplex, the salicylates should be tried.

(*d*) *Purpura Hæmorrhagica.*—In all kinds of purpura the patient should be confined to bed. An abundance of nourishment, by supporting the patient's power, is of the greatest service. Internally, ergot, turpentine, tincture of the chlorid of iron, acetate of lead, and dilute sulphuric acid enjoy the widest reputation. Calcium chlorid, suggested by Wright, should be tried when other remedies fail. The following combination, recommended by Hardaway, I have found useful:

℞. Ext. ergotæ fl.,
Tr. ferri chlorid., *āā* f℥ij (64.0).—M.
Sig. Three to ten drops in water, t. i. d.

HEMOPHILIA.

(*Bleeder's Disease.*)

Definition.—An hereditary affection, transmitted by females who are themselves not affected (Nasse's law). It is characterized by frequent uncontrollable hemorrhages that are either spontaneous or due to slight traumatism.

Pathology.—The constitutional changes or peculiarities on which the disease depends are to be found in the blood-vessels rather than in the blood itself (Henry); microscopic changes have been found in the arterioles, the middle muscular tunic being either absent or much atrophied. Vaso-motor influences also play an important part in causing an attack, as is shown by the frequent flushings of the face preceding an attack, and also by the fact that bleeding may follow emotional excitement (Henry). Synovitis with hemorrhages into the joints may sometimes be observed. The blood presents slight change.

Etiology.—Hemophilia is more distinctly hereditary than any other known disease, but Nasse's law is not of such universal application as is supposed. R. Kolster found that of 50 hemophilic families, 18 cases followed this law, 16 others with some exceptions to its provisions, and 12 without any regard to it. The law embraces the following points: The daughter (not herself affected) of a bleeder transmits the tendency to her sons, who become bleeders; her daughters do not suffer, but in turn transmit the disease to their sons. Females, however, may be bleeders, and, according to Virchow, one woman is affected to every seven men. The disease has been traced for centuries in a few families.

It is observed in all classes of society, and is most frequent in families whose members are large, vigorous, and have delicate complexions, the complaint usually manifesting itself before the end of the second year of life, though exceptionally as late as puberty. An acquired hemorrhagic diathesis is seen occasionally in connection with certain acute infections and more commonly in the graver anemias (leukemia, pernicious anemia).

Symptoms.—The occurrence of profuse and persistent bleedings that are either spontaneous or the result of slight injury characterizes hemophilia. The character of the injuries that lead to dangerous bleedings is often exceedingly trivial; thus a slight scratch, cut, blow, the extraction of a tooth, and other minor surgical operations (*e. g.* circumcision) may be followed by severe hemorrhage.

If we include spontaneous hemorrhages, bleedings take place most frequently from the nose. Legg has made three clinical groups, based on the intensity of the symptoms, as follows: (1) Seen most frequently in men, and characterized by external and internal bleedings of all kinds and by joint-affections; (2) most frequent in women, and distinguished by spontaneous hemorrhages from mucous membranes only; and (3) characterized simply by ecchymoses.

The capillaries ooze blood—a process that may vary in duration from a few hours to as many weeks. A fatal result may thus occur in a few hours, while, on the other hand, recovery may follow a slow oozing of blood that has continued for many days. In the latter instances profound anemia follows, the blood, however, being rapidly replaced. Extensive blood-extravasations (hematomata) usually follow contusions. Petechiæ, when they occur, are apt to be spontaneous. The blood coagulates, except in long-standing hemorrhages, when it becomes thin and watery (late). Fussell made blood examinations in two cases, and found the leukocytes slightly increased (14,000 and 15,000 per c.mm.), while the red cells were moderately diminished.

Arthritic symptoms are common, the larger joints, and especially the knees, being most frequently affected and showing swelling that is due chiefly to hemorrhages into the joints. In other instances febrile synovitis may be present, resembling rheumatism. The joint-symptoms may either announce an approaching hemorrhage or pain alone may be exrienced. The attacks are liable to recur, especially in cold, damp eather, and may result in stiffened, deformed joints (Musser).

Diagnosis.—When persistent capillary oozing occurs in a person ith a clear, hereditary disposition the diagnosis is clear. Without an herited tendency we cannot be certain of the diagnosis unless proracted hemorrhages from insufficient causes are repeatedly manifested. The presence of joint-involvement is very helpful.

Differential Diagnosis.—*Peliosis rheumatica* is an affection which, as Osler remarks, touches hemophilia very closely, particularly in the reation of the joint-swelling. It is true that the former may also show tself in several members of a family, but the presence in this affection of more or less edema, and often of wheals of urticaria, accompanied by intense itching, aids greatly in its elimination.

Prognosis.—In undeveloped forms the outlook is not particularly grave, since in these the tendency may either lessen or become altogether arrested after childhood. In the majority of well-marked cases the children do not survive this period. On the other hand, those who live to become full-grown show a diminished, and in a small class of cases an absolute, disappearance of the tendency. The first hemorrhage rarely proves fatal. Boys suffer from a more serious form than girls. Moreover, menstruation, though sometimes very copious, does not to any great extent endanger the life of a hemophilic woman. Of 130 cases of pregnancy and labor, the death of the mother occurred in only 3, and abortion in 16 cases (Kolster).

Treatment.—The physician can do most in the direction of prophylaxis. All surgical operations that are not absolutely necessary must be avoided; neither should the teeth be erupted nor the operation of circumcision be permitted. Leeches are not permissible. Females who belong to bleeder families, as well as males who have had hemophilia, should not marry.

During the attack absolute rest—mental and bodily—must be enjoined. and light compression, and if this fail strong pressure or styptics, should be tried. In epistaxis ice, tannin, and turpentine should be tried before using nasal plugs; and if the latter prove indispensable, the lightest only should be employed. J. Greig Smith regards lint saturated with spirit of turpentine as the best local application in epistaxis. The application of normal human blood to the bleeding surface is warmly recommended. Gelatin has recently been used topically with a successful result.[1] Internal medicines are of secondary importance, though they may be tried, and opium is unquestionably of signal value, since it tends to quiet the patient, thus favoring repose. The remedies that have been given are various. Delafield, Fürth, and others have used successfully the fluid extract of hydrastis canadensis, the dose being from 20 to 40 drops daily; among other hemostatics, gallic acid, turpentine, and iron perchlorid produce the best results. The dose of the latter should be ʒss (2.0) every two hours, with a purge of sulphate of soda (Legg). The use of calcium salts has produced good results in some cases and merits a trial. Thyroid extract and inhalations of oxygen have also been advocated. During convalescence arsenic, iron, the bitter tonics, and a liberal dietary, will aid full recovery.

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[1] J. B. Nichols, M. D., *Medical News*, Dec. 2, 1899.

HEMORRHAGIC DISEASES OF THE NEW-BORN.

(*a*) **Epidemic Hemoglobinuria** (*Winckel's Disease*).—This affection, which is *septic* in nature, is occasionally met with in lying-in hospitals, and occurs in children from one to ten days after birth. The infants refuse the breast and show hematogenous (?) icterus; gastro-enteric catarrh is an attendant of the disease. The stools are meconic; the urine is scanty, dark-colored (from the presence of methemoglobin), often albuminous, and may contain casts. Hemorrhages occur into organs other than the kidney and into the mucous membranes, there also being mild fever, rapid emaciation, and often mild convulsions. It is a very fatal disease. Bacteriologic experiments have shown that the disease may be produced by the growth of the colon bacillus in the buccal epithelium of infants. Kilham and Mercelis[1] report an epidemic of 10 cases occurring in the New York Infirmary; complete bacteriologic studies were made in all, and the organism discovered suggested the diplococcus of pneumonia or the pneumococcus group. There is, however, great confusion in regard to the possible specific micro-organism of this disease.

(*b*) **Acute Fatty Degeneration of the New-born** (*Buhl's Disease*).—This disease may be similar to Winckel's in nature. It was first described by Hecker and Buhl as an infectious disease of the new-born, characterized by cyanosis, jaundice, and copious visceral hemorrhages. The chief *pathologic change* is an acute fatty degeneration of the internal organs.

(*c*) **Syphilis Hæmorrhagica Neonatorum.**—Either at birth or soon thereafter bleedings take place into the skin (ecchymoses) and from the mucous surfaces and the navel. Jaundice may be associated. The viscera are found upon post-mortem examination to be the seat of syphilitic lesions.

(*d*) **Morbus Maculosus Neonatorum.**—Hemorrhage from the gastro-intestinal mucosa of the new-born (melæna neonatorum) occurs, and may be due to intracranial lesions during birth; it may also take place independently of the latter. Preuschen has collected the reports of 37 cases, in 5 of which the brain was examined, and all of these showed cerebral hemorrhages. The latter may occur in spontaneous births and give rise to melæna neonatorum. Gärtner believes the disease to be an infectious one, and claims that in 2 cases he was able to identify a bacillus for which the navel is believed to be the entrance-point. The blood may also come from the mouth, nose, navel, etc. Townsend found morbus maculosus neonatorum in 45 cases in 6700 deliveries, and in most of these instances the bleeding was general. The hemorrhage usually sets in during the first week, rarely later, and the duration of the disease is between one and seven days, the mortality being a little over 50 per cent. Vomiting of the blood which the child has drawn from the breast must not be confounded with true melena. The *treatment* is by gallic acid and ergotin, the latter hypodermically; stimulants may also be required, and warmth to the extremities if the peripheral circulation be sluggish.

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[1] *Archives of Pediatrics*, March, 1899.

PART III.

DISEASES OF THE BLOOD AND THE DUCTLESS GLANDS.

ANEMIA.

Definition.—A pathologic condition, characterized either by a diminution in the quantity of blood or by a deficiency in one or more of its constituents. Anemias may be subdivided into—I. Primary or Essential (simple, chlorotic, and pernicious); II. Secondary (symptomatic); III. Leukocytosis; IV. Leukocythemia (splenic, myelogenic, and lymphatic).

Pathology.—Anemia, in its different forms, is characteristic of diseases of the blood or of the blood-making organs. It may be manifest, on examination, as a diminution of the total quantity or body of the blood (*oligemia*); of the number of red corpuscles (*oligocythemia*); of the hemoglobin (*oligochromemia*); and of other constituents, as albumin (*anhydremia*). The diminution of hemoglobin gives rise to the most obvious sign of anemia or impoverished blood—namely, the pallor of the cutaneous surface—but it is important to point out here that the quantity of hemoglobin in the blood is not necessarily proportionate to the number of red corpuscles. Thus the percentage of hemoglobin contained by the red corpuscles may vary in disease, so that a reduction in its amount does not necessarily involve a corresponding decrease in the number of red corpuscles. Conversely, a diminution in the number of the latter may not be accompanied by a proportionate diminution in the amount of hemoglobin, the corpuscular richness in coloring-matter being quite normal. As a matter of fact it frequently happens that oligochromemia is associated with a certain degree of oligocythemia, and *vice versâ*, though where they coexist the degrees of reduction may neither be relatively nor proportionately equal.

Anemia can be positively ascertained only by an adequate examination of the blood. It may be inferred from the presence of pallor, languor, dyspnea, palpitation, etc.; but it should be borne in mind that not every pale person has anemia, since pallor of the face may be hereditary, and, at the same time, perfectly consistent with good health, a normal number of corpuscles, and a normal percentage of hemoglobin. Conversely, a person with marked vascularity of the face, and a rosy complexion even, may have anemia.

The anemias embrace those conditions, also, in which there are changes in the shape of the red corpuscles (*poikilocytosis*), and in their size (*micro-*, *macro-*, or *megalocytosis*).

I. THE PRIMARY OR ESSENTIAL ANEMIAS.

Primary anemias constitute those forms in which, so far as our present knowledge of their etiology and pathology goes, no other tissues or organs than the blood and the blood-making organs are either at fault or are directly affected. Future investigations of the life-history of the blood may reveal the exact causation of what are now regarded as primary or essential anemias, and thus permit of a clearer discrimination and a more accurate classification.

SIMPLE OR BENIGN ANEMIA.

This form is not infrequently met with as a congenital, constitutional affection, without any assignable cause, and is entirely free from pernicious manifestations or tendencies. There is no discoverable element of relationship between simple benign anemia and chlorosis, nor is the former symptomatic of any disease in which anemia is common, such as tuberculosis, carcinoma, and nephritis.

Etiology.—Simple constitutional anemia is often met with among the poorer classes, and from this fact it is probable that living or working in a vitiated atmosphere, as well as deficient sunlight and nutriment, is primarily active in reducing the general health. In this way is often caused a lifelong pallor, due to an interference with the normal process of blood-making (*hemogenesis*). There are also certain individuals in whom slight pallor and systemic feebleness have existed from infancy (thus probably congenital), and whose modes of life and environment have been more or less uniformly hygienic and provident. In such cases we may assume that there is some innate imperfection—anatomic or physiologic, or both—in the blood-forming organs.

Finally, in the later manifestations of slight general anemia a developmental strain or abnormality may start a disorder of hematopoiesis in organs congenitally insufficient for new and greater demands for blood made by the system.

Symptoms.—There is some pallor, often with languor, slight palpitation, and dyspnea, occasional headache, and a tendency to fatigue. The general health is not otherwise disturbed, and an active life may be enjoyed for many years. Examination of the blood shows a slight reduction in the number of the red cells and of the hemoglobin (relative). This degree of anemia persists without aggravation or amelioration. It may be found to affect males and females, and is observed principally in adult life.

The **diagnosis** of simple, benign, or constitutional anemia should be made with considerable caution and reserve, and it should be arrived at only after the closest scrutiny of all the symptoms and signs, the most careful study and judicious balancing of the data entering into the previous history of the patient. If there be a latent or incipient tuberculosis, carcinoma, or nephritis, a previous attack of some infectious fever, rheumatism, etc., this fact clearly bears upon the case, and the diagnosis of simple anemia is precluded.

The **prognosis** is usually favorable. On account of the possible existence of one of the above-mentioned diseases, or from the fact that grave variety of anemia may be superadded, however, it should be arded in the mind of the physician, at least.

The **treatment** of simple, benign anemia is an expectant one in ost instances. Hematinics (iron, arsenic, etc.) are seldom required, they have little if any influence upon the blood or upon the pallor or ther symptoms. A rigid system of hygiene, together with attention proper food and drink and to the manner of eating and drinking, ill probably ensure to the patient all the benefit that may be obtained. ardiac tonics (digitalis, strophanthus) may be useful in controlling the alpitation. It is worse than futile to attempt to eradicate any conenital defect of the blood-vessel system or hematopoietic organs.

CHLOROSIS.

(*Green Sickness.*)

Definition.—A blood-disease, occurring chiefly in adolescent females, dependent upon defective hemogenesis, and characterized principally by a deficiency of hemoglobin in the red corpuscles. It runs a mild course, though with a tendency to relapse.

Pathology.—It is so seldom that death occurs in cases of chlorosis that autopsies of this disease have not been frequent enough to determine definitely the nature of the findings. There is no loss of fat in the body, but signs of physical degeneration and disorders of development are quite common, hypoplasia of the vascular system and of the genital organs seeming to be the most prominent. Incurable cases of chlorosis are nearly always characterized by anomalies of the blood-vessels and genitalia (Rokitansky). Virchow has also shown that congenital arrest of development of the aorta and larger arteries, as indicated by their small size, their soft and elastic walls, is quite constant in chlorotics. The uterus (especially) and adnexa manifest the hypoplasia, and yellowish spots and streaks of fatty degeneration are sometimes seen in the intima of the arteries. The cardiac muscle is softened, the whole heart is dilated, and the left ventricle is usually somewhat hypertrophied.

Etiology.—Chlorosis occurs most frequently in girls at or near puberty, and also may appear between that period and twenty or twenty-five years of age. It usually happens that the condition dates from a scanty menstruation, beginning late in the "teens," but it should be recollected that amenorrhea is not, as formerly supposed, a cause, being rather an effect of the underlying blood-disorder. Blondes are oftener affected than brunettes. In males the disease is rare, though cases may develop at puberty or during adolescence.

The influence of *heredity* in the causation of chlorosis is undoubted in those cases described by Virchow, in which congenital hypoplasia of the blood-vessels and genitalia is found to exist. Other cases also bear the stamp of heredity, in that their mothers have been, and their sisters are, chlorotic. A *family tuberculous taint* may predispose to chlorosis (Jolly); it is probable, however, that constitutional predisposition implies merely delicacy of organization. Such *unhygienic conditions* as bad air, dimly lighted rooms, a lack of nutritious food and out-door exercise, a sedentary occupation, hasty and irregular eating, excessive tea- and coffee-drinking, irregular and insufficient hours of rest and sleep; bodily fatigue, as from stair-climbing and standing in constrained positions without intervals of rest—all these predispose to the disease. And yet girls living amid the most luxurious and favorable surroundings have had chlorosis. It is probable that, as the late Sir Andrew Clarke believed, *copremia*—the absorption of the toxic ptomains and leucomains from the colon in constipation—is often the cause of chlorosis, though physiologic chemists fail to find in the urine the evidences of intestinal putrefaction (*i. e.*, an increase of the aromatic sulphates). Sometimes a previously existing simple constitutional anemia appears to be an underlying cause for an exacerbation of genuine chlorosis. In such instances, however, I believe additional exciting causes to be operative.

Sudden emotional excitement and prolonged mental over-exertion operate as causative agencies. Shock from bad news, such as loss of relatives, home-sickness, disappointment in love, rankling grievances, and perhaps ungratified sexual desires, may contribute to the "neuropathic" origin of chlorosis. A change of climate seems to operate as a cause, and is manifested especially in the case of girls emigrating from Ireland to enter domestic service here (Townsend). A *late chlorosis* has also been described, but its existence is doubtful.

Symptoms.—A brief outline of the more frequent and prominent general manifestations of chlorosis—or "green sickness"—may be narrated at the outset. The gradual onset is usually marked by languor, indisposition to either physical or mental exertion, motor weakness, irritability or inertia of mind, depression of energy, and a more or less constant fatigue. *Palpitation of the heart* and *dyspnea* on slight exertion are much complained of in most cases; *headache* is also an early symptom, and may be accompanied by vertigo in some cases; and *dyspepsia* and *constipation* occur in 65 per cent. of cases (Townsend). Probably in one-half of all cases cessation of, or scanty and irregular, menses may form the burden of complaint.

Gastro-intestinal Symptoms.—The appetite is either poor or perverted, and a capricious desire for such innutritious substances as chalk, slate-pencils, and even bits of earth (*pica*), or for sour, highly spiced, and unwholesome articles of food (*malacia*), is not uncommon. Morning vomiting or regurgitation of food and eructations occur, in some cases, pain after eating may be noticed, and dilatation of the stomach and high position of the diaphragm are found in many instances. The tongue is pale, flabby, often dry, and the edges show indentations.

Constipation is usually present, though sometimes *diarrhea*, lasting for a day or two, may alternate, as after the ingestion of some unwholesome article that has been eaten to satisfy the perverted appetite.

General Appearance.—The subcutaneous fat is not only well retained, but in many cases is even increased, and the rotundity of the body and members preserved. The peculiar *greenish-yellow tint* of the complexion is, however, the most striking manifestation to the eye. It differs thus from the muddy pallor of cancerous anemia, from the lemon-yellow tint of pernicious anemia, from the saffron hue of jaundice, and from the blanched pallor after severe hemorrhages. The *scleræ* are often pearly- or bluish-white ("cerulean hue"), and, though this is considered by many the earliest positive indication of anemia, when the skin-tint is not characteristic, yet, according to Townsend's analysis of 87 ses of chlorosis, it is not the most constant. The *nails* showed pallor 95 per cent. of the cases; the cheeks, tongue, and lips were paled in 9, 84, and 76 per cent. respectively, while the scleræ were pale in but per cent. On exertion the cheeks and lips may become quite ruddy cases of moderate anemia (*chlorosis rubra*).

Circulatory symptoms are breathlessness, palpitation, and the tendency vertigo and syncope complained of in the majority of cases; other rculatory disturbances may occur. The *skin* and the extremities are equently cold, owing to sluggish heart-action. The *pulse* is usually ull and easily compressible, and, owing to its excitability, it may be ccelerated for the time being by various external influences (see Fig. 1). Visible undulating pulsations of the carotid vessels are frequent, and a pulsation at the base of the heart and in the peripheral veins is also observed at times. Physical examination shows the *heart* to be slightly dilated. Systolic murmurs, soft and "whiffing" in character, are heard at the base, though in severe cases they may be heard at the apex of the heart also. *Systolic blowing murmurs* of hemic origin are not infrequently heard over the carotid arteries. More common and characteristic, however, is the *venous hum* or *bruit de diable*—the soft continuous murmur heard over the large cervical veins. Thrombosis of the larger veins or of a cranial sinus may occur, and is always ominous.

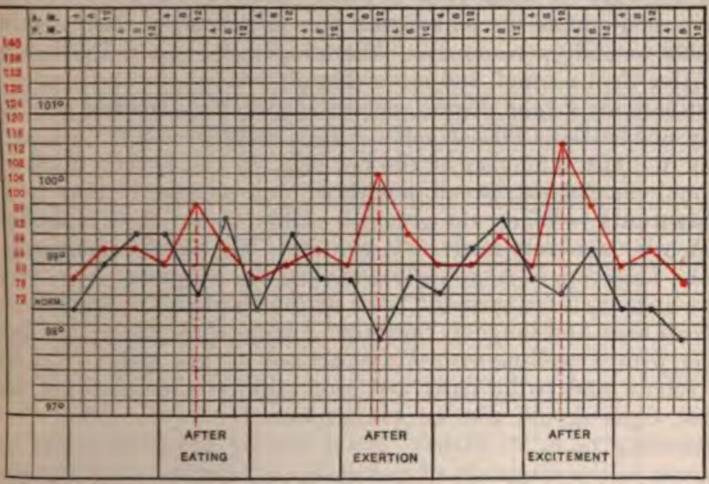

FIG. 31.—Pulse- and temperature-chart of a case of chlorosis, showing the effect exerted upon the pulse by eating, exertion, and excitement.

Of the *nervous manifestations* that are often present, neuralgias of the head, mental depression, hyperesthesia of the skin, particularly of the abdomen, gastralgic attacks, and hysteria, are most frequently met with. Tinnitus aurium and anemic amaurosis have been known to occur.

Edema of the ankles is found in perhaps one-third of the cases. The *urine* is generally pale, free in quantity, and its specific gravity is somewhat lowered; and according to recent studies there is a diminished excretion of urea, despite the abnormal destruction of albuminoids.

Blood-examination.—The blood flowing from a punctured finger-pulp or ear-lobule is pale, though seldom thin or hydremic, and the paleness is due to a qualitative rather than a quantitative change. There is a disproportionate reduction of the *hemoglobin* as compared with the number of the red cells. The hemoglobin may range from 50 per cent. to as low as 16 or 17 per cent. in severe cases, the average quantity being about 38 or 40 per cent. On the other hand, the number of red corpuscles is not greatly reduced, and may even be normal. The moderate *oligocythemia* and *marked oligochromemia* are almost distinctive of chlorosis: these features, however, may be closely simulated by the chloroanemia of syphilis or early tuberculosis. The average number of red corpusles is from 3,700,000 to 4,100,000 per cubic millimeter of blood, but the count in very severe cases may be as low as 1,900,000. Approximately, the number of red corpuscles is from 70 to 85 per cent. of the normal, while the leukocytes are only slightly increased in number (8000 to 8500 per c.mm.). *Microscopically*, the red cells are seen to be paler than normal, and somewhat altered in size and shape. Some are distinctively larger than is usual (macrocytes), but the majority are slightly undersized (microcytes). Irregularity in shape (pokilocytosis) is seen in quite a number of the red cells in the severe cases, and an occasional normoblast (small nucleated red corpuscle) may be noted.

Diagnosis.—When the greenish pallor of the face is marked this can often be correctly made at a glance. The blood-examination must be made, however, to completely establish the diagnosis, even when distinctive symptoms are present, such as the shortness of breath, palpitation, weakness and languor, faintness, amenorrhea, capricious appetite, together with a well-nourished appearance of the body. The bluish-white scleræ and pallid nails are confirmatory when observed, and the physical signs should also be sought for.

According to F. P. Henry, the following blood-variations may be considered in the diagnosis of chlorosis: (1) the red corpuscles may be normal in number and in size, the only change being a deficiency of the hemoglobin; (2) the corpuscles may be normal in number, but diminished in size, while the percentage of hemoglobin is normal; (3) the corpuscles may be diminished in number, with either a diminished, normal, or perhaps an increased percentage of hemoglobin.

Differential Diagnosis.—The primary character of the anemia may be determined in doubtful cases, or in those in which *incipient tuberculosis* ("chloro-anemia"), or *syphilis*, or *Bright's disease* may be suspected, by exclusion. Here the physical examination of the chest, the history, and urinalysis should supplement the blood-examination. In the chloro-anemia of chronic phthisis fever and progressive emaciation are also observed. *Organic disease of the heart* may be simulated by the breathlessness, palpitation, vertigo, and edema.

Prognosis.—This is always favorable, except in those cases in which congenital or developmental anomalies of the vascular system are associated. The discontinuance of proper treatment before a substantial cure is effected is often followed by a relapse, and even after apparent cure one or more recurrences may be witnessed before the age of thirty. The average duration of a case of chlorosis is from two to three months. In cases of very severe type, in which the dividing-line between this disease and pernicious anemia may not be marked clearly, the prognosis should be made with due reserve.

Treatment.—While the treatment of chlorosis by the administration of iron is wellnigh specific, the *hygienic measures* are also important, and particularly in order that relapses may be avoided.

Hygienic.—Pure air, wholesome food, and plenty of rest and sleep, with regular habits, are prime requisites. Sometimes a change of occupation, even temporary, where confinement may be replaced by an outdoor life and sunshine, as in the case of store-girls and mill-operatives, is of great value in bringing about a rapid improvement. Patients in better circumstances may be sent to rural districts, the mountains, or sea-shore. In cases marked by much palpitation, dizziness, and dyspnea, rest in bed for a week or so is often imperative at the outset. As improvement goes on, however, light and then moderate exercise may be permitted out of doors, and the increasing appetite should be gratified by a generous, easily assimilable diet (milk, meat, eggs, fish, purées of green vegetables, stewed fruit). Fats and carbohydrates should generally be avoided. Coffee, tea, and alcoholics do harm. Hot baths continued for fifteen or twenty minutes, thrice weekly, have been recommended.

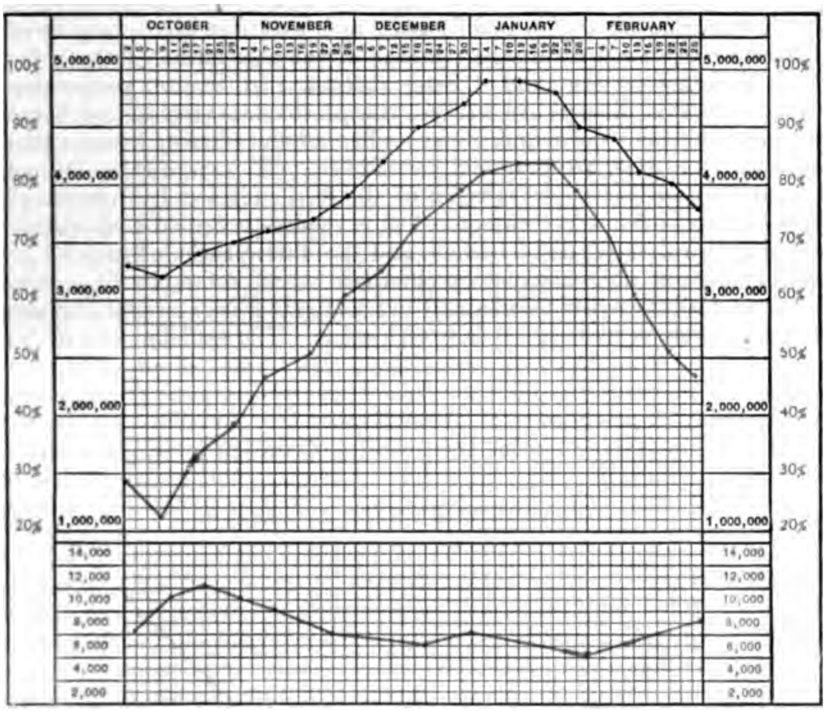

FIG. 32.—Chart of a case of chlorosis, showing the improvement following the administration of iron. Convalescence almost complete; relapse. Black, red corpuscles; red, hemoglobin; blue, white corpuscles.

Medicinal.—The one remedy, *par excellence*, on both rational and empirical grounds, is a good preparation of *iron*. This should be given methodically and persistently until the percentage of hemoglobin is 90, and then maintained there by continuing the administration of the iron for several weeks to prevent a recurrence (Fig. 32). Exactly how the iron acts in curing chlorosis has not been definitely proved, but its almost specific action is indubitable. Not all preparations of iron are equally well borne by the stomach, however, and several changes may be necessary during the course of a given case. Probably the best form for general use is the dried sulphate, usually given together with potassium carbonate in the well-known Blaud's pills—2 grains (0.129) of each to the pill. Starting with one pill thrice daily for a week or ten days, the daily dosage is increased until nine pills daily are administered in the third week, and continued for several weeks or as long as the case may require. It is very important, meanwhile, that the bowels should be kept soluble by the use of cascara sagrada, salines, and the like. A preliminary course of intestinal antiseptics for a week or so is strongly advised by some authorities, and is worthy of recommendation. Beta-naphtol, thymol, guaiacol, and salol are used for this purpose. The hematinic effect of the iron seems to be produced earlier and better when this plan is followed; and this fact seems to give corroborative evidence to Bunge's theory of the absorption of the iron in chlorosis—in a certain class of cases at least. Other iron preparations of value in this disease are the citrate, protoxalate, lactate, carbonate, the succinate, and the reduced iron. The albuminates of iron, so much vaunted for a time, are practically worthless. In severe cases Quincke uses at first a 5 per cent. solution of the ferric citrate, hypodermically (♏vijss–ʒijss—0.5–10.0, daily). The preparation known as ferratin is also highly recommended by some, and the therapeutic efficacy of glycerin extract of bone-marrow in chlorosis is as yet doubtful. Bitter tonics and dilute hydrochloric acid are indicated in a certain number of cases in which indigestion is troublesome. The acid tincture of iron chlorid is sometimes used in such cases. Mild cases often yield to the simple use of remedies for the cure of gastro-intestinal derangement. Adjuvants in the treatment of chlorosis that may be of use are arsenic, manganese, mercuric chlorid, and arsenite of copper in minute doses.

PROGRESSIVE PERNICIOUS ANEMIA.

(*Idiopathic Anemia; Biermier's Anemia.*)

Definition.—A grave blood-disease characterized by a great destruction of red corpuscles, and a persistent tendency from a bad to a worse condition. It usually ends in death, and seldom exhibits causal lesions other than those of the blood or blood-making organs.

The term "idiopathic anemia" applied to this disease by Addison, whose first clear description of its clinical history has become classical, is applicable to a proportionately smaller number of cases to-day than during his time. This is owing to the later discovery (*post-mortem*) of adequate causes for the pernicious anemia that during life could not be found. Thus, while Biermier's anemia is usually considered a special disease-entity, for descriptive purposes it will be convenient to classify both groups under the title of *progressive pernicious anemia* in order to describe the invariable tendency of both. Under Diagnosis (*vide infra*), however, will be found differential clinical features.

Pathology.—The subcutaneous fat is rarely diminished, so that emaciation is exceptional. The skin is pale and of a lemon-yellow tint, and most of the tissues and organs are anemic, except the muscles, which are often decidedly red in color. The fat is usually pale and yellowish, and fatty degeneration is one of the most striking changes in this affection. The heart is usually large and flabby, and on section of the ventricular walls there is a marked pallor, as well as a friability, and a fatty change shown by the yellow tint. Microscopically, the fibers or columns of heart-muscle are seen to be distinctly fatty. The heart-cavities contain little light-colored blood. Other organs showing the fatty degeneration (of the epithelium) are the liver, kidneys, gastric and intestinal walls, and the intima of many of the smaller blood-vessels (in patches). This general fatty change is probably due to the deficient oxygenation of the tissues and to the anemic blood-supply.

Owing to the above degenerative change, and consequent weakening in the vessel-walls, small extravasations of blood are found in different parts. Most frequently these punctiform hemorrhages are seen in the retina and on serous membranes, as on the inner surface of the dura mater, the pericardium, and the pleura. Ecchymoses are also observed occasionally on the mucous membranes and on the skin. More or less general edema and dropsical accumulations in the serous cavities are not uncommon. The spleen and liver are seldom and only very slightly enlarged. The lymph-glands are often somewhat swollen and intensely red in color, owing to the unusual number of red corpuscles.

A marked and important pathologic feature of pernicious anemia is the presence of abundant deposits of iron-pigment, especially in the liver, but also in the spleen, kidneys, pancreas, and other organs. The fact that the abnormal quantity of iron in the liver is peculiarly distributed about the periphery and middle zone of the lobules is particularly noteworthy, and quite characteristic of pernicious anemia. The origin of this iron is doubtless the enormous destruction of red corpuscles, and that the pigment in the hepatic lobules is ferruginous may be determined by a micro-chemic test with ammonium sulphid, granules of black sulphid of iron being formed.

Of special interest are the lesions found in the bone-marrow on account of its hematopoietic function. This is virtually hypertrophied, and is in many cases deep-red instead of yellow, and more like the hemoblastic marrow of childhood (H. C. Wood). While formerly held to be causative, this change is now regarded as being secondary to the severe anemia. Cellular hyperplasia may be seen microscopically in the great number of large and small granular medullary cells, and also in the nucleated red cells.

An atrophied condition of the gastric and duodenal mucosa is noticed in some cases. The sympathetic ganglion cells may also show changes. More constant, however, is the sclerosis of the posterior columns and, to some extent, of the lateral columns of the spinal cord: this is especially marked, according to Burr, in the cervical swelling. Patveu examined 9 cases; in 4 he found hyaline degeneration of the vessels of the white substance, and in 5 small hemorrhages. These changes are probably due to a toxic agent.

Etiology.—There are three etiologic categories into which cases of pernicious anemia may be grouped: (1) those cases in which no discoverable cause for the hemolysis (blood-destruction) is ascertained, either during life or after death—*i. e.* the idiopathic variety of Addison; (2) those in which an adequate cause is found *post-mortem* only; (3) those that are plainly traceable, *ante mortem*, to some primary causal condition acting directly or indirectly.

(1) As regards the *obscure* (*genuine*) *cases* of idiopathic anemia, the essential cause of the symptomatic condition is evidently an actively increased *hemolysis*. The blood-generation (hemogenesis) may be normal in power, or there may be a congenital or acquired underlying deficiency in hemogenic power. Stengel believes that the hemolysis originates in the gastro-intestinal capillaries, and depends upon poisons generated or absorbed from that tract—an auto-intoxication. William Hunter[1] concludes that the disease is of infectious (streptococcal) nature, dependent primarily upon caries of the teeth.

(2) *Apparently causeless* cases of a pernicious type of anemia may be found *post-mortem* to have been caused by (*a*) obscure malignant disease; (*b*) parasites, especially the *Anchylostoma duodenalis*, and rarely by the *Bothriocephalus*. Not infrequently, by a careful study of the anamnesis of a patient, aided by modern methods of examination, the cause of pernicious anemia may be detected during life. Atrophy of the stomach and chronic gastritis, with polypoid growths of the mucosa, were formerly included in this category. It is now held that this atrophy of ventricular and intestinal glands is an effect rather than the cause of the anemia (Grawitz).

(3) Certain exhausting causes, operating directly or indirectly, may precede this affection, as severe or prolonged hemorrhages, or diarrhea, fevers, mental shock, profound chlorosis, pregnancy, and parturition.

Predisposing Causes—Unfavorable hygienic surroundings and insufficient nourishment, habitually kept up, may also favor the development of the disease; but, as in chlorosis, the most favorable environment is not by any means preventive of its development. Males are more frequently affected than females, and especially does it occur during middle life. Griffith has collected several cases occurring under twelve years of age. The disease is widely distributed, and it has been observed to behave almost endemically at times, as in Switzerland and Leipsic. Changes left in the tissues (bones ?) after syphilis may be the pathologic basis, and osteosarcoma may act similarly.

Symptoms.—Idiopathic pernicious anemia develops so slowly and insidiously that it is hardly ever possible to fix upon any precise date as the commencement of the disease. The transition from health to progressive pernicious anemia, particularly in persons previously feeble and pale, is usually too gradual to be demonstrable; though a rapid and acute onset is rare, it may occur in pregnant or puerperal women.

Pallor is soon noticed and gradually increases, or when there has been a previous pallor, this becomes more marked. *Shortness of breath* and *palpitation of the heart*, especially on exertion, are complained of; the patient is also easily fatigued, and becomes quite languid. Occaional nausea may come on early in those cases in which a previous astro-intestinal disturbance has been noted, and headache, vertigo, tinnitus aurium, and anorexia ensue and grow progressively worse. General weakness increases, and occasional attacks of faintness and vomiting supervene. Meanwhile, the skin takes on a bloodless, waxy appearance, and soon the characteristic *lemon-yellow tint* appears. The mucous membranes (lips, gums) are likewise pale and colorless. *Prostration in* *ed* gradually becomes almost absolute as the feebleness and flabbiness of the tissue increase. *Malleolar edema* is sometimes noticeable, and ecchymoses—mucous and cutaneous—are seen in profound cases of anemia. Although the intellect is not impaired, except that mental exertion becomes irksome, the tone and manner of speech are feeble. As the debility becomes severe the mind wanders, and, to use Addison's words, the patient "falls into a prostrate and half-torpid state, and at length expires."

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[1] *Lancet*, Jan. 27, 1900

Emaciation is rare, the fat being preserved and sometimes increased in quantity. Pulsation in the large arteries is abnormally visible, and a diffuse, exaggerated cardiac impulse is felt. The *pulse* early in the case may be strong, and generally it is rapid (100–120), soft, and compressible, and as full and quick, often, as the water-hammer pulse of aortic regurgitation. Auscultation reveals the constant and characteristic *hemic murmurs*, best heard at the base of the heart, and the *bruit de diable* in the veins of the neck. There may also be visible pulsations in the latter.

Gastro-intestinal symptoms may be the most prominent signs in cases where gastritis polyposa and gastritis atrophica are causal. Diarrhea, dyspepsia, nausea, and vomiting are then present throughout the long course; otherwise, constipation, eructations, and simple anorexia are most common.

An ophthalmoscopic examination shows the cause of the *anemic amaurosis*, in the profound cases of anemia, to be one or more retinal hemorrhages. The whites of the eyes become pearly, the conjunctivæ pale. The liver and spleen are rarely palpable. The bones, and especially the sternum, are sometimes sensitive to pressure.

Respiratory Symptoms.—The breathing is accelerated, and the *anemic* dyspnea may become very pronounced and stertorous, accompanied by a sense of oppression in the chest and a "hunger for air." Near the end pleural and pericardial serous effusions and pulmonary edema tend to appear.

The *urine* is of low specific gravity, and, on account of its pigmentation with pathologic urobilin, dark in color. The urobilin is detected both by chemic and spectroscopic examination. In the former the addition of a few drops of an alcoholic solution of zinc chlorid to the urine gives a green fluorescence. The presence of *indican* in the urine points to albuminous decomposition in the intestines. Albumin and glucose are absent, but uric acid and urea are both increased in amount, the former occasionally and the latter usually.

Fever of a moderate degree is commonly, though not invariably, present, the evening temperature sometimes reaching 102° F. (38.8° C.). Previous to death the temperature may be subnormal.

Nervous Symptoms.—Paresthesia, spastic paralysis of the limbs, and a loss of control of the sphincters indicate the paralytic tendency of those cases in which sclerosis of the cord occurs. Tabetic symptoms are sometimes marked.

Blood-examination.—The blood is usually pale, though sometimes dark and watery, and the oligocythemia is distinctive of pernicious anemia. The number of red corpuscles may be reduced to less than 200,000 per c.mm., and is seldom more than 1,000,000. There is ordinarily no increase in the number of leukocytes; on the contrary, they may be somewhat diminished. This diminution usually affects the polynuclear cells most. The percentage of hemoglobin may be approximately proportionate to the number of red corpuscles, but more often it is relatively increased, so that the individual corpuscles are rich in hemoglobin. In other words, although there is a reduction in the total amount of hemoglobin, it is usually not so great as the reduction in the number of erythrocytes; therefore, the color index is nearly always relatively higher than that of the red globules (see Fig. 33), a condition in marked contrast with chlorosis. Macrocytes (which cause the relatively higher percentage of hemoglobin), microcytes, poikilocytes, and polychromatophilia are constantly present, and the former abundant. The presence of nucleated red corpuscles is also a striking characteristic of pernicious anemia. When normal in size they are known as *normoblasts;* when very large, as *gigantoblasts*. In the former, according to Ehrlich, the eccentrically-placed nuclei stain deeply; in the latter the large nuclei stain faintly. The former are typical of those nucleated red globules found in the hematopoietic organ of adults; the latter, of those found in the blood-development of embryonic life. The gigantoblasts are numerous in this disease. There are other and various forms of degeneration of the red cells, but these are of minor import. There may be an increase in the small lymphocytes at the expense of the polynuclear cells; and, according to Cabot, there was always a marked leucopenia, in a series of 110 cases.[1] The blood-plates are generally fewer than normal. The relative proportion of the proteids in the blood-plasma is altered (Adami). Cabot[2] found that a blood-examination made for the

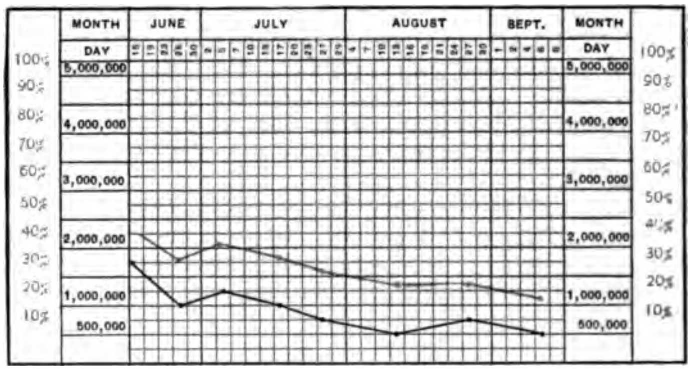

FIG. 33.—Blood-chart of a case of progressive pernicious anemia. Black, red corpuscles; red, hemoglobin.

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[1] Cabot, *Medical News*, May 5, 1900.
[2] *Amer. Jour. Med. Sciences*, Aug., 1900.

first time during the period of remission may give a result similar to that in chlorosis.

Diagnosis and Differential Diagnosis.—The clinical characteristics of the affection, particularly their steady progression with remissions, are quite as important as microscopic study of the blood. The possibility of hidden carcinoma, gastric atrophy, the anchylostoma or other parasite, and incipient tuberculosis should be borne in mind also. *Intestinal parasites* may be inferred from the microscopical examination of the feces after a brisk purge if the eggs of the parasites or the parasites themselves be found. *Atrophic gastritis* may be discriminated by examining the viscus and gastric juice by modern methods. The following table will permit the elimination of obscure gastric carcinoma *as a rule:*

PROGRESSIVE PERNICIOUS ANEMIA.	OBSCURE GASTRIC CARCINOMA.
The blood shows characteristic changes, and the red corpuscle count falls to or below 1,000,000 per c.mm.	Blood shows characteristics of secondary anemia, and the count does not fall to 1,000,000, as a rule.
Found earlier in life.	Occurs after middle life.
Gastric symptoms not so prominent.	Gastric symptoms more suggestive.
Lemon-tinted skin common.	Skin of a pale, muddy-color, or only slightly jaundiced (saffron-yellow).
Adipose tissue fairly well preserved.	Progressive emaciation.
No glandular enlargements palpable.	Supraclavicular or inguinal glands may be palpable.
No physical signs over stomach.	There may be an area of increased resistance over the stomach.
Examination of gastric contents after test-meal usually negative.	Examination of gastric contents shows deficiency or absence of free hydrochloric acid and presence of lactic acid.
Some improvement may be brought about —even cure, though very rarely.	Condition becomes steadily worse until death ends the case.

From *chlorosis* the affection may be differentiated easily by the blood-examination. The relative increase in hemoglobin, the presence of gigantoblasts and many macrocytes, and the severe oligocythemia are pathognomonic of pernicious anemia, and are in marked contrast to the oligochromemia, and slight, if any, reduction in the number of red globules of chlorosis. Again, the progressive pernicious character of the former and the tendency to hemorrhage should be remembered, as well as the contrasting factors of age and sex in the two affections.

Prognosis.—The disease, as a rule, terminates fatally, though not so frequently now as at one time, for obvious reasons. The course of pernicious anemia is usually slow and gradual, and may be interrupted by improvement or apparent recovery. Recurrences, however, invariably occur. Idiopathic anemia is therefore almost hopeless, although a few apparently substantial recoveries have been reported. The duration of the disease is seldom more than a year, and may not be more than two or three months. The nucleated red corpuscles usually become much more numerous shortly before death (Billings). Death may be caused either by syncope, cerebral hemorrhage, or by slow asthenia.

Treatment.—**Hygienic measures** must be regarded as of signal importance, and rest in bed, together with light nutritious food given at short regular intervals, is indicated first of all. Salt-water baths and gentle and systemic massage when the patient is at absolute rest and is not too weak, are useful adjuvants.

The value of *arsenic* in this disease is, I think, analogous to that of iron in chlorosis. The best action of the drug will be obtained by the administration of gradually ascending doses of Fowler's solution or of arsenous acid. Beginning with four or five drops of the former, three times daily during the first week, and thereafter adding one drop to the dose every day or two up to the point of tolerance, as much as twenty or thirty drops, well diluted, may be taken (see Fig. 34). Evidences of gastro-intestinal irritation should be watched for, and the arsenic either discontinued or the daily dose reduced should they appear. Sometimes it is advisable to use the remedy hypodermically. Arsenous acid is given in pill form, commencing with $\frac{1}{30}$ or $\frac{1}{20}$ gr. (0.0021–0.0032).

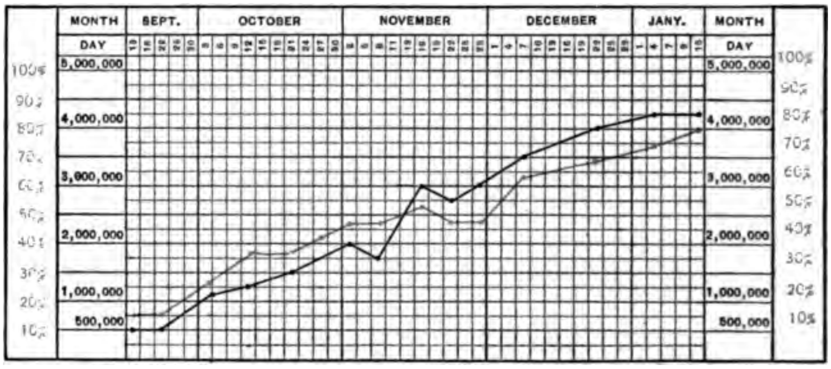

FIG. 34.—Chart of a case of progressive pernicious anemia, showing the improvement following the administration of arsenic. Black, red corpuscles; red, hemoglobin.

The introduction by Fraser of Edinburgh of bone-marrow in the treatment of pernicious anemia has been followed by various results: some cases have been reported in Great Britain and in the United States in which it has seemed to do good, while in others it was found to be useless. While the glycerin extract is the preparation generally used, it is not so reliable as the raw red bone-marrow, or that freshly prepared each day by mixing with it an equal quantity of glycerin; an ounce or two may be administered daily. Hunter suggests the use of anti-streptocccus serum coupled with the antiseptic care of the mouth and gastro-intestinal antisepsis.

Near the end of the disease the danger often greatly increases, owing to the marked reduction in the quantity of the blood (*oligemia*). This may be combated by the injection of warm water or a weak saline solution into the colon (enteroclysis) and also into the subcutaneous tissue (hypodermoclysis). Both the former procedure and gastric lavage are of value in ameliorating the gastro-intestinal disturbance from fermentation. *Intestinal antiseptics* (thymol, guaiacol carbonate, salol, betanapthol, and hydro-naphtol) should be given by the mouth in conjunction with the injections and lavage.

Anthelmintics must be used in those cases of pernicious anemia in which intestinal parasites are associated. Dilute hydrochloric acid and bitter tonics are serviceable in cases in which digestion is impaired.

During the convalescence in favorable cases iron seems to be peculiarly valuable, sometimes alone and frequently in conjunction with arsenic. Thus, arsenious acid and either the carbonate of iron or reduced iron may be combined in pill form, or Fowler's solution and the tincture of the chlorid of iron, or Blaud's pill, may be used with satisfactory results. Recurrences will yield to the same treatment, if they yield at all, except that the doses may have to be increased according to the tolerance of the individual case.

II. THE SECONDARY ANEMIAS.

The secondary anemias are symptomatic of abnormal processes or of existing disease, whether acute or chronic, and their causes are numerous and various. I have already stated that secondary anemia may occur when the true primary form cannot readily be determined and when the course of the anemia is progressive and pernicious. Furthermore, several possible causes may exist in a given case of symptomatic anemia, and it may be quite difficult to discover which of these is the active factor in the condition.

In certain secondary anemias, also, the associated impairment of the blood-making organs is so evident that the anemia may assume almost a primary importance. This was exemplified in Strümpell's case of carcinoma and anemia, with secondary implication of the bone-marrow. The variety and uncertainty of the causes of secondary anemias thus prevent a satisfactory classification.

The Blood.—In most cases this distinctly differs in character from the blood of the primary or essential anemias. There is *oligocythemia*, usually of a moderate degree, about 3,000,000 red corpuscles per cubic millimeter being noted, though in cases of severe hemorrhage the reduction may be as great for a time as in pernicious anemia. There is also a relative decrease in the amount of *hemoglobin*, and sometimes the percentage may be relatively lower even than is compatible with the decrease in the number of the red corpuscles. There is a relative, and often an absolute, increase in the number of *leukocytes* (*vide* Fig. 35). Either a few or many poikilocytes, a few macrocytes, microcytes, and nucleated red cells, some showing fragmented nuclei, are found. Free nuclei may occur. Gigantoblasts are not seen, and the relative increase in the percentage of hemoglobin is also absent.

The most important etiologic groups of secondary anemias are as follows: (1) **Hemorrhage.**—Hemorrhages occur under a great variety of circumstances, and if copious result in an acute secondary anemia. Thus there may be the rupture of an aneurysm, menorrhagia, postpartum hemorrhage, hemoptysis, gastrorrhagia, enterorrhagia, etc., all of which produce the same general effect upon the system. Repeated small hemorrhages may finally produce the same result as a single large one, and spontaneous hemorrhages or epistaxes, such as occur in persons of a hemorrhagic diathesis (hemophilia) or in purpura and scurvy, may cause profound secondary anemia. Females are most tolerant of losses of blood, but infants of both sexes bear depletion very badly. The total mass of blood may be much diminished (oligemia), and the sudden loss of a great volume of blood may prove fatal in a few moments; but it is often surprising how recovery can take place, and often does, after the rapid loss of several pounds of blood—*e. g.* in hemoptysis, hematemesis, or menorrhagia. Sometimes the source of bleeding is obscure, as in cases of intestinal parasites, hepatic cirrhosis, or duodenal ulcer; or it may be intentionally kept *sub rosa* by females having uterine disorder or bleeding hemorrhoids. The quick blanching of the countenance, the weakness, the coldness of the skin, faintness, dimness of vision, tinnitus aurium, sighing respiration, and feeble, rapid pulse are characteristic symptoms of *acute anemia.* *Unconsciousness* and *epileptiform*

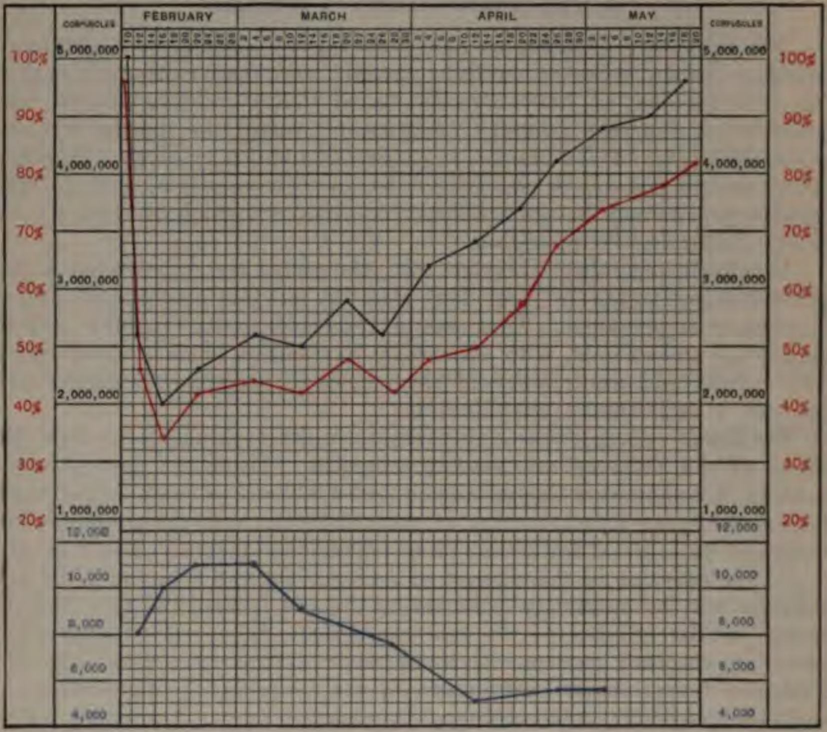

Fig. 35.—Blood-chart of a case of symptomatic anemia. Black, red corpuscles; red, hemoglobin; blue, white corpuscles.

convulsions precede death in cases in which the total volume of blood lost is sufficiently large. When *recovery* takes place the blood-regeneration goes on rapidly, so that within from one to three weeks restitution is complete. The normal volume is soon restored—first by the absorption of water, hydremia existing for several days before the saline and albuminous elements are renewed. The white corpuscles are earlier restored than the red, so that there is a temporary relative leukocytosis. The hemoglobin is restored still more slowly than the red corpuscles.

(2) **Inanition.**—Anemia from inanition may be caused by a food-supply that is insufficient either in quantity or quality, or both; or, even with abundant food of sufficient nutritive qualities the digestive power may be so impaired as to cause defective assimilation. Esophageal

carcinoma and chronic gastritis, especially of the atrophic variety, may thus cause anemia from inanition. The reduction of the blood-plasma forms a feature, while the corpuscles may be affected but slightly.

(3) **Excessive albuminous discharges,** as in chronic Bright's disease, prolonged suppuration, long-continued lactation, chronic dysentery, etc., drain the system so that marked anemia may be produced.

(4) **Toxic Agents.**—The poisons may either be organic or inorganic, though toxic anemias are most common from the absorption of lead, arsenic, mercury, and phosphorus. The poisoning is usually chronic, and affects principally the corpuscles. Anemia due to the poisons of acute or chronic infectious diseases is also frequently met with, and may thus be observed after typhoid fever, diphtheria, yellow fever, and inflammatory (articular) rheumatism among the acute diseases, and during chronic malaria, tuberculosis, and syphilis ("syphilitic chlorosis"). There is considerable destruction of the red corpuscles in some of these diseases, either directly or indirectly, and the greater the pyrexia the greater the action upon the blood or blood-making organs.

Symptoms.—The *common indications* of secondary anemia are the pallor of the face and mucosæ, muscular and mental weakness, loss of nerve-function, neuralgias, coolness of the skin, dyspnea on exertion, cardiac palpitation, impaired appetite and digestion, and a weak pulse. The physical signs are those of the primary or essential anemias.

Diagnosis.—Here may be advantageously contrasted the distinguishing features naturally grouping themselves under symptomatic and essential anemias, respectively:

SYMPTOMATIC OR SECONDARY ANEMIA.	IDIOPATHIC OR ESSENTIAL ANEMIA.
A symptomatic blood-condition secondary to disease elsewhere.	A primary disease of the blood and blood-making organs.
Occurs at any age.	Occurs principally during adolescence and early middle life.
Previous or associated history of traumatic or spontaneous hemorrhage, chronic suppuration, prolonged lactation, chronic Bright's disease, carcinoma, chronic lead-poisoning, chronic malaria, etc.	Previous history negative in its bearings upon the disease.
Blood-changes not so marked and more variable; steadily progressive in malignant disease.	Distinctive blood-characteristics, and often profound changes, both as to blood-cells and hemoglobin.
Moderate reduction in both, merely the relative proportion being maintained.	Marked reduction in either the hemoglobin percentage or in the number of red corpuscles.
General symptoms and signs usually subordinate in manifestation to those of the primary disease or lesion.	General symptoms and signs also more characteristic of the respective form of anemia in the case.
Gravity of anemia depends on that of the primary disease.	Gravity depends on type of blood-changes and progressiveness of disease.
Often responds to treatment, depending on the cause; in a few instances, as in hemorrhage, it is short in duration.	One variety (chlorotic) quite curable, the other (progressive pernicious) fatal.

The **prognosis** depends upon the cause of the anemia.

Treatment.—Symptomatic anemia is amenable to treatment according to the cause. The traumatic acute variety does well under simple hygienic measures after the urgent indications have been met. Plenty of pure air, wholesome food, and graduated rest and exercise may suffice, and drugs not be needed. Cases in which it is difficult or wellnigh impossible to remove the cause of the anemia of course do not improve under any treatment other than that which may favorably influence the primary affection. Nutritious aliment, iron in some form, a judicious hygienic regimen calculated to increase the assimilation, and stomachic and general tonics are required in the majority of cases. Toxic substances must be eliminated, their re-introduction into the body prevented, and the repair of the blood and tissue actively promoted.

LEUKOCYTOSIS.

Definition.—A temporary increase in the number of polymorphonuclear leukocytes in the blood though rarely in the mononuclear elements. Leukocytosis, however, may be continuous. The number of white corpuscles in a moderate leukocytosis would be about 10,000 per cubic millimeter; in marked leukocytosis there might be as many as from 20,000 to 40,000; a count of 50,000 leukocytes to the c.mm. may, however, usually be considered to indicate leukemia. Von Limbeck, notwithstanding, reported a case of leukocytosis accompanying carcinoma of the kidney with metastasis, in which there were 80,000 white corpuscles per cubic millimeter.

Physiologic leukocytosis occurs in infants during the first few days after birth, in pregnancy, during digestion, and after exercise. According to Carter, the "digestion leukocytosis is present after a meal of proteids or hydrocarbons, but not after a meal of carbohydrates."[1] Massage and cold baths also produce leukocytosis, probably by stimulating the circulation, and not by increasing the actual number of leukocytes, some of which have simply become stagnated.

Pathologic leukocytosis is secondary to various affections. It may be temporary, as in the curable primary diseases, or permanent, as in those that do not permit of recovery. It is also found to be well marked in acute inflammations and in infectious febrile diseases accompanied with exudation, such as pneumonia, pertussis, and diphtheria. In pleuritis, peritonitis, pericarditis, erysipelas, and in all suppurative processes there is an excess in the number of polynuclear neutrophiles. Inflammations of the serous membranes, when not tuberculous, causes leukocytosis, so that a purulent meningitis may be differentiated from tuberculous meningitis by the pronounced leukocytosis in the former and its absence in the latter. As a rule, the greater the local reaction and the stronger the resistance to severe infections the greater the leukocytosis. As is well known, the pus-cells of an abscess consist almost wholly of dead white corpuscles—phagocytes—that have been overcome or exhausted, directly or indirectly, in the struggle against the toxin of the infection. *Cachectic states*, as in cases of malignant tumors, are often attended with an increase in the number of mononuclear cells in the blood, especially in the region of the tumor and where the lymph-glands are involved. Leukocytosis may be very marked in carcinoma, nd near the close in any marantic condition. Chemical irritants, uch as turpentine, may also produce leukocytosis, and whatever the ubstance causing the condition it is spoken of as *positively chemotactic* —attractive to the white blood-corpuscles—in contradistinction to *negatively chemotactic substances*, which repel the white corpuscles.

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[1] *Univ. Med. Magazine*, vols. vii and viii, p. 181, Dec., 1894.

In *non-leukocytotic* infectious diseases (typhoid fever, influenza, malaria, and acute miliary tuberculosis), the diagnosis of a complicating pleuritis or peritonitis, for example, may be confirmed by the detection of the leukocytosis. Leukocytosis under such circumstances has *prognostic* importance. Diminishing leukocytosis during the height of a grave disease may be significant of lessening powers of resistance, though this is not an invariable rule, since just before the crisis of a pneumonia a diminution of the leukocytes is apt to occur.

The *object* of the leukocytosis is naturally protective, beneficent, and reparative. It is accomplished either by direct antagonism or by the formation of substances that enter the fluids and tissues of the body, and counteract the influence of the toxic substances causing the disease. The existence of leukocytosis can best be determined by the examination of stained specimens of the blood. Post-digestive leukocytosis is to be discriminated from the *pathologic variety* by making the examination several hours after the last meal has been taken. It has been found that the number of leukocytes in blood taken from the cold finger is less by 50 per cent. than in a count made from the warm finger.

LEUKOCYTHEMIA.

(*True Leukemia.*)

Definition.—A blood-disease, usually chronic, characterized by a peculiarly marked and persistent increase in the number of leukocytes, associated with lesions occurring either respectively or unitedly in the spleen, bone-marrow, and lymphatic glands.

Pathology.—Bodily emaciation and pallor are pronounced, and edema, with dropsical effusions in the serous cavities, is by no means uncommon. The cardiac chambers and principal veins are distended with large blood-clots of a greenish-yellow or, in extreme cases, yellowish-white, purulent appearance. Subserous ecchymoses of the pericardium and endocardium are frequent, and the myocardium is often found to have undergone a moderate degree of fatty degeneration. Various abnormal substances have been found in leukemic blood—leucin, tyrosin, acetic, formic, and lactic acids, and certain albuminous substances (deutero-albumose and nucleo-albumin)—resulting probably from the destruction of blood-corpuscles. The alkalinity of the blood is diminished. The minute, octahedral (Charcot's) crystals are found most abundantly in settled leukemic blood, and have also been detected in the spleen, bone-marrow, and liver, as well as in other affections.

Although the spleen, bone-marrow, or the lymph-glands may alone show the pronounced pathologic changes of leukemia, it is usual to find all more or less affected. Purely splenic or myelogenic leukemia, and the latter especially, are rarer than the lymphatic type, so that it is customary to speak of two principal groups: (1) *splenic-myelogenous* (or *spleno-medullary*) leukemia, the most frequent variety; and (2) lymphatic leukemia.

There is nearly always some splenic enlargement, and in many cases the enlargement is considerable, as in **spleno-medullary leukemia.** Leukemic spleens sometimes weigh as much as from two to eighteen pounds, and their lengths may vary from six to twelve inches. The enlargement is generally uniform, and the notches upon the anterior border may be much exaggerated. White patches of perisplenitis and a thickened capsule adhering to the surrounding organs and the abdominal wall may also be noticed. The consistence of the spleen is firm and resistant to the knife, though in the earlier stages it may be quite soft and pulpy. The cut surface is either of a uniformly brown color or mottled by the presence of grayish- or yellowish-white circumscribed lymphoid tumors, or by deep-red or brownish-yellow hemorrhagic infarcts. The Malpighian bodies may or may not be visible. The blood-vessels at the hilum are enlarged. *Microscopic examination* shows hyperplasia of the organ, although most pathologists regard the change as lymphadenomatous. The cells of the pulp sometimes show granular and fatty degeneration, and in advanced cases the trabeculæ may be thickened by a considerable amount of firm connective tissue.

In the majority of cases the bone-marrow is affected as well as the spleen, and a purely myelogenous leukemia is extremely rare. Indeed, the few reported cases of the latter may be doubted. The medullary substance, instead of being fatty, is rich in lymphoid and blood-cells in various stages of development, and is either reddish-brown or greenish-yellow in color. Neuman regarded the marrow-change as a constant and essential lesion of leukemia, and called the former transformation "lymph-adenoid," and the latter "pyoid." The pus-like marrow and the dark-red may exist side by side, although the former is more common.

A fine reticulum may be seen between the cells, especially in the dark-red variety, and small hemorrhagic infarcts may also be noted occasionally. *Microscopically,* the medulla contains an abundance of lymphoid cells and nucleated red corpuscles. Eosinophilic, mononuclear, and polynuclear leukocytes are also present, the first-named being quite numerous, as are also certain myelo-plaques and cells showing karyokinetic figures. The *lymphatic glands* are more or less enlarged in the splenic and medullary forms of leukemia.

In the **lymphatic variety,** especially when acute, an early and marked hyperplasia of all the glands takes place. The cervical, axillary, inguinal, and mesenteric glands are usually involved, and may form distinct, soft, and movable tumors, their color being a reddish-gray. Rarely, the primary lesions may be in the lymphadenoid tissues of the gastro-intestinal tract.

The histologic examination shows an increase in the cellular elements. A similar hyperplasia occurs in those glandular tissues that are allied to the lymphatic glands, such as the tonsils, lymph-follicles, the tongue, mouth and pharynx, thymus gland, and the solitary and Peyer's agminated intestinal glands.

The *liver* may be greatly enlarged; indeed, some of the instances of greatest enlargement of this organ have been those due to leukemia, the weight being as much as fourteen pounds. The enlargement is uniform and due to a diffuse leukemic infiltration. The capillaries and interlobular tissue are distended with leukocytes, and disseminated whitish or grayish nodules, usually quite small, consisting of lymphoid cells undergoing indirect division of their nuclei, are frequently found. Sometimes these leukemic nodules appear as definite growths, with an adenoid reticulum between the cells, on account of which they have been called lymphomata or lymph-adenomata.

Similar changes are observed in the *kidneys*, enlargement, paleness, and diffuse and circumscribed leukemic infiltration of the capillaries and intertubular tissue all being noted. Leukemic nodules may also be found in other parts of the body, such as the retina, brain, serous membranes, lungs, testicles, and skin. Karyokinetic figures are numerous in the cells accompanying these leukemic growths.

Etiology.—The primary cause of leukemia is unknown; that it directly affects the blood-forming organs, however, is most probable, though with differences of selection and co-ordination and with different degrees of intensity. The combination of lesions in the spleen, lymph-glands, and bone-marrow, along with the histologic similarity of the leukemic growths to the infectious granulomata, and the clinical history of cases of acute leukemia, would seem to point strongly to the *microbic origin* of the disease. Moreover, various cocci and bacilli have been found, but not one of them has been definitely proved to be the specific cause of the disease. *Auto-intoxication* by toxic albuminoids from the digestive tract is believed by Vehsemeyer,[1] who analyzed 600 cases, to be the important point of departure of the disease. It is likely that the direct cause of the leukocythemia is a simple increase of the cytogenic function of one or more of the hematopoietic organs. Kottnitz held leukocythemia to be a reactive condition following auto-intoxication with peptones, and consequently a leukolysis, the over-action of the hematopoietic organs leading to hypertrophy. Whether the reduction of the erythrocytes is due to diminished production or to increased destruction is not positively known, although the former factor is more probably operative.

The disease has often been preceded by an *injury* or *a blow* in the splenic region, but its direct traumatic origin is hypothetic only. *Intestinal ulceration* has been a frequent feature prior to leukemia, and undoubtedly affords a source of possible infection from the tract. *Stomatitis* also may furnish a means of entrance for the infectious agent. The causal relation of *pseudo-leukemia* and *true leukemia* is uncertain, although a few cases of the one have been observed to pass into the other.

In a considerable proportion of cases leukemic patients have had *malaria* of some form. Syphilis may be associated with the disease, but it is not probable that it acts in a causative manner.

Hereditary influences undoubtedly play a part; a "lymphogenous diathesis" may thus be transmitted, and several generations may be affected by the disease. *Adverse hygienic* and *social conditions* may also predispose to leukemia. It may also develop after *pregnancy*, or more commonly at the *climacteric*. Anxiety, worry, and mental depression have been mentioned as predisposing causes, with doubtful justification.

Leukemia occurs most frequently in males during the middle period of life, and is apt to attack young persons. It has occurred during infancy, and as late also as the seventieth year, but the average age ranges from twenty-five to forty-five years. Sometimes the previous condition was one of apparently perfect health.

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[1] *International klin. Rundsch.*, Vienna, Nov. 25, 1894.

Symptoms.—**Acute leukemia**, although comparatively rare, may be described briefly first. It usually occurs in an adolescent who may have enjoyed previous good health. Fussel and Taylor collected 56 cases from the literature. Its onset is sudden, and usually begins with prostration, hemorrhage of the mucous membranes, and high fever. Acute splenic tumor rapidly develops; the lymphatic glands may enlarge; and palpitation, dyspnea, and gastro-intestinal symptoms of a severe type appear. The skin becomes anemic, and edema of the feet is common. The blood shows a marked increase in the number of leukocytes, the ratio to the red corpuscles being 1 to 30 or 1 to 50, instead of the normal 1 to 350 or 1 to 600. In acute lymphatic leukemia the lymphocytes are very numerous. Large mononuclear leukocytes and myelocytes are also numerous, while the eosinophilic cells are few in number compared with those found in the blood of chronic leukemia. The case grows progressively worse; hematemesis, cerebral or retinal hemorrhages, and petechiæ supervene perhaps, and the clinical features may then resemble an infectious disease with hemorrhagic and purpuric manifestations. Death occurs in from two to three months.

In **chronic leukemia** the *onset* is generally slow and insidious and its development imperceptible, and for many months the earlier symptoms may not differ from those of simple anemia. Languor, a deranged appetite, dizziness, noises in the ears, faintness, breathlessness on exertion, and palpitation may all appear. Sometimes, however, not even these symptoms are present, common as they are to most anemic cases, and the patient may first consult the physician, because of a swelling or distress in the left side of the abdomen—the *enlarged spleen*. Early manifestations may be *hemorrhagic* in some cases (epistaxis, hematemesis, enterorrhagia), with nausea, vomiting, and diarrhea; or *increasing pallor* of the countenance, yet at times a patient may appear to be plethoric; or troublesome priapism may be the first indication. As the disease progresses the anemia becomes more marked, *edema* of the dependent portions of the body may appear, and *fever*, though slight at first (99.5° F.—37.5° C.), may gradually rise to 102° or 103° F (39.4° C.), either remaining constant or alternating with periods of apyrexia.

The *pulse-rate* is increased; in quality it is soft and compressible, though sometimes full in volume. The *dyspnea* may be aggravated by the hydrothorax of a general dropsy in advanced cases, or by the upward displacement of the diaphragm owing to the increasing splenic and hepatic enlargement. *Epistaxis* may become obstinate. Retinal hemorrhage is common, and there may be aggregations of leukocytes (leukemic growths). Hemorrhages from mucous membranes are common, and localized gangrene may occur, in which case the symptoms of infection appear. Hemic murmurs are quite constant.

Ulcerative processes in the bowels may give rise to severe *dysenteric diarrhea*. *Ascites* is usually present in advanced cases on account of the splenic tumor, or owing to pressure upon the portal vein by enlarged glands. *Jaundice* is an occasional event. *Leukemic peritonitis* may occur from the presence of lymphomatous growths in the membrane.

Nervous symptoms, such as headache, vertigo, and syncopal attacks, are liable to recur as the anemia and prostration increase and the liability to hemorrhage becomes more frequent. Sudden coma and hemiplegia following upon the rupture of a cerebral vessel (apoplexy) may be the immediate cause of death. Minute brain-hemorrhages, which may occur at any period of the disease, probably account for deafness. Priapism may be very troublesome. Peripheral paralysis of several cranial nerves, due to hemorrhages into their sheaths, has been reported.

Cutaneous ecchymoses are sometimes observed, and sometimes there is a troublesome pruritus. The *urine* contains an excess of uric acid, but albuminuria does not occur, except as a complication.

Along with the anemia and debility are the signs of splenic and lymphatic involvement, and rarely of the bone-marrow. The liver may also become enlarged.

Leading Symptoms in Detail.—*The Spleen.*—This organ is generally enlarged in all forms of leukemia, but especially in the spleno-medullary, the most frequent form. It is a prominent feature, both on account of its being the first subject of complaint, and because of the huge size it frequently attains. The enlargement is gradual, and there may be neither pain nor tenderness over it. The tumor may cause a visible projection below the ribs, and in marked cases great abdominal distention may be produced, pushing up the diaphragm and thoracic organs, and extending to the navel in the median line and to the pelvis below, in which case the cardiac pulsation is seen at the second or third interspace. The edge and notch or notches may be felt easily in such instances, while the surface is smooth and the consistence firm. A friction-fremitus is felt sometimes during respiratory movement. The tumor may vary in size, and after severe hemorrhage or diarrhea it may become swollen. Gastric distress after eating and obstructive constipation are usually complained of in cases of great splenic enlargement. Jaundice may also be present. Pulsation has been noted and a systolic murmur—"splenic souffle"—has been heard at times over the tumor. The percussion-note is dull over the tumor, and areas of movable dulness, due to fluid occupying the peritoneal cavity, are not infrequent. A wave of fluctuation may be detected over the abdomen. The liver is often enlarged.

Lymphatic Glands.—In the splenic-lymphatic variety, which is less common than the splenic-myelogenous, and in the still rarer purely lymphatic leukemia, the superficial lymph-glands may be both visibly and palpably enlarged, though not in bunches as in Hodgkin's disease. They are soft, resilient, and movable.

The Bones.—Purely myelogenous leukemia is very rare, and local bone-symptoms are scarcely ever manifested. There may be some tenderness on immediate percussion over the sternum or some of the long bones, and slight swelling, irregularity, or deformity of the ribs, the sternum, or other bones may result from leukemic hyperplasia.

The Blood.—It is by the blood-examination alone that the pathognomonic features of leukemia are determined. The blood is paler than normal, and sometimes has a brownish-red or chocolate color. Upon a microscopic examination of the blood in the spleno-medullary form of the affection the striking increase in the number of leukocytes is observed at once. The count shows usually from 85,000 to 500,000 white corpuscles per cubic millimeter, and the ratio of the white to the red

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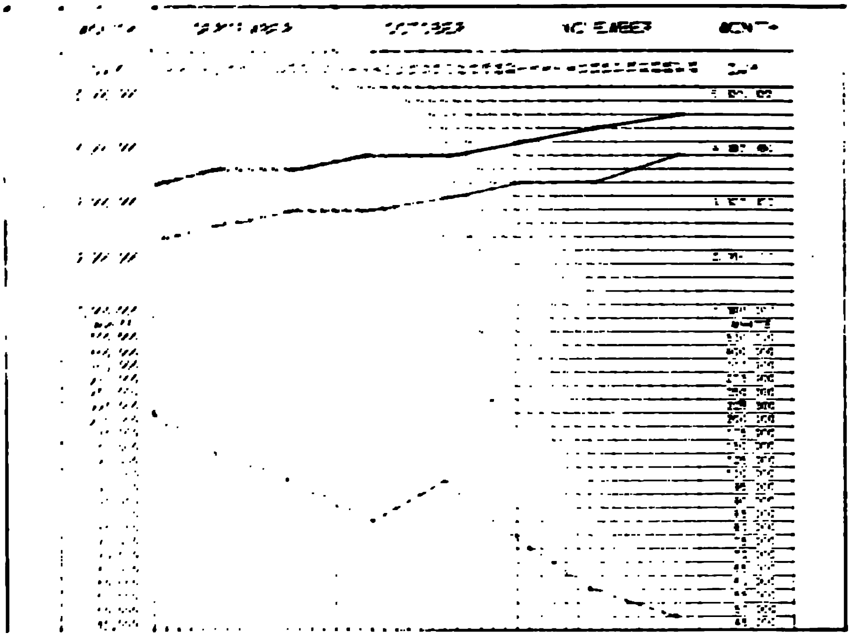

Fig. 6. Blood tracing of a case of leukemia. Black, red corpuscles; red, hemoglobin; blue, white corpuscles.

nuclei of both blood and marrow-cells showing karyokinetic figures. The polymorpho-nuclear leukocytes may be normal in number, but usually they are relatively diminished to about 65 per cent. instead of 75 per cent., as in normal blood. The polymorpho-nuclear cells showing coarse basophilic granules are increased, and may equal in number the eosinophiles. When Ehrlich's triacid stain is used these cells appear as non-granular polynuclear bodies. The lymphocytes are also relatively less in number, making up but 1 or 2 per cent., instead of the normal 15 to 30 per cent. The bright, acid stained eosinophiles, though absolutely increased, are not always relatively so. They possess but little diagnostic value, being common to many other conditions.

Moderate *oligocythemia* is noted, the reduction being seldom lower

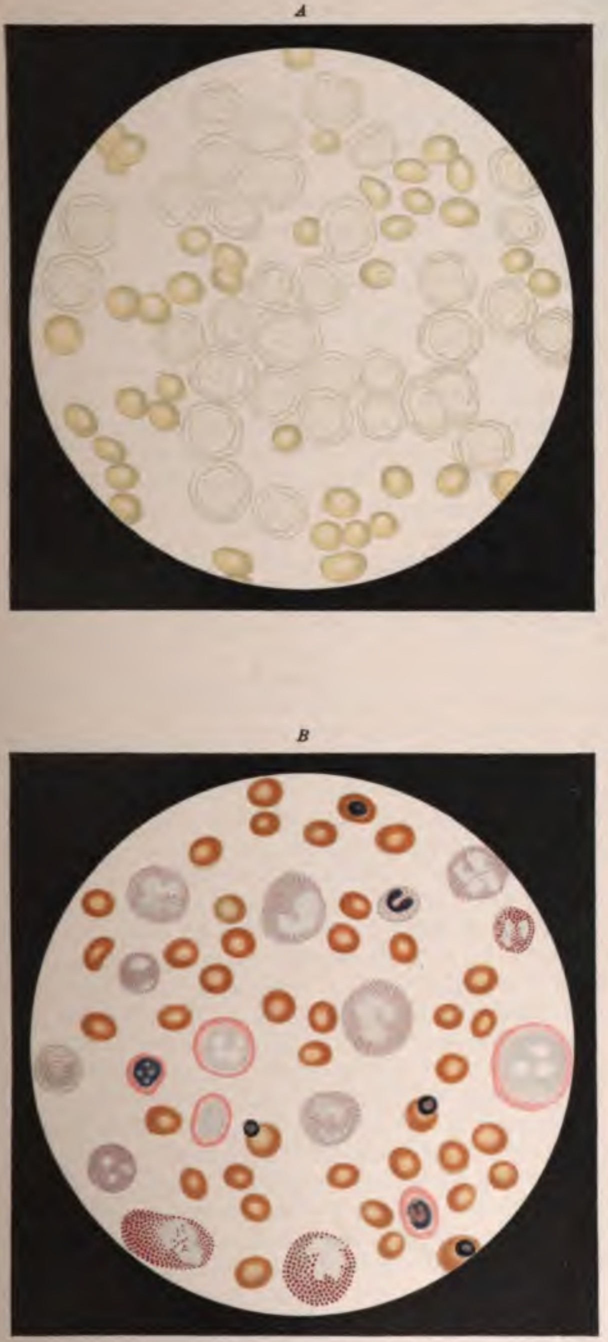

FIG. 37.—*A*. Fresh preparation from the blood of a case of leukemia (× 550); large mononuclear leukocytes of immature form; *B*. Preparation of a case of the lieno-myelogenetic variety (Ehrlich's triple stain); numerous eosinophile and immature leukocytes, myelocytes, and nucleated red blood-cells (Grawitz).

han to 2,000,000 per c.mm. The percentage of hemoglobin may also e reduced relatively or in slightly greater proportion. Nucleated red orpuscles, chiefly normoblasts, are frequently found in considerable numbers. Cells with large, pale nuclei are occasionally found, and cells with fragmented nuclei are common. Gigantoblasts may be present. Osler asserts that blood of the type of pernicious anemia has subsequently developed a true leukemia. Litten and Musser have also described such cases.

In *lymphatic leukemia*, which is rarer and more quickly fatal than the preceding variety, the blood-changes are also different. The *lymphocytes* are the ones increased, all other leukocytes being relatively much diminished in number. Instead of the normal percentage (15 to 30 per cent.), the lymphocytes may number from 90 to 97 per cent. of all the leukocytes. This increase affects the small forms in most cases, however. Cabot has shown that in some instances this increase affects the larger lymphocytes—cells of the size of polynuclear leukocytes. Nucleated red corpuscles are often present in large numbers, but myelocytes occur in small numbers, as a rule. Eosinophiles are rare. *Mixed forms* of leukemia are, however, not at all uncommon, so that the proportions of the various types of normal and abnormal cells are quite variable.

The *blood-plates* may be quite abundant in many leukemic cases, and Charcot's octahedral crystals appear in specimens of the blood if allowed to stand for any length of time. An unusually dense and thick fibrous network is also often found.

Complications.—Fatal hemorrhages may occur at any time, and pulmonary tuberculosis, pleuritis, pneumonia, septico-pyemia, renal disease, severe diarrhea, and edema may complicate leukemia and cause death.

Diagnosis.—This can be made easily and accurately by the blood-examination alone, the distinguishing characteristics of the blood having been enumerated above, both as to the existence of leukemia and the differentiation of its several varieties. It may be necessary in doubtful cases to examine the blood by Ehrlich's staining methods, since the mere excess of leukocytes alone is not proof of leukemia, and also because the disease may exist without an excess, owing either to previous medicinal treatment or to natural temporary improvement.

Differential Diagnosis.—Leukemia is differentiated from a marked *leukocytosis* by the fact that in the latter there is usually a more moderate increase in the number of leukocytes, and this increase, as a rule, is principally of the polynuclear neutrophiles.

Hodgkin's disease may be simulated by the purely lymphatic leukemia on account of the enlarged glands; but in leukemia the lymph-glands are not found in such large bunches, and the blood-examination will show the characteristic changes of lymphatic leukemia if that disease be present. Simply a leukocytosis is present in pseudo-leukemia.

Malignant growths of the spleen and lymphatic glands, and also a malarial and passively congested spleen with anemia, may simulate leukemia. The simple leukocytosis here will exclude leukemia.

Prognosis.—Many cases are mild and gradual in their progress; children, however, when affected, succumb more rapidly than do adults. Lymphatic leukemia is always fatal earlier than the spleno-medullary variety. Although recovery does occur occasionally, most cases of leukemia, of whatever form, prove fatal certainly within five years, generally in two or three years, and sometimes in seven or eight months or even less (from two weeks to two or more months) in acute leukemia. In an advanced case the prognosis is hopeless. It should be borne in mind that apparent improvement is usually only temporary, and that a fresh exacerbation is apt to follow. Grave symptoms heralding an early termination are profound debility, anemia, emaciation or edema, severe and obstinate hemorrhages, cerebral apoplexy, persistent diarrhea, and high fever. Intercurrent affections not infrequently cause death, while, on the other hand, cases are recorded in which the appearance of intercurrent infectious diseases has favorably affected the course of leukemia.

Treatment.—At present no remedies are known to have any permanent curative effect, although several agents are used for their favorable influence. The aim should be to improve the general condition of the patient and endeavor to prolong life by hygienic and medicinal means.

The environment should be made as favorable as possible—physically, mentally, socially, and morally. Out-of-door life in a mild, dry climate, an abundance of nutritious and easily digestible and assimilable food, calm and moderate exercise of mind (depending upon the strength and endurance of the patient), should all be advised and encouraged. On the other hand, traumatism and inflammation, irregular habits of body, worry, excitement, and passionate emotions and appetites should be regulated and avoided.

Arsenic gives the best results in most cases, and should be pushed to the limit of tolerance, as in pernicious anemia. It should be given continuously, regardless of apparent improvement under its use, as the latter may be only the natural remission—a not uncommon incident in the disease. Quinin, iron, and the oil of eucalyptus have been recommended in those leukemic cases in which a clear history of malaria has been obtained. Bone-marrow, either raw and spread upon bread or in the form of a glycerin extract, may be tried when arsenic fails. Oxygen-inhalations and blood-transfusion have been suggested. The so-called "splenic remedies," whether systemic or local, have no controlling influences upon the disease. Electricity may afford some local comfort or contribute to psychic ease. Complications and intercurrent affections may often be greatly relieved by appropriate treatment.

PSEUDO-LEUKEMIA.

(*Hodgkin's Disease; Adenia; General Lymphadenoma; Multiple Malignant Lymphoma; Malignant Lympho-sarcoma.*)

Definition.—An anemic disease characterized by the anatomic peculiarities resembling those of lymphatic leukemia—viz., progressive hyperplasia of the lymph-glands, occasional secondary lymphoid growths of other organs (liver, spleen); and by the absence of the destructive blood-changes of true leukemia.

Varieties.—Although the disease that bears his name was first described by Hodgkin of Guy's Hospital in 1832 as an affection of the lymphatic glands and spleen, two varieties are included under the title pseudo-leukemia (or Hodgkin's disease), as follows: (1) that which esents simply an enlarged spleen (the less frequent one); and (2) that which the lymphatic glands are chiefly involved.

Pathology.—The lymph-glands show different degrees of hyperplas-c enlargement and consistency. In the earlier stages they are small, olated, and movable, while in advanced and well-developed cases of e disease they are larger, fused together into great bunches, and more r less fixed by fibrous investment. As a rule, the glands are soft and lastic, though sometimes they are hard and dense, and masses as large s an orange or pineapple may be seen. Single glands may be as large s a hen's egg, and the gland-capsules may show connective-tissue proiferation and a thickening periadenitis. Extension of the lymphatic growth into the surrounding tissues by perforation of the capsule may ccur. As a rule, the overlying skin is freely movable, though it may rarely be adherent. On section the tumors display a smooth white or reddish-gray surface in the case of the soft and almost fluctuating glands, and a grayish or a yellowish-white color if they are firm. The fusion of the swollen glands into nodular masses is also seen, and when ulceration through the skin has taken place suppuration of the glands may be revealed. In the harder tumors areas of necrosis having the appearance of caseation may be visible, and shining, more or less hyaline masses of fibroid tissue may also be detected.

Microscopically, there is a typical hyperplasia of the lymph-cells, often obscuring completely the reticulum of the gland, except in the harder enlargements, where the fibrous proliferation shows a very distinct network. The change is a lymphadenoma of the lymphatic glands.

The cervical glands are most prominently involved. The superficial chains of glands—axillary, mediastinal, scapular, and pectoral—especially along the great vessels, are often found connected, and the inguinal, bronchial, and lumbar glands are also affected, though less frequently. The retroperitoneal glands are more frequently affected than the mesenteric, and sometimes the thoracic vessels are completely surrounded by enlarged lymph-glands; they have occasionally projected externally by perforation through the sternum. The abdominal vessels, nerves and nerve-plexuses, and ducts may be compressed also by huge groups of enlarged glands.

The *spleen* is enlarged in about four-fifths of the cases, but only slightly. In the majority of cases there are disseminated throughout the organ whitish, lymphomatous growths or nodules from the size of a pea to that of a nut. Their histologic structure is like that of the lymph-glands (lymphadenoma). Occasionally the spleen alone is hyperplastic.

Lymphomata may also develop in the tonsils, lingual follicles, intestinal lymphatics, liver, kidneys, lungs, brain, heart, testicles, retina, and skin. Invasion of the spinal cord may occur by erosion of the vertebræ or through the blood-current by metastasis. The bone-marrow often has the same appearance as in pernicious anemia.

Etiology.—There are no well-established *predisposing conditions* to which Hodgkin's disease is referable. In 75 per cent. of cases males are affected, and young and middle-aged persons—between the ages of ten and forty years. In an analysis of 100 cases 30 were under twenty years, 34 between twenty and forty, and 36 after forty (Gowers). Heredity may possibly be a cause. Neither has an *exciting cause* been discovered as yet. The disease would seem to belong to the group of infectious granulomata, but the infectious agent is not known. Flexner thinks that certain protoplasmic foreign bodies (found in the larger nodules of two cases) may possibly have a causal relation to the disease. Malaria, syphilis, chronic skin-diseases, and various irritative conditions, especially of the mouth, giving rise to local glandular swellings, have also been assigned as causes. In undoubted instances of Hodgkin's disease the lymphatic glands frequently harbor tubercle bacilli; hence it has been thought that the latter exercise a distinct causative influence. It must be remembered, however, that some of these may be examples of secondary accidental infection; others of primary diffuse lymphatic tuberculosis, indistinguishable from or mistaken for Hodgkin's disease. It is not uncommon to find pseudo-leukemia developing in a person who immediately preceding the beginning of the disease was apparently in perfect health.

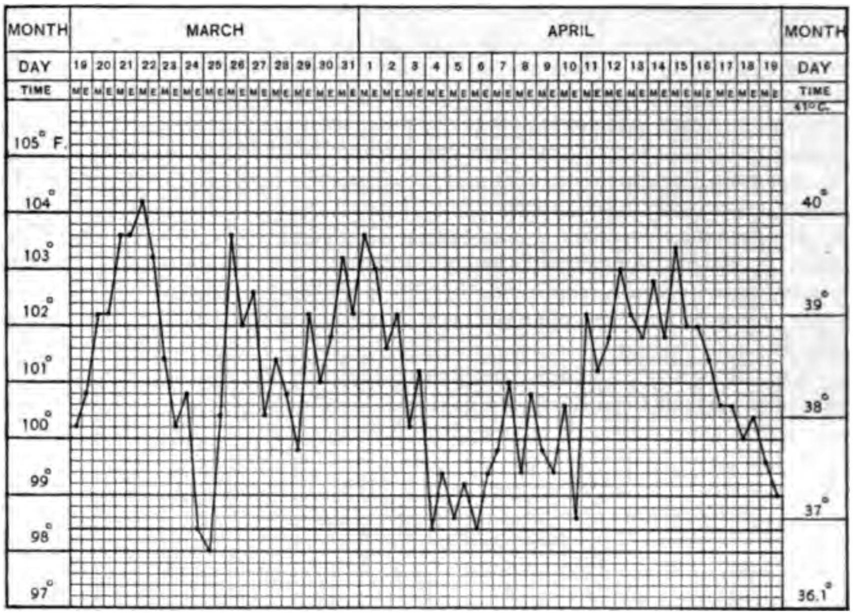

FIG. 38.—Temperature-chart of a case of pseudo-leukemia.

Symptoms.—Usually the first thing to attract attention is the enlargement of the submaxillary and cervical glands, often on one side of the neck alone. These grow gradually until they may finally appear on both sides as large as a fist, and produce considerable disfigurement. Sometimes several years may elapse before other glandular groups are affected, but, as a rule, it is a matter of months only before the axillary, then the inguinal, and perhaps the internal, glands are invaded. The changes vary greatly in rapidity and extent.

At first the *general health* may be but slightly affected. A little constitutional disturbance and some pallor may be complained of, though seldom before the glandular swellings are noticed. Then as the disease progresses the paleness increases and all the symptoms of a marked anemia appear—languor, failure of physical strength, beginning emaciation, gastro-intestinal derangement, headache, giddiness, palpitation, dyspnea, and edema of the legs. Later, the serous cavities contain effusion and there is a tendency to *hemorrhages*. Epistaxis and metrorrhagia may occur, and petechial spots, especially on the lower extremities, are not infrequent. The *physical signs* of anemia—hemic murmurs —are also present. An irregular slight or moderate pyrexia is common to most cases. Fever of a peculiar intermittent type has been observed, the intermissions and paroxysms each lasting for several days or weeks (see Fig. 38), and the term "chronic relapsing fever" has been applied in consequence. When these pyrexial exacerbations occur the cases generally run a more acute course. Ague-like paroxysms may persist for even months, as described by Pel, *of Amsterdam*.

The symptoms due to *mechanical compression* by the lymphomata are varied and numerous, depending upon the number, size, and distribution of the tumors. Hundreds of tumors may be present throughout the body, but, unless they press upon the adjacent nerves, the glands are not usually painful. Enlargement of the tracheal and bronchial glands may cause dysphagia, dyspnea, thoracic pain, disturbed phonation, and venous congestion, by pressure respectively upon the esophagus, trachea, bronchi, thoracic nerves, recurrent laryngeal nerves, superior vena cava, and the jugular veins. The obstruction to respiration may become so great as to produce death by suffocation.

Circulatory Symptoms.—*Congestion* of the head and upper extremities may be quite marked, and in such cases compensatory dilatation of the superficial veins is observed. *Edema* of the hand and arm may result from venous obstruction due to the pressure of very large axillary glands. The *heart's action* may be disturbed by pressure on the pneumogastric, and the heart itself may be dislocated by great gland-tumors within the chest. Under such circumstances the latter may be detected by dulness on percussion over the anterior mediastinal space.

Edema of the feet and legs may be an early indication of enlarged abdominal glands pressing upon the femoral veins. Albuminuria is not uncommon; ascites and hydrothorax are late conditions *Jaundice* is sometimes attributed to pressure upon the bile-duct. *Gastro-intestinal disturbances* may be troublesome, and are usually symptomatic of lymphoid growths in the stomach and bowels. In thin individuals gland-masses may be palpable over the abdomen. Deafness may be caused by growths in the pharynx.

Nervous Symptoms.—Inequality of the pupils and unilateral sweating of the face, owing to glandular pressure upon the cervical sympathetic, may be noticed in some cases. Sharp lancinating pains along the nerves may also be felt. *Pressure-paraplegia* and *neuralgic pains* variously distributed throughout the body should also be mentioned among the nervous manifestations.

Cutaneous Symptoms.—It has been suggested that the bronzing of the skin sometimes seen in Hodgkin's disease may be due to the pressure of enlarged glands upon the suprarenal capsules. An intense pruritus has been complained of, and the skin may be erythematous. Occasionally the thyroid and thymus glands are involved.

Spleen.—The slightly or moderately enlarged spleen can usually be felt just below the ribs, projecting toward the navel. Tenderness over the spleen and bones may be elicited. The characteristic feature in splenic pseudo-leukemia is the decided enlargement of the spleen without involvement of the lymphatics.

The *blood* shows a moderate diminution in the number of red corpuscles, and a corresponding diminution in the hemoglobin, the former in most instances numbering from 2,000,000 to 4,000,000 per cubic millimeter. There may be more or less leukocytosis, and sometimes the lymphocytes may preponderate relatively; if the latter be present in great numbers, the blood may show great similarity to that of lymphatic leukemia. An occasional normoblast may be seen.

Diagnosis.—Pseudo-leukemia is more readily confused with *tuberculous adenitis* than any other disease, particularly at the outset. Although an acute tuberculous adenitis may very closely simulate Hodgkin's disease and render a diagnosis almost impossible, more often the glands of tuberculous adenitis are slower in enlarging and extending than in this disease. In fact, extension of the lymphatic enlargements of tuberculosis is rarely seen as compared with pseudo-leukemia. Again, tuberculous adenitis is most common in the young, is unilateral rather than circumferential in the neck, and attacks the submaxillary glands oftener than the cervical chains along the sterno-cleido-mastoid. Again, periadenitis, adhesion, and suppuration of the glands occur in tuberculosis. Tuberculous foci in other organs may also be found. Intermittent attacks of pyrexia are an indication favoring Hodgkin's disease. In doubtful cases a gland may be removed for microscopic examination.

The blood should be examined in order to differentiate from *leukemia.*

Syphilis must be carefully excluded by the history, symptoms, and therapeutic test. *Neoplasms* of the lymph-glands may sometimes be difficult to distinguish from pseudo-leukemia.

The diagnosis of *splenic pseudo-leukemia* is to be made on the decided splenic enlargement without involvement of the lymphatics. The following conditions, however, must be distinguished: (*a*) Pernicious anemia with enlargement of the spleen; this is readily done by a blood-examination; (*b*) Cirrhosis of the liver, in which there is splenic enlargement; (*c*) The splenic tumor of chronic malarial poisoning. Here the blood should be repeatedly examined for the organism of Laveran, if the patient resides in a malarial region; (*d*) Idiopathic enlargement of the spleen without any anemia.

Prognosis.—This affection runs an almost invariably fatal course. The remissions and exacerbations of the disease are, however, notable. In some cases the termination may occur in a few months, but usually death ensues after the lapse of two or three years. It should be remembered that some instances of Hodgkin's disease seem to merge into a true lymphatic leukemia.

Grave indications are the rapid extension of the glandular enlargements, great debility, anemia, emaciation, steadily increasing and continuous pyrexia, thoracic pressure-symptoms, hemorrhages, and marked anasarca. Sometimes the tumors diminish greatly before death. In certain cases general streptococcus infection, intercurrent diseases, or such complications as empyema or nephritis, may be the immediate cause of death.

Treatment.—Local or surgical treatment is of no avail. Hygienic asures and the use of all possible agencies to support the strength of e patient should be resorted to, and the administration of arsenic in adually ascending doses, as for pernicious anemia and leukemia, ould be begun as soon as the diagnosis of pseudo-leukemia is made. he value of arsenic is undoubted in many cases, and Fowler's solution very generally used. Phosphorus has also been recommended. Inections of ichthyol, iodoform, or green soap may be tried for their sychic effect, and the galvanic current may also be applied topically. onics and nutrients may be of temporary service.

ANÆMIA INFANTUM PSEUDO-LEUKÆMICA.

Definition.—The above title was given by von Jaksch to a form of nemia occurring in childhood that bears certain similarities to leukemia, but is without the tendency to a fatal end. It is probably the same class of cases that Italian writers have classified under the name of *anæmia splenica infettiva dei bambini.*

Pathology.—Splenic enlargement is the most striking lesion. The organ is hard and dark red, and perisplenitis may be observed. The histologic examination shows a uniform hyperplasia of the tissue, such as is witnessed in ordinary splenic hypertrophy due to various conditions. The liver is enlarged in most cases, but presents practically normal appearances on section; slight enlargement of the lymphatic glands may also occur, though never lymphomatous tumors. Diffuse reddening of marrow has been described.

Etiology.—Children under the age of four, and particularly during the second half year of life, are especially prone to this condition. It is equally common in the two sexes, and is most often met with in rachitic infants, 16 of 20 cases collected by Monti and Berggrün having exhibited this etiologic factor. Hereditary syphilis, intestinal disturbances, and other diseases doubtless play a part in the etiology. The disease is a rare affection, occurring very seldom even in the largest children's clinics.

Symptoms.—The *onset* is gradual. The child becomes pale, weak, and often emaciated, and *enlargement of the spleen* is the most striking feature. Sometimes this reaches such a grade that the left half of the abdomen is practically filled, variations in its size being observed from time to time. Hepatic enlargement is frequently present, but does not correspond to that of the spleen, and the lower border of the organ is found to be sharp instead of rounded, as is the case in leukemia. Gastro-intestinal disturbances may occur in the course of the disease, and gradually increasing weakness may lead to a fatal end; death may also occur from peritonitis, bronchitis, or pneumonia.

Blood.—An examination of the blood will in many cases show an inordinate reduction in the number of red corpuscles. Nearly always the number is below 3,000,000. Degeneration of the corpuscles, polychromatophilia, and poikilocytosis are seen in the severer cases. Large numbers of nucleated erythrocytes, especially the normoblasts, may be found, and karyokinetic figures are frequently observed in the nuclei. A marked increase in the number of leukocytes is one of the characteristics, the number ranging from 40,000 to over 100,000, and the proportion of the red to the white at times being as low as 12 to 1. Considerable fluctuations in the number of leukocytes may occur from time to time. Von Jaksch insisted that the different forms of leukocytes occur in their usual relative proportions, or that the polymorphous forms are specially increased. The mononuclear elements, however, may rarely be increased.

The *nature of the disease* is difficult to determine, though the favorable termination of many cases, the lesser grade of hepatic enlargement, and the character of the leukocytosis distinguish these cases from leukemia. It is not even certain that it is a special disease-entity, and the evidence is in favor of its being a type of secondary anemia with peculiar features.

Diagnosis.—Some points of distinction from leukemia have been referred to above. In addition I would say that the absence of hemorrhages, purpura, and lymphomatous enlargements, and the presence of abundant nucleated corpuscles showing karyokinesis, together with the existence of rickets, point to a non-leukemic affection.

Prognosis.—The disease tends to a progressive increase of the anemia, but under treatment the majority of cases terminate favorably.

Treatment.—Hygienic measures together with the administration of remedies directed to the anemia constitute the treatment.

SPLENIC ANEMIA.

Definition.—A condition described by H. C. Wood in 1871; it is looked upon by most authors as a splenic form of Hodgkin's disease. Strümpell, Banti, and Bruhl also regard it as a distinct variety of anemia (*vide* p. 452). Some of the cases are, I am inclined to believe, instances of pernicious anemia, others of secondary anemia with splenic enlargement. Wentworth[1] contends that the blood changes in splenic anemia are those of secondary anemia.

Pathology.—Among pathologic characters, splenic enlargement is to be specially noted. It is an idiopathic enlargement of the spleen with anemia and without lymphatic involvement (Osler).

Etiology.—So-called splenic anemia is not rare in persons who have resided in malarial districts for a long period of time (although cases are recorded in which no previous history of malaria existed), and in rachitic persons.

Symptoms.—The affection is characterized by three stages:[2] (1) The initial stage, which shows extreme anemia, with marked loss of power and muscular wasting, emaciation, however, being usually slight, notwithstanding.

(2) The second stage is characterized by progressive enlargement of, and pain in the region of the spleen. Anemia is now profound, loss of strength extreme, and attacks of epistaxis or other mucus bleedings are common. Hemorrhages of the skin are also noted. The fever is apt to be hectic in type (ranging from 100° to 102°, and rarely even higher).

(3) The condition is that of progressive asthenia, ending in death.

The Blood Characters.—The anemia is of the chlorotic type. The red cells are usually near the normal count (3,700,000 to 4,000,000), while the hemoglobin is greatly reduced (40 per cent. or lower). Poikilocytosis may exist. The leukocytes may be normal or slightly increased. The *treatment* coincides with that of the graver forms of anemia.

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[1] *Boston M. and S. Jour.*, Oct. 31, 1901.

[2] S. West, in Allbutt's *System of Medicine.*

CHLOROMA.

Owing to its clinical resemblance to leukemia and pseudo-leukemia a brief reference to this comparatively new and rare affection may be made here. *Pathologically*, it consists of a sarcomatous growth, the primary seat of which is in the periosteum and bone in and about the orbit. The growth shows a pea-green pigmentation. Secondary growths may be widespread, the metastatic nodules being also green in color, but more circumscribed than are the lymphatic infiltrations of ordinary leukemia. In 2 cases reported recently, 1 by Dock and the other by Ayers,[1] the ages were fifteen and seven years respectively.

Symptoms.—Pain in the orbital region, exophthalmos, and deafness were noted early, and severe conjunctival hemorrhages and epistaxis occurred. Rounded elastic swellings were observed in the temporal and parotid, as well as in the orbital regions, corresponding to the chloromata. The blood was pale and watery, and leukocytosis was present, multinuclear leukocytes being noted. The eosinophiles were slightly reduced.

The *course* of the disease—spoken of by French writers as "green cancer"—is rapid, and death usually comes on within a few months.

DISEASES OF THE DUCTLESS GLANDS.

DISEASE OF THE SUPRARENAL CAPSULES.

ADDISON'S DISEASE.

Definition.—A constitutional disease, characterized by a degeneration of the suprarenal capsules or semilunar ganglia, a bronzed or pigmented skin, great bodily and mental asthenia, feeble circulation, and gastro-intestinal irritability.

This affection is named in honor of its discoverer, Thomas Addison of Guy's Hospital, London, who first described it in a monograph published in 1855, entitled "The Constitutional and Local Effects of Disease of the Suprarenal Capsules.

Pathology.—Addison emphasized the fact that while the suprarenal bodies were affected with a fibro-caseous alteration in many cases, the anatomic changes were by no means always the same. Both suprarenal capsules are usually diseased at the same time. Tuberculosis is the commonest condition, and is often associated with tuberculous lesions in other parts of the body, as in the lungs, bones, and other glands. Rarely, it seems to be primary, no other evidences of tuberculous infiltration being found. The capsules are enlarged, firm in places, and nodulated on the surface, owing to the caseous masses surrounded by fibrous tissue. Sometimes there is marked cicatricial contraction of the adrenals, and the adjacent structures may be found matted together with the capsules. *Microscopic examination* shows a reticulum of connective tissue surrounding a soft cheesy, granular, and fatty detritus, lymphoid cells, and some giant cells. Other morbid processes in the adrenals that are non-tuberculous in nature have also been found associated with Addison's disease, such as atrophy of one or both glands from interstitial cirrhosis, carcinoma or sarcoma, and chronic inflammation.

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[1] *Jour. Amer. Med. Assoc.*, Nov. 7, 1896.

Especial attention has recently been given to the condition of the *solar plexus* and *semilunar ganglia* of the abdominal sympathetic, and implication of these nervous structures by compression, cicatricial contraction, entangled in the cicatricial tissue surrounding the suprarenal bodies or by chronic inflammation, is not infrequently discovered, together with a degeneration and deep pigmentation of the semilunar ganglion-cells.

Enlargement of the solitary and agminated follicles of the intestine, and slight enlargement and some softening of the spleen are noted at times; parenchymatous or fatty degeneration of the heart, liver, and kidneys has also been noted in some instances. The thymus gland may be found to have remained normal, or even to have enlarged, perhaps. The deposition of pigment is in the same anatomic elements as in the negro—in the lower layers of the rete Malpighii.

The pathologic connection between the symptomatic phenomena of Addison's disease and the anatomical lesions has not been satisfactorily made out. The experimental evidence regarding the functions of the adrenals is imperfect; but it seems quite probable that some essential "internal secretion," influencing the normal metabolism of the skin and muscles, is diminished or absent in Addison's disease. On the contrary, cases exhibiting the clinical phenomena of this affection have occurred in which no suprarenal morbid processes could be found *post mortem.* Again, marked changes have been observed in these glands, while during life no symptoms of the disease had been noted. Hence, it is maintained by some that the abdominal sympathetic nerves and ganglia are directly concerned in producing the clinical manifestations, either by an independent morbid process or by extension from some adjacent organ. Others hold that both the adrenals and the sympathetic ganglia are the seat of pathologic changes. The data are not sufficient, however, to determine whether the principal involvement is nervous or secretory.

Etiology.—This is obscure. It has been held that some infection of the blood from without precedes the suprarenal and nervous lesions of Addison's disease. A *tuberculous* diathesis or infection has also been emphasized by some investigators, and Fleming and Miller[1] have reported a family with probable Addison's disease. A history of *injury* to the trunk has been noted in several cases. The disease is more common in Europe than in America, though it is rare everywhere. Analysis of 183 cases showed 119 males and 64 females (Greenhow). While the disease may affect all ages (it may even be congenital), it is usually found in early or middle life—between fifteen and forty years of age. That Addison's disease is due either to a general neurosis or to disturbed hematopoiesis is merely hypothetic.

Symptoms.—While it does not seldom happen that tuberculosis or carcinoma affects the adrenals, the purest and most typical symptoms of Addison's disease are apparently primary in their develop-

[1] *British Medical Journal,* April 28, 1900.

ment, and not those that usually attend the course of the former diseases.

Cutaneous Symptoms.—The gradual pigmentation of the *skin* of various parts of the body may be one of the first evidences of the affection. This pigmentation may have either a dusky-yellow, bronze or yellowish-brown, olive, deep or greenish-brown, or even black color. Although sometimes diffuse, the discoloration is not uniform over all parts of the body, but commences earlier, and becomes deeper especially on the exposed parts and where the normal pigmentation is marked, as the face, neck, backs of the hands, the axillæ, abdomen, groins, genital regions, and the areolæ of the nipples. Pigment-spots, often somewhat bluish in color, are also found on the *mucous membranes* of the mouth, lips, conjunctiva, and vagina. On the lips the discoloration takes the form of a dark streak, running lengthwise, near the junction of the skin and mucous membrane; or brownish patches or streaks corresponding to the points of pressure by the teeth may be noticed. Irregular stains with ill-defined borders may also be shown on the skin, corresponding to the lines of pressure exerted by garments, strings, suspenders, garters, etc. (Greenhow). White patches of *leukoderma* may be seen here and there, in marked contrast to the pigment-deposits.

General Symptoms.—The constitutional symptoms may exist in a slight degree before the pigmentation first attracts the patient's attention. There is gradual and progressive *asthenia* without apparent cause, great lassitude and loss of physical and mental energy, breathlessness, headache, dizziness, tinnitus aurium, sighing, and fatigue. The *blood-examination*, however, rarely shows any marked reduction of the erythrocytes or hemoglobin; nor is there any leukocytosis. The fat, particularly of the abdomen, may be well preserved.

Circulatory Symptoms.—The *heart's action* is weak and the pulse small and feeble; attacks of faintness and palpitation on exertion are common, as are functional murmurs and coldness and clamminess of the extremities. The blood pressure is greatly reduced before death (Turner).

Gastro-intestinal symptoms are usually prominent. There is a loss of appetite, and nausea and vomiting may occur early and either be paroxysmal or persistent. The *tongue* may be clean, and the gastric disturbances do not seem to follow errors in diet. *Diarrhea* may be troublesome in the latter stage, and is often associated with intractable vomiting. *Neuralgic attacks* of either sharp or dull, aching pain are referred to the epigastric, hypochondriac, and lumbar regions in about one-third of the cases. The *mind* is usually clear until near the last, but mental weariness is constant, and, as the later stages of the disease come on, the patient often lies in a somnolent, semi-comatose state. The physiognomy expresses fatigue, dejection, and apathy; the speech becomes slow and incoherent, and in many cases the patient passes into delirium. *Prostration* is profound, the weakness being disproportionate to the general condition.

Renal Symptoms.—Polyuria is sometimes evident, but albumin is seldom present. The amount of indican is increased, as it is in the urine of all of the cachectic diseases associated with destruction of albuminoids.

There is usually a diminished excretion of urea, but urobilin and uromelanin may be present in abnormal quantity. Tubercle bacilli may be found in the sputum.

Diagnosis.—The principal error in diagnosis is in the assumption that the case is one of Addison's disease, simply from the presence of patches of pigmented skin. Other conditions in which the discoloration may simulate that of Addison's disease are the following: (1) Carcinomatous and tuberculous disease, particularly when seated in the abdomen and when involving the peritoneum; (2) Hepatic disease, such as the cirrhosis of diabetes, protracted jaundice, chronic congestion, and lithemia ("liver-spots"); (3) Pregnancy, and uterine disease, in which the patchy discolorations (chloasmata) appear principally upon the face; (4) Irritation of lice and dirt and exposure, as in the case of tramps and vagrants ("vagabond's disease"); (5) Tinea versicolor; (6) Melanotic sarcoma; (7) Exophthalmic goiter; (8) Post-eruptive staining of syphilitic eruptions; (9) The administration of silver nitrate for a long time (argyria); (10) Marked brunette complexions and racial admixture.

When the pigmentation is scanty, of course the diagnosis is more difficult; but in all cases of pigmentation in which other causes may be excluded the progressive asthenia, unaccountable vomiting and diarrhea, easily compressible pulse, great bodily weakness, mental hebetude, and lumbar and epigastric pain render the diagnosis of morbus Addisonii, or *melasma suprarenale*, justifiable. It is to be remembered that the bronzing of the skin may precede as well as follow the constitutional symptoms.

In the negro the diagnosis of this affection is extremely difficult, both on account of the naturally dark skin and because of the dark discolorations of the oral mucous membrane, found even in health in many individuals.

Prognosis.—The course of Addison's disease is almost always chronic, though cases have been reported occasionally in which the onset has been sudden, with febrile phenomena and a comparatively acute course of a few months, or weeks even. Usually the disease lasts about one year, although some cases may continue over five or even ten years. Temporary remissions may be observed, but death is inevitable in by far the majority of instances. The termination is gradual, and by profound asthenia, or sometimes by coma, delirium, or convulsions (epileptiform).

Treatment.—The hygienic and medicinal treatment must have virtually the same objects in view as in the other grave cachectic diseases, and is both sustentative and symptomatic. As quiet a life as possible should be strictly enjoined, owing to the dangers of a sudden and fatal syncopal attack. Rest in bed is necessary in moderate and advanced cases during a part of the day for the former and constantly for the latter. The diet should be restricted to light nutritive, concentrated, and easily assimilable food, and particularly to the nitrogenous or proteid substances. An absolute milk diet may be necessary in some cases.

Iron and arsenic may be administered in the anemic cases, and strychnin, guaiacol carbonate, phosphorus, and the nuclein preparations may also be given, along with bitter tonics. Bismuth and salol may be of great service in controlling the diarrhea that often occurs. The nausea and vomiting may be relieved by unfermented grape-juice, albumin-water, champagne, cracked ice, cerium oxalate, creasote, and the like. Electricity is often a valuable adjunct in the treatment of the muscular weakness and nervous exhaustion, and even in reducing the pigmentation.

It seems quite probable that the administration of the extract of suprarenal capsules will prove to be of considerable value in causing marked improvement, if not a permanent cure, in a certain percentage of cases. In one instance mentioned by Osler, in which a glycerin extract of a pig's suprarenal was given at first in doses of half a glass three times a day, improvement was noted in the temperature, pulse, weight, and physical and mental vigor from the first week of the treatment, which was continued for three months and a half. Eight months after the treatment was begun the patient appeared to be well and strong, and attended to business; the pigmentation, however, was not removed. In a recent case of my own this remedy produced like results. Robin mentions a case treated by the administration of suprarenal gland that has shown persistent good health for three years. For the present, however, too positive a value should not be attributed to the suprarenal extract, and results contrary to the above are to be found in the literature.

DISEASES OF THE THYROID GLAND.

THYROIDITIS.

Definition.—Acute inflammation of the thyroid gland. The gland may either have been previously healthy or the seat of a goitrous enlargement; when inflammation attacks previously diseased or enlarged thyroid tissue the term *strumitis* is often used.

Pathology.—The gland is swollen, boggy, and the seat either of a single large or of multiple small abscesses; the numerous large blood-vessels are engorged; and hemorrhages, thrombi, and areas of tissue-necrosis are frequently found.

Etiology.—Thyroiditis is seldom primary in origin. It may be caused by traumatism; but usually it is secondary to one of the infectious diseases, such as small-pox, typhus, typhoid fever, or malaria. Rheumatism has also been given as a cause. Hemorrhages into the substance of a goiter, whether apoplectic or traumatic, may predispose to a strumitis that may be excited by the introduction of streptococci by an unclean needle, etc. Repeated congestions of the thyroid or a simple acute congestion may also dispose to thyroiditis.

Symptoms.—There are *fever*, *pain*, *swelling*, and *suppuration* in one or the other lobe of the gland. *Venous obstruction* may be serious and give rise to vertigo, headache, cyanosis, and epistaxis; and compression of the windpipe by the great swelling may cause death before the abscess bursts. Resolution occurs infrequently, especially in the "strumous" cases. Indeed, the symptoms of a strumitis are usually more severe, owing to the greater size of the thyroid, a tendency to metastasis, and to the burrowing of pus into adjacent tissues leading to perforation and rupture of the abscess into the trachea or esophagus.

Diagnosis.—Thyroiditis must be differentiated from the *laryngeal perichondritis* that is also seen in the course of infectious diseases, as typhoid fever and small-pox. The higher and more median position and the smaller swelling of laryngo-chondritis are distinctive points.

Prognosis.—The outcome is usually favorable in all cases in which spontaneous rupture occurs externally or when evacuation of the pus is effected. Strumitis runs a less favorable course for the reasons mentioned above.

Treatment.—This is antiphlogistic and surgical. The pus must be evacuated, and tracheotomy or thyroidectomy may become necessary.

GOITER.

(*Bronchocele.*)

Definition.—A chronic hypertrophy and hyperplasia of a portion or the whole of the thyroid gland. It is of obscure origin, involving one or more of the structural tissues, and is subject to various degenerative changes.

Pathology.—Several different varieties are described. In the *simple hypertrophic* or *parenchymatous* form there is a hyperplasia of all the original tissue-elements. The *follicular* form shows an increase of the true glandular elements alone.

Fibrous goiter is that variety in which the interstitial tissue or stroma is increased out of all proportion to the hyperplasia of the follicles, which are also involved in a much slighter degree. This variety of goiter may have an inflammatory origin (thyroiditis). In old cases marked sclerosis may be assumed. There is also a *vascular* variety, in which the blood-vessels are enormously dilated. More commonly the veins are affected; but in the so-called aneurysmal variety the arteries are chiefly involved. The intense venous variety of vascular goiter has been denominated "cancerous tumor of the thyroid," and the whole gland may in such cases be quite elastic and like spongy erectile tissue. Follicular hyperplasia is often associated with vascular enlargement.

The special varieties of goiter due to degenerative changes are the *cystic*, *amyloid*, *colloid*, and *calcareous*, and of these the first named is the most common. It consists in the development in a large goiter of one or more large or small cysts filled with different kinds of fluid of varying consistency. Sometimes the liquid is colloid or mucinous in nature, and it may be chocolate-colored and contain the residue of hemorrhages (cholesterin, fatty products, and albumin). Amyloid changes affect principally the vessels; colloid changes are also frequent, while calcareous infiltration is seen in very old fibrous goiters. Inflammation and suppuration of the goitrous gland may ensue.

Etiology.—Goiter may occur anywhere sporadically. Endemically and in its worst forms it occurs in the mountainous districts of Europe, Asia, Mexico, and South America, particularly in the Alps, Pyrenees, and Andes. It has also appeared in certain limestone regions, such as New England and Ontario, Canada, where the *habitual use of limestone-water* for drinking purposes seems to induce the disease. *Heredity* undoubtedly plays a part in its causation, certain children having been born with goiter. Occasionally it has become epidemic in certain sections of the goitrous districts in Europe where military garrisons have been stationed, thus indicating the possibility of some infectious influence. Women are more liable to goiter than men, and it is more common to find it after ten or twenty years of age. It has been alleged that pregnancy also influences the development of this condition.

Symptoms.—The enlarged thyroid is readily *recognized* and *felt*, though the patient may complain of nothing but the disfigurement, except when the tumor is of sufficient size to cause symptoms of compression. The goiter develops very gradually, and may vary in dimensions from the merest perceptible enlargement to a growth that overhangs the chest and greatly hinders the movements of the head. It may or may not be uniform in its development, and is often more enlarged on the right side and in front than on the left side. It is not infrequently observed to increase in size with each succeeding pregnancy and during or after each menstrual flux.

The tumor is *painless*, is not adherent to the overlying skin or to any of the neighboring bones, and rises and falls during the act of swallowing, moving with the larynx. The *veins* covering it are swollen and prominent. It interferes with respiration oftener than with deglutition, causing dyspnea; alteration or loss of the voice may also ensue. Displacement and distortion of the trachea, the vessels, and other cervical tissues may be produced. Large *pendulous growths* usually cause less serious discomfort than the small encircling tumors that extend downward into the thorax. Headache, somnolence, and marked cerebral symptoms, such as tetany and convulsions, have been described.

The *general health* or nutrition seldom fails unless inflammation and suppuration (strumitis) attack the goiter during the course of some infectious disease, as not infrequently happens, or in cases in which the thyroid function is abolished, leading to the profound nutritional and cerebral disorders of cretinism in children or myxedema in adults.

Dettrich and Osler have each reported an instance of a goitrous growth affecting aberrant portions of thyroid found in the upper region of the pleural cavity, one on the right and one on the left side.

Sudden death may ensue in a few cases, either from pressure on the vagi, or from a severe hemorrhage.

Auscultation often reveals a loud blowing murmur, especially marked in the vascular bronchoceles. *Palpation* over the tumor often shows the bossellated surface present in cystic goiter; fluctuation may also be detected in such cases, as well as over the abscess of a strumitis.

Diagnosis.—Goiter is easily differentiated from other enlargements. The constant location and the character and course of growth of the bronchocele are distinctive. If both lobes of the thyroid are affected, making a symmetric swelling, the diagnosis is almost assured. Bronchocele is not easily confounded with other cervical tumors, such as *lymphadenoma*, *glandular tuberculosis*, *carcinoma* or *abscess* of the *thyroid*, or sebaceous cysts. A characteristic feature of tumors of the thyroid is their vertical movement during the act of deglutition.

Prognosis.—This is guardedly favorable as to life, but unfavorable as to cure. The course is chronic.

Treatment.—Prophylaxis should be practised in goitrous districts by the drinking of boiled water only, and removal to a non-goitrous region is advisable. The majority of drugs recommended for internal and external use have been proved valueless, though in the parenchymatous and follicular forms potassium iodid by the mouth and the vigorous and methodic use of iodin over the tumor have been much lauded. Mercurial ointment—the red or biniodid especially—has also been recommended for local application. Ergot or belladonna in progressively increasing doses may do good in vascular goiters. The younger and softer goiters may also be benefited by electrolysis, needles attached to the negative pole being inserted into the substance of the tumor while a large sponge or clay positive electrode is placed in the vicinity.

In the older, fibrous, and degenerated goiters surgical treatment alone may be of service. Injections of iodin, tapping of cysts, incisions of the isthmus, and ligature of the thyroid arteries have been practised among the lesser operations. Thyroidectomy, or a partial extirpation of the thyroid, is the radical and final operation.

Recently, the fresh, chopped thymus gland of the sheep, spread on bread, was given in 20 cases of follicular and parenchymatous goiter with gratifying results. A perceptible diminution in the size of the goiter was demonstrated by actual measurements. Complete recovery, in an anatomical sense, however, was realized in two cases only.

EXOPHTHALMIC GOITER.

(*Graves's Disease; Basedow's Disease.*)

Definition and Nature.—Although the view cannot be unreservedly accepted, exophthalmic goiter is probably of thyroid origin and is dependent upon an abnormal action (or over-action) of the thyroid gland; it is characterized clinically by tachycardia, tremors, enlarged thyroid, and exophthalmos. Among other leading theories the following may be briefly stated: (1) that it is due to disturbed innervation (Buschan); (2) that the seat of the disease resides in the medulla oblongata; (3) that it is an affection of the sympathetic nerves; and (4) that it is a disease of the central nervous system associated with a chronic intoxication.

The theory held by Möbius, that exophthalmic goiter is attributable primarily to a disturbance of the function of the thyroid ("hyperthyroidation"), a condition directly opposed to the lack of thyroid function, as in myxedema, is amply supported by clinical evidence, the complex symptom-group of the former being directly antagonistic to that of the latter disease. Thyroid-feeding, moreover, while it sometimes causes parenchymatous goiters to disappear rapidly, usually aggravates the symptoms of Basedow's disease. Regarding the *pathologic changes* in the thyroid little is known. Brissaud[1] found in 25 cases of various chronic diseases changes in the thyroid, and, although the glands in exophthalmic goiter showed no changes peculiar to that disease, yet quantitatively the lesions were always such as to make "hyperthyroidation" possible. Persistence of the thymus gland is common (Hector MacKenzie). Muscular changes, probably resulting from toxemia, explain the profound muscular weakness (Askanazy). Jaunin[2] and Gautier[3] contend that chronic iodism and exophthalmic goiter are practically the same condition. Minor[1] affirms that the disease may be due to gastro-intestinal autointoxication.

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[1] *Mercredi méd.*, No. 34, 1895. [2] *Rev. méd. de la Suisse rom.*, No. 5, p. 301, 1899.

[3] *Med. Record*, Dec. 2, 1899.

Etiology.—It is more common in women than in men. A table of 200 cases showed 161 females and 39 males (Eshner); and, although it has been met with at both extremes of life, it is seen usually in adults. The influence of heredity is undoubted, and several members of a family may suffer, persons that possess a sensitive nervous organization being especially prone to the disease.

Among direct causes are emotional disturbance, worry, severe acute disease (noted in two of my cases), and prolonged mental or physical strain.

The disease may also occur as a secondary complication in the course of simple goiter, affections of the nose, and pregnancy; this variety, however, is to be distinguished from the primary or essential form.

Symptoms.—The development of the characteristic symptoms is generally *gradual*, though it may rarely be *rapid*. In the so-called abortive form the symptoms arise somewhat rapidly, but early subside.

In *acute* Basedow's disease the symptoms consist of an excessively rapid action of the heart, incessant vomiting, purging, and marked exophthalmos, with or without pronounced cerebral symptoms. J. H. Lloyd's case proved fatal after an illness of three days.

In the *chronic* form heart-hurry is almost constantly a conspicuous early symptom, and not seldom have I found that it precedes for a long period of time the appearance of the remaining characteristic features (enlargement of the thyroid, exophthalmos, and tremor). The pulse remains at or over 100 beats per minute, and upon unusual exertion or excitement the heart's action becomes violent and irregular, the pulse even reaching 160 or over. Palpitation, often with breathlessness, is a distressing symptom.

Cardiac Physical Signs.—*Inspection* reveals a forcible impulse that is not displaced, though late in the affection it may be much extended in superficial area. The carotids and the abdominal aorta beat more or less violently, and the capillaries and veins of the hands may also pulsate visibly. *Palpation* detects an increased force of the cardiac impulse. The area of *percussion-dulness* may be somewhat increased, as hypertrophy and secondary dilatation supervene. On *auscultation*, blowing murmurs over the heart and the great vessels, as well as an increased accentuation of the valvular sounds, may be audible even for some distance from the patient. Distinct *bruits* may be heard over the base and manubrium.

Exophthalmos.—Protrusion of the eyeballs is usually present. The degree of exophthalmos varies greatly from time to time in the same case—a fact that points to an increased amount of blood or lymph in the orbit as its cause. In advanced cases permanent prominence of the balls may be attributable to augmentation of the orbital adipose tissue. On closing the eyes a rim of white is visible above and below the cornea, and Graefe's sign, immobility of the upper lid when the eye is turned downward, are two symptoms of great diagnostic importance. Möbius has called attention to the inability to converge the eyes upon near objects; and Stellwag, to an apparent separation of the eyelids, due to spasm or retraction of the upper lid. The pupils and the vision are unaffected while the patient winks less often than in health. Abnormalities are rarely presented by the optic nerves, and ulceration of the cornea may supervene. The retinal arteries pulsate.

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[1] *Med. Record*, Dec. 2, 1899.

Thyroid enlargement may either accompany or follow the exophthalmos, and has for its cause the great dilatation of the vessels, particularly of the arteries. The enlargement is usually moderate, and may be general or partial, the size of the gland exhibiting sudden variations, since it is dependent upon the circulatory disturbance. *Inspection* may also show visible pulsation; *palpation* feels a thrill, and *auscultation* renders audible a double systolic murmur. The latter sign is probably present in most instances, though not constantly.

Nervous Symptoms.—Muscular tremors form an early symptom; they are involuntary, and fine in character, numbering about eight to the second (Osler). The characteristic features of *neurasthenia* appear and gradually increase in intensity. *Mental disturbances*, particularly marked depression or great excitability, are common, and even mania (which may prove speedily fatal) or melancholia may be observed.

Cutaneous Symptoms.—The *temperature* may at intervals be moderately elevated, and this symptom may be associated with profuse sweatings. Among other cutaneous phenomena, though these are for the greater part occasional, are *pigmentation* (which, in the case of a physician whom I recently saw suffering from Basedow's disease, was as pronounced as in typical Addison's disease), scleroderma, urticaria, pruritus, and circumscribed solid edema. In the advanced stage malleolar *edema* sets in and may become general. A marked diminution in the cutaneous resistance to the electric current has been noted by Charcot. The forehead is not wrinkled as in health.

General Symptoms.—Muscular weakness, either local or general, is pronounced; the patient becomes anemic and is at last extremely emaciated. *Vomiting* and *purging* may appear at different times and assume great gravity, and in some cases *hemorrhages* (epistaxis, hemoptysis, hematemesis) tend to supervene. *Albuminuria* and an increased amount of urine, with glycosuria, are among the commoner complications. Louise Bryson has maintained that diminution in the chest-expansion is a characteristic sign of exophthalmic goiter; and Patrick,[1] who examined 40 cases, found that there was an average diminution, but believed it to be proportionate to the amount of the general muscular weakness. Rarely a *myxedematous* condition is associated; probably the disease is also remotely related to scleroderma.

Diagnosis.—The diagnosis of Graves's disease may be made when tachycardia or delirium cordis and fine, general muscular tremors are present. Exophthalmos and enlargement of the thyroid are often late-appearing symptoms, and are as often temporarily lacking even in fully-developed cases. Rarely, either or both of these signs may be permanently absent. On the other hand, in a few cases exophthalmos is the sole characteristic feature for a long time, though it is eventually followed by an unmistakable symptom-group. Parenchymatous goiter presents a non-pulsating tumor, and hence is easily distinguishable from the thyroid enlargement of Basedow's disease with its additional unequivocal symptoms.

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[1] *Deutsche med. Woch.*, Dec. 20, 1894.

Course and Prognosis.—The chronic form of the disease endures, as a rule, for a few years. A gradual subsidence of the cardinal symptoms for a long period has been noted, and in such cases complete recovery may be claimed. In fully-developed cases the prognosis formerly was almost hopeless, but since the introduction of the operative treatment many cases have been greatly benefited, and others, though constituting a smaller number, have been entirely cured.

Treatment.—This is (*a*) *Hygienic*, (*b*) *Medicinal*, and (*c*) *Operative*.

(*a*) **Hygienic.**—The environment, both physical and mental, should be made as favorable as possible. A change of climate, and especially moderate elevation, in cases not too far advanced, bring about beneficial results. Such elevation (3250 feet) produces a sedative effect upon the nervous state that reacts most favorably upon the circulatory organs, while the purity and tonic quality of the air have a general strengthening and restorative effect (Yeo). Among other promising measures may be mentioned the wet-pack, methodical hydrotherapy with massage, and a continuous galvanic current. The electric treatment should be given a thorough trial over three or four months (Osler). The local use of an ice-bag to the precordium has acted admirably in reducing the heart-hurry in a few cases of my own. I have also observed favorable results from carefully graduated physical exercise. Rest in bed for a few weeks at a time, at intervals, is often followed by improvement, though I have never seen complete cure follow this plan of treatment.

(*b*) **Medicinal Treatment.**—This is probably secondary to the hygenic and operative measures. In two cases of my own, however, recovery followed the persistent use, for about six months, of the following prescription:

℞. Extr. digitalis,	gr. iv	(0.259);
Extr. ergotæ (Squibb),	ʒss	(2.0);
Strychninæ sulph.,	gr. ss	(0.032);
Ferri arsenias,	gr. ij	(0.129).

M. et ft. capsulæ No. xxiv.
Sig. One t. i. d. after meals.

In 2 other cases (one, a trained nurse) the use of sodium salicylate (gr. x—0.648—four times a day) was followed by almost total relief. L. Webster Fox also warmly advocates the latter remedy in this affection. Trachewsky, in Kocher's clinic, found that sodium glycerophosphate (gr. xx—1.296—three or four times a day), had the effect of diminishing the size of the enlarged thyroid glands, and Starr[1] has also found this remedy of great service in several cases. Other therapeutic agents that have been extensively employed, but with doubtful advantage, are aconite, veratrum viride, and belladonna. From all of the clinical testimony at hand I feel convinced that thyroid-feeding is contraindicated in the treatment of Basedow's disease, unless a myxedematous condition be associated, when it may prove efficient. S. Solis-Cohen and others have used extract of suprarenal gland with good results.

(*c*) **Operative Treatment.**—Starr[2] has collected 190 cases in which some form of operation was performed. Of these, 74 are reported as completely cured, many of them having been watched two to four years before the result was published; 45 of the cases were improved, and 23 died immediately after operation. The symptoms preceding the fatal result are sudden hyperpyrexia, with rapid pulse, nervous distress, sweating, cardiac failure, and collapse. The statistics of Kinnicutt and of Abram[1] (particularly the latter) show less encouraging results from operation. It is to be remembered that under the most favorable circumstances a complete cure will not be attained immediately, and frequently not for several years. I am convinced that removal of the entire gland is not to be advised, since myxedema will likely result. Bilateral resection of the sympathetic nerve has been done by Schwartz and others with marked benefit. Rehu[3] presents a statistical report of 32 resections of the sympathetic: 31.1 per cent. were cured, 50 per cent. improved, 12.5 per cent. were unimproved, and 9.5 per cent. proved fatal. Resection is recommended as the best operation.

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[1] *Medical News*, April 18, 1896.

[2] *Loc. cit.*

MYXEDEMA.

(*Sporadic Cretinism.*)

Definition.—A general nutritional disorder, consequent upon atrophy and loss of function of the thyroid gland, and characterized by a myxedematous infiltration of the subcutaneous tissue and a cretinoid cachexia.

Three varieties occur, as follows: (1) True myxedema; (2) Cretinism (the absence of thyroid function—congenital, or lost during childhood); (3) Operative myxedema, due to total removal of the glands for surgical reasons or in experiments upon lower animals.

Nature of Myxedema Proper of Adults.—Charcot, who gave the name of *cachexie pachydermique* to this disease, believed it to be of trophoneurotic origin. Atrophy of the thyroid is pretty constantly present, and the gland may either be converted into a small fibrous mass or be entirely absent, so that the causal relation between myxedema and functional and structural alterations of the thyroid seems to be conclusive. Moreover, the therapeutic test of improvement under the administration of thyroid extract sustains this view. It is probable that the active thyroid supplies some essential secretion which maintains normal metabolism, though this product has not been isolated. Its existence being inferred, however, it has been called *thyroidin.*[3] Ponfick has pointed out that the hypophysis sometimes shows changes resembling those in the thyroid gland. The fact that in a good many cases of myxedema a considerable portion of the thyroid gland is unaltered and partly capable of functionating arouses a suspicion that the hypophysis may share in the production of this disease.

Etiology.—The thyroid was destroyed by *actinomycosis* in a case of myxedema reported recently. Myxedema may also be secondary to *exophthalmic goiter*, but it is then, as in the case of a simple acute goiter, only a transient condition. Women are much more frequently affected than men, and a neurotic condition may precede some cases. The disease may affect several members of a family, and hereditary transmission through the mother has been observed. Sisters may suffer, one from myxedema and the other from exophthalmic goiter. Pregnancy may cause a disappearance of the myxedematous symptoms (Osler). The symptoms may reappear after delivery.

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[1] *American Year-Book of Medicine and Surgery*, 1897.

[2] *Soc. Rep., Munch. med. Woch.*, No. 41, p. 1357, 1899.

[3] The term "thyroidin" has also been given to a substance possessing specific therapeutic activities that has been obtained from the thyroid gland of the sheep.

Symptoms.—The myxedematous condition is most plainly noted in the *face*, the skin being swollen, but inelastic, rough, dry, and firm. The lines of facial expression are obliterated, and the features are broad, coarse, immobile, and bulky. The *physiognomy* is stupid, dull, and phlegmatic, and simulates imbecility. The *hair* falls out, owing to deficient nutrition; and the general bulk of the body is markedly increased. Pressure does not produce *pitting*, as in true edema. According to Ord, the local tumefaction of the skin and subcutaneous tissue is most frequently prominent in the supraclavicular regions. The *mucous membranes* are also infiltrated, and the teeth may become loosened. The tongue, lips, and nose are thickened, and the voice is monotonous, slow, and has a "leathery tone," "with curious nasal explosions at short intervals during speaking." Bodily movements are slow, and the gait is heavy and uncertain on account of disturbed co-ordination.

Nervous Symptoms.—Mental perception and thought are also slow, and the memory is defective and slow to respond. Not infrequently there may be considerable irritability, or hebetude alternating with sudden excitability. The patient may become suspicious, and later is subject to delusions and hallucinations; or the apathy may pass into a melancholia, ending at last in dementia. Ord mentions "the aggravation of all symptoms during low climatic temperatures;" and "among the minor or accessory signs may be quoted abnormal subjective sensations, belonging particularly to taste and smell; occipital headache; marked alterations of temper; and a curious persistence of thought and action, overriding all attempts at interruption by friends or observers."

The *temperature* in myxedema is usually either normal or subnormal. Albumin and sugar are occasionally found in the urine, but the quantity of nitrogen excreted is small, owing to the diminished metabolism of proteids. *Hemorrhages* from the nose, gums, and bowels sometimes occur. *Ascites* also may be present in some cases, and may simulate ovarian tumor. The thyroid is not palpable, partly because of its atrophy, and partly because of the thickened myxedematous tissues of the neck.

The **diagnosis** is not difficult if one bears in mind the characteristic manifestations described above. Mxedema could hardly be mistaken for acute or chronic nephritis in the absence of pitting, etc., as some have supposed. Chapman[1] mentions a solid appearance of the conjunctiva as an early sign of diagnostic value.

The **prognosis** is guardedly favorable in a majority of the cases since the introduction in the treatment of thyroid-feeding. The course of the disease is slow, however, often lasting from five to fifteen years, and death from intercurrent disease is not uncommon.

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[1] *Lancet*, Sept. 30, 1899.

Treatment.—Until the advent of thyroid-feeding the treatment of myxedema was palliative, and usually unsuccessful.

A warm and equable climate is very desirable, owing to the subnormal temperature from which the patients frequently suffer. The various warm baths—as the Turkish, Russian, and electric—should be employed for the same reason. Pilocarpin has been recommended, and strychnin and arsenic have been administered for their tonic effect.

Since the brilliant results obtained by Murray, however, the internal use of the thyroid gland of sheep or calves has come into a well-deserved favor in the treatment of all cases of myxedema, whether of the so-called true form, of sporadic cretinism, or of the cachexia strumipriva. The gland may be given raw or cooked, in the form of the glycerin extract, or in the dried and powdered extract; the last named is sometimes put into tabloid form. If cooked, the gland should be only partially "done." The fresh thyroid is minced and often spread on bread, and from one quarter to half a gland may be taken daily.

The glycerin extract is readily made. "Several dozens of thyroids of young sheep or calves are carefully separated from the connective tissue, cut into small pieces about the size of a bean, and then put into a jar and covered with glycerin of the best quality, allowing 2 c.cm. of glycerin for each lobe of the thyroid used. The mixture is permitted to stand for twenty-four or thirty-six hours, and is then squeezed through a cloth, so as to get out as much liquid as possible. Of this, 2 c.cm., corresponding to about half a gland, may be given at a dose. If used for hypodermic injection, to a dram (4.0) of the glycerin extract is added half a dram (2.0) of a 1 per cent. solution of carbolic acid in distilled water, of which mixture from 10 to 15 minims (0.66–1.0) may be injected three or four times a week."[1]

It is safest—for reasons that will be pointed out below—to begin with quite small doses, and gradually increase, especially if there is much gastric irritation. Not more than 5 minims (0.333) of the glycerin extract should be given at the start. This dose may be increased gradually until 15 or 20 minims (1.0–1.33) are taken three times daily. From 3 to 5 grains (0.194–0.324) of the powdered gland or tabloid form will be a safe commencing dose in adult myxedema: a caution, however, is necessary regarding the various manufactured preparations of the thyroid gland, some of which are impure and even dangerous, owing to the careless handling or fraudulent substitution in order to meet the demand for thyroid extracts on trial in other affections.

The toleration of thyroid-feeding does not depend upon the volume, but upon the functional activity, of the gland, and this fact, together with the evidences of toxic action reported in some instances of the administration of thyroids to a maximum degree, make it important to urge again—as intimated above—the necessity of small dosage at the beginning of treatment, and the most careful and judicious increase in the quantity given. The additional fact of an occasional cumulative action should also be emphasized. Should vomiting, renal pain, tachycardia, suffusion of the face, syncope, vertigo, or marked headache supervene, the remedy should be stopped at once. The treatment may be resumed again cautiously, alternating with intervals of cessation. I have observed that by combining arsenic with any of the preparations of thyroid the toxic effects of the latter can be in great measure obviated. Good results are obtained usually within a month, though it is probable that even after all the symptoms have subsided the treatment may have to be continued off and on if the thyroid gland seems to be permanently atrophied.

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[1] Osler, in the *Amer. Text-book of Therapeutics*, pp. 926, 927.

Cretinism, Sporadic and Endemic.—Here there is a congenital atrophy or absence of the thyroid gland, or an enlargement by the growth of fibrous tissue at the expense of the glandular elements. Cretinism may also develop in early infancy. The patients are often the children of parents noted for violent emotion and having various neuroses and goiter, and syphilis has also been supposed to have a causative influence. Congenital myxedema is quite common only in regions where goiter is endemic, and hence it is rare in America. A marked sporadic case has, however, been in the Philadelphia Hospital for many years.

Symptoms.—Cretins are dwarfs with large heads and faces, thick lips, thick protruding tongues, broad bodies and members, and prominent abdomens. The subcutaneous tissues are myxedematous. Umbilical hernia has been noted. The mental condition is that of idiocy, and physical growth is retarded and slow. Speech is unintelligible or nearly so, and the voice harsh. Walking may never be accomplished, and is always slowly developed. There is anemia, the blood being of a fetal type. Rheumatic symptoms sometimes occur.

Prognosis.—The disease is progressive until about the fifteenth year in those cases developing during early childhood. Congenital cases usually die shortly after birth. At the twentieth or thirtieth year "the mental and physical characters are those of childhood."

Treatment.—Thyroid-feeding has been followed by beneficial results, the checked growth having recommenced and the cretinic aspect having been largely lost. S. Küh[1] has employed iodothyrin in one case with quite as satisfactory results as those from the dried thyroid, while it caused no disturbance of the stomach or other disagreeable effects.

Operative Myxedema, or Cachexia Strumipriva.—Extirpation of the thyroid for surgical reasons has given rise to the gradual production of symptoms and conditions identical either with true myxedema or with the cretinoid state. Partial removal of the gland is not followed by cachexia strumipriva, nor is complete thyroidectomy when accessory glands are present elsewhere.

The administration of raw or broiled thyroids, or of their various extracts or preparations, must also be employed in this form of myxedema, and should be continued throughout the rest of the patient's life, perhaps with intervals of withdrawal of the feeding until the improvement gained begins to lapse.

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[1] *Philadelphia Medical Journal*, April 8, 1899.

PART IV.

DISEASES OF THE RESPIRATORY SYSTEM.

I. DISEASES OF THE NOSE.

ACUTE RHINITIS.

(*Acute Nasal Catarrh; Acute Coryza.*)

Definition.—An acute catarrh of the Schneiderian membrane, sometimes tending to involve the adjacent sinuses and passages. It is known to the laity as "cold in the head."

Etiology.—Its most conspicuous cause is exposure to draughts of air and to the influence of the atmospheric vicissitudes that are especially prevalent during the winter and spring seasons. It often results from the inhalation of irritants (physical, chemical, or biological). It may also display epidemic behavior, and this fact seems to point strongly to its microbic origin. Hence local disturbances of the circulation due to exposure are to be regarded as the accidental means of preparing the soil for bacterial invasion. Acute rhinitis may be also secondary to, or propagated from, inflammations of the faucial mucosa by contiguity.

Symptoms.—Sensations of *chilliness*, succeeded by *feverishness* (the temperature reaching 100° to 101° F.; 37.7°–38.3° C.), frequent *sneezing*, *headache*, and a feeling of general ill-health are among the prominent features that attend the development of coryza. Pains in the extremities and back tend to appear only in severe cases. The pulse is frequent, the skin dry and unduly warm, thirst is increased, while the appetite is impaired, and constipation often attends. The *nasal mucosa* is swollen, and thus interferes both with the nasal respiration and the senses of smell and taste; its color is deepened, its surface covered at first with opaque mucus, and later with a muco-purulent secretion. Among early symptoms is the discharge of a watery, irritating secretion from the nares and a maceration of the epidermis, with resulting abrasions. On account of the swelling of the mucosa of the lacrymal ducts the tears flow down over the cheeks. Adjacent mucous surfaces then become involved, giving rise to conjunctivitis, catarrhal pharyngitis, laryngitis, and finally, in the severer types, bronchitis. Naso-labial herpes is not uncommon. As the affection progresses the secretion becomes more abundant and turbid and more or less pyoid. The symptoms due to extension of the catarrhal inflammation vary with the organs or structures involved. The disease runs its *course* within five or six days, but the nasal discharge, which gradually diminishes, usually persists for a few days longer.

Diagnosis.—In the presence of the above-mentioned symptoms the disease is readily recognized. In well-marked cases, however, the possibility that an infectious disease may be developing, the beginning of which is characterized by nasal catarrh (measles, influenza, etc.), is to be recollected.

Prognosis.—Except in neglected cases, which result in bronchitis, and occur at one or other extreme of life, the disease is free from danger. The nursing infant may have to be fed with a spoon temporarily.

Treatment.—At the outset a purge, consisting of calomel (gr. ij—0.129), or a pill of blue mass (gr. v—0.324) at night, followed by a Seidlitz powder in the morning, is advisable. To children a dose of castor oil may be given. The early administration of a diaphoretic, such as Dover's powder (gr. v–x—0.324–0.648) at night may arrest the complaint, and quinin in a large dose (gr. xij–xv—0.777–0.972) at night may cut short the course of the disease. When the above-mentioned abortive measures fail, the following tablet produces good results:

℞. Quinin. sulphat.,	gr. ijss	(0.162);
Extr. belladonnæ fl.,	♏jss	(0.099);
Sodii salicylatis,	gr. xxx	(1.944);
Camphoræ,	gr. ijss	(0.162).

M. et ft. tablet No. x.
Sig. One tablet every hour or two.

For the fever aconite may be employed, and, if the throat be involved, bryonia may be given in conjunction.

Local Treatment.—This aims at soothing as well as at reducing the swelling of the Schneiderian membrane. The compound tincture of benzoin forms a soothing inhalation (ʒij to a pint—8.0 per half liter—of water) when raised nearly to the boiling-point; the vapor is inhaled for ten or fifteen minutes at a time. With a view to reducing the swelling a solution of cocain (strength 2 to 4 per cent.) may be temporarily used; Mackenzie recommends this admirable combination:

Menthol,	gr. v	(0.324);
Pinol	♏v	(0.324);
Benzoinol,	f℥j	(32.0).

In severe cases the patient should be kept in-doors and in an atmosphere of even temperature.

CHRONIC RHINITIS.

(Chronic Nasal Catarrh.)

Two forms are recognized, the hypertrophic and atrophic, and these, though, as a rule, occurring separately, may be found in combination.

Pathology.—The morbid changes in hypertrophic rhinitis consist in an enlargement of the lower turbinated processes, together with redness and swelling of the nasal mucosa that may be general or limited either to the anterior or posterior nares. As the disease progresses the thickening of the membrane increases, until it finally encroaches upon the nasal chambers at every point. In addition to the nasal obstruction there is a hypersecretion of mucus. Opposite changes occur in atrophic rhinitis, such as thinning or atrophy of all the structures, with enlargement of the nasal cavities. The nasal mucosa is coated with thick, yellowish-green, decomposing crusts, which emit a characteristically fetid odor, and the frontal, ethmoid, or other accessory sinuses may, by an extension of the inflammation from the nasal chambers, be invaded by mucopurulent inflammation. The atrophic process does not affect the glandular structures of the upper third of the nose, and this fact explains the most unpleasant feature of the affection—namely, the horrible secretion.

Etiology.—Frequently occurring attacks of acute rhinitis may produce the chronic form, and syphilis and, less commonly, tuberculosis are also among its causes. Abel[1] regards atrophic rhinitis as infectious, claiming that the cause is the *bacillus mucosis ozenæ*, which resembles closely the pneumobacillus, but is distinguishable from it.

Symptoms.—(*a*) In the *hypertrophic* form nasal respiration is impeded, owing to the hypertrophy of the turbinated bodies. The sense of smell is impaired, and there is a discharge of secretion from the nares, particularly the posterior, inducing "hawking." The diagnosis is set at rest by a rhinoscopic inspection of the parts. While this is a common affection everywhere, it is wellnigh universal in this country.

(*b*) In chronic *atrophic* catarrh there is some degree of nasal obstruction, occasioned by the presence of the thick crust, but the most conspicuous symptom is the disgusting odor, which makes the patient repellent in society. The sense of smell is lacking. After cleansing the membrane the rhinoscope will show the nasal chambers to be unduly capacious.

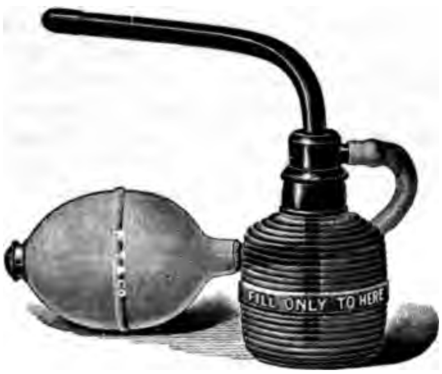

FIG. 39.—Apparatus for cleansing the nasal passages in chronic rhinitis.

Treatment.—(1) **Chronic Hypertrophic Rhinitis.**—The treatment is divisible into *general* and *local.* The physician should procure an environment for his charge most favorable for promoting the general nutrition, which is often below the health-standard. The selection of a suitable climate, then, forms an important part of the management, and a residence in a locality that possesses a mild, equable, comparatively dry and pure atmosphere is to be advised and encouraged. Various tonics may then be demanded by the general condition of the patient, and strychnin and electricity are useful in restoring the loss of power in the contractile elements of the intercellular walls.

Local measures are employed to facilitate thorough cleanliness and disinfection of the affected parts, though in incipient and mild cases energetic treatment is scarcely needful. The best method of cleansing the nasal passages is by means of the coarse spray (Fig. 39). The apparatus of Lefferts is also to be employed when the secretion is inspissated or tightly adherent. An excellent combination for use in this manner is the following:

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[1] *Zeit. f. Hyg. u. Infektionskrank.*, Bd. xxi. H. 1.

℞. Sodii biborat.,		
Sodii bicarb.,	*āā.* ʒj	(4.0);
Acid. carbolici,	gr. viij	(0.518);
Listerin.,	℥j	(32.0);
Aquæ destillat.,	q. s. ad ℥iv	(128.0).—M.

Sig. Use as a spray three times daily.

It is often desirable to use warm or even hot liquids, in which case the application is made by the use of the anterior and posterior nasal syringe. Powders are harmful, and, as the nasal douche is dangerous in unskilled hands, these should both be abandoned.

In hypertrophic rhinitis the obstruction to nasal breathing is to be removed, and to accomplish this caustics (chromic, glacial acetic, and nitric acids) are used, of which the most efficacious is chromic acid. This should be applied by means of a pointed glass rod, the application being followed by a sloughing away of the diseased tissues. Among other modes of removing the nasal obstruction that may be mentioned are the galvano-cautery, the thermo-cautery, and the cold-wire snare; these modes, however, are practised chiefly by the specialist.

(2) In **atrophic rhinitis** a cure is to be despaired of, but the patient can be rendered free from the offensive discharge, and hence to a great degree comfortable. As this is often but an advanced stage of hypertrophic nasal catarrh, the general treatment is similarly directed: it is therefore well to overcome, as far as possible, by a mental stimulus, the depressed mental state due to the fetor. If the diathesis be tuberculous, cod-liver oil, iron, arsenic, and strychnin, together with a generous diet, are to be advised. If syphilis is associated, appropriate measures must be instituted. Moreover, since a subject of atrophic rhinitis is a fertile source of atmospheric contamination, his living and sleeping apartments must be highly ventilated.

Local Measures.—An antiseptic spray of Seiler's or Dobell's solution, and oiling the nasal cavities, are measures to be first tried. If they prove non-efficacious, the crusts may then be removed with a cotton applicator coated with a solution of hydrogen peroxid. We may then use a spray of liquid albolene and menthol; this serves not only to lubricate, but to supply moisture, both of which are important therapeutic indications. Small ulcerations occur in this affection and induce oft-repeated epistaxis; consequently, an attempt should be made to heal the latter and to obtain an even, moist surface. To accomplish this the method of Clarence C. Rice may be followed—*i. e.* to rub the ulcerations thoroughly by means of a cotton-carrier with a small hard pledget of cotton moistened with listerin or borolyptol for a few seconds at a time. These antiseptic frictions are made at intervals of two or three days for two or three weeks.

AUTUMNAL CATARRH.

(*Hay Asthma; Hay Fever.*)

By this term is meant a form of asthma that occurs exclusively during the warm season (spring and late summer, particularly).

Etiology.—The **direct causes** are the odorous principles given off from certain plants (the pollen of the *Anthoxanthum odoratum*, of the rose, etc.), and inorganic dusts of various sorts. In some instances it appears to arise without obvious exposure to a special irritant—for example, it may be excited by strong emotional disturbance.

Predisposing Factors.—The male *sex* suffers more frequently than the female. *Age* has a slight though decisive influence, more than 33 per cent. of the cases occurring before the twentieth year. Often some *abnormality of the nasal passages* (*e. g.* a polypoid tumor, hypertrophy of the mucosa, a deflected septum) acts as a source of reflex irritation. *Heredity* is among the potent contributing causes. The inhabitants of cities are more liable than those in rural districts, though the air of agricultural regions intensifies the condition. Perfect *immunity* is enjoyed by the dwellers in certain climates—chiefly mountainous and marine.

Symptoms.—The symptoms are (*a*) local and (*b*) general.

(*a*) **Local.**—Hay fever has an *abrupt onset*, and the attacks return annually at or about the same time. The *invasion* is marked by pronounced coryzal symptoms, with much sneezing, stoppage of the nasal passages, copious rhinorrhea, the discharge being thin and watery as a rule, and rarely mucopurulent. Suffusion of the eyes, with itching of the lids and free lacrymation are constant features; the decided itching sensation of the palate and pharynx is also at times a very distressing symptom. The sense of smell may be lost, and taste and hearing are often impaired.

The *course* as regards the local symptoms is marked by alternate amelioration and aggravation of the symptoms, the exacerbations being due to exposure to the open air, especially in changeable weather. Later the catarrhal process invades the bronchi, and cough and asthmatic seizures appear, these often becoming very distressing.

(*b*) **General disturbances** are varied, and comprise subjective sensations, such as anorexia, insomnia, lassitude, and chilliness alternating with slight feverishness.

The *course* is from four to six weeks, and cases that develop in August are terminated by the occurrence of a decided frost. Wyman also describes the "rose cold," which comes on in the spring.

Diagnosis.—The recognition of hay asthma is unattended with difficulty, provided that such facts as the time of its occurrence and its annual periodicity are carefully noted. The sudden onset of severe coryza-like symptoms in a manifestly neurotic person, particularly in the month of August, should always direct the attention to this disease.

Prognosis.—This is favorable both as to life and length of days, though a permanent cure is a rare event unless permanent removal from the influence of the specific causes can be effected.

Treatment.—Whenever possible the patient should travel till he finds a locality in which he ceases to suffer, and subsequently he should there spend the period of annual attack, and by these means escape the exciting causes. The Adirondacks and White Mountains usually bestow immunity. If the patient cannot make the necessary change, the general nutrition is to be improved by the use of such measures as phosphorus, strychnin, quinin, and arsenic. Much is to be gained, moreover, by hygienic means, especially avoidance of physical and mental overwork and the adoption of a proper mode of life.

The local symptoms demand the topical application of various agents to the nasal chambers, such as cocain hydrochlorate solution (1 per cent.) applied on cotton with a probe, followed by a 4 per cent. solution of antipyrin, prolong the palliative effect (Gleason). Hollopeter has had uniformly good success, in over 200 cases, by daily *sterilizing* the nasal chambers by means of the ordinary Dobell's solution used first with an atomizer; then swabbed thoroughly over the whole naso-pharynx. The membrane is then dried and the nose loosely plugged for a short time with cotton saturated with a mild solution of menthol in albolene. The local symptoms are also greatly benefited by the internal use of atropin, which allays the irritability of the mucous membrane involved and diminishes the rhinorrhea, thus indirectly mitigating the constitutional disturbances and sometimes directly relieving the asthmatic paroxysms. When given internally the dose should not exceed gr. $\frac{1}{300}$ (0.0002), to be repeated every hour till dryness of the throat appears.

My own best results have been derived from the hypodermic use of this drug (gr. $\frac{1}{200}$—0.0003) at intervals of three to four hours till the desired effect is produced. Gleason has obtained satisfactory results from nitro-muriatic acid freshly prepared (dose ♏v t. i. d. after meals in a tumbler half full of water). Recently the internal use of 5 grain tabloids of suprarenal extract has met with fair success in the hands of S. Solis-Cohen and others. "Adrenalin," the alkaloid, in aqueous solution, applied to the nasal mucosa, promises to be highly serviceable.

EPISTAXIS.

(*Nose-bleed.*)

Etiology.—The causes of nose-bleed are various, and a convenient grouping is the following: (*a*) Affections of the nasal mucosa (*e. g.* ulcer, polypi, intense hyperemia). (*b*) Injuries, either external, as from a blow, or internal, as from plugging with a foreign body, nose-picking, etc. In this category may also be included epistaxis due to fracture at the base of the skull. (*c*) Acute infectious fevers, particularly typhoid (at the onset) and influenza. (*d*) Chronic affections, such as pernicious anemia, leukemia, and the hemorrhagic diathesis or hemophilia. (*e*) Vicarious menstruation. (*f*) Rarefaction of the air. (*g*) Plethora; here may be mentioned cerebro-congestion with intense headache. (*h*) Severe over-exertion. (*i*) Frequent epistaxis may be caused by arteriosclerosis even in the earlier stage while yet amenable to treatment.

Symptoms.—Except when due to traumatism the blood usually drops slowly from one and occasionally from both nostrils. Rarely, the blood may flow as a continuous stream or the nares may present a projecting coagulum. The blood may also gravitate into the pharynx and be coughed up, or it may be swallowed and vomited. A rhinoscopic examination often reveals the source in cases in which a previous diagnosis of hemoptysis or hematemesis has been made.

The immediate results of nose-bleed are weakness and a moderate anemia, but these are not prolonged, as a rule. Cases arising from fracture at the base of the skull will generally prove fatal.

Treatment.—A careful search for a local cause is especially demanded in cases in which there are frequently recurring attacks. In most cases a spontaneous arrest occurs, but if not, a resort to simple household measures, such as the application of ice to the nose or to the back of the neck, holding the hands up, or the injection of very cold or very hot water into the nares, are to be encouraged. Various astringents (tannic acid, acetate of lead, alum, zinc) may be employed, and a saturated solution of antipyrin is also highly praised. When an ulcerated bleeding point can be reached, there may be applied to it a solution of chromic acid or it may be cauterized by solid silver nitrate. Prolonged pressure applied upon the facial artery as it passes over the inferior maxilla may be efficacious. The late D. Hayes Agnew successfully employed a bougie made of a long strip of the rind of bacon, "passing it through the nostril and allowing it to stay there some time." A solution of gelatin may be injected into the nostril. I have little confidence in internal astringent remedies. The oil of origanum, administered in large doses, has seemed to do good in a few of my own cases, but in obstinate cases the posterior nares should be plugged.

II. DISEASES OF THE LARYNX.

ACUTE CATARRHAL LARYNGITIS.

(*Acute Endolaryngitis.*)

Definition.—An acute catarrhal inflammation of the larynx, characterized by cough, hoarseness, and painful deglutition.

Pathology.—The anatomic changes present during life are all lacking *post mortem*. The laryngoscopic appearances will be given below.

Etiology.—Acute laryngitis may be a primary affection—and particularly *laryngitis sicca* (Molinie)—but oftener it is associated with and secondary to catarrh of the nose and nasopharynx. Wright attributes laryngitis sicca to the coccus of Löwenburg.

Catarrhal laryngitis has for its chief direct causes traumatism, exposure to cold and dampness, the inhalation of irritating vapors or gases, rheumatism (rarely), and the corrosive effect of certain poisons and hot fluids. A certain degree of predisposition is engendered by immoderate smoking, particularly by the cigaret-habit, and by the use of concentrated alcoholic drinks. These agencies induce hyperemia of the laryngeal mucosa, which is easily converted into active inflammation. Acute laryngitis is often associated with acute infectious diseases.

Symptoms.—There are two conspicuous symptoms—*alteration in the voice* (hoarseness) and *cough*. At first there is merely a huskiness of the voice, but later there may be pronounced hoarseness or even complete aphonia. The cough is dry and characteristically painful until secretion is free. In the early stages the patient complains of sensations of tickling or the presence of some small object in the larynx, causing a frequent desire to clear the throat. In severe instances deglutition is painful. Edema of the larynx may tend to supervene and cause intense dyspnea, with a feeling of distressing oppression. There is, as a rule, a slightly elevated temperature.

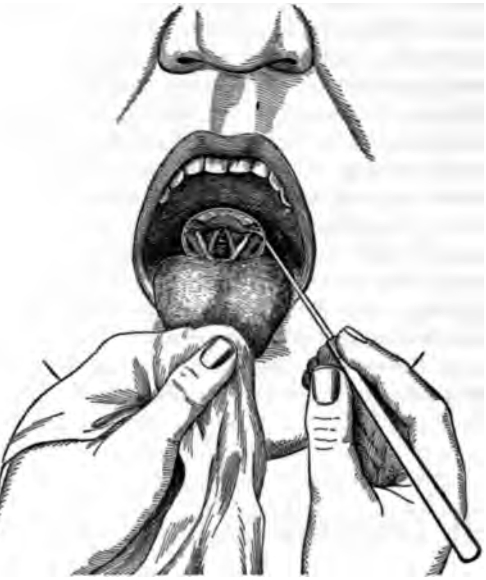

FIG. 40.—Method of making a laryngoscopic examination.

The patient is placed in front of the operator, on an arm-chair, with the back of the chair high enough to afford his head a comfortable rest, and with the source of the light over the right shoulder. The operator then adjusts the head-mirror (the fixed apparatus), warms the throat-mirror over a light sufficiently to prevent the moisture of the breath from being deposited upon it, and touches the hand with the mirror before passing it into the mouth, so as not to use it too hot. The patient's tongue is then protruded, and by means of a napkin is seized between the thumb and the fore-finger and drawn well forward to lay the fauces open to observation. The throat-mirror is then held in the right hand in the same way as one holds a pen. "Finally, it is introduced into the mouth, its handle being inclined downward and outward, its base being parallel with the dorsum of the tongue; it is then passed backward without altering this relation until the edge of the mirror nearly touches the soft palate, the shaft of the mirror in this movement striking the angle of the mouth as a resting-place and fulcrum. The subsequent movement consists in turning the mirror by twisting its shaft between the fingers until it is inclined at an angle of 45 degrees to the line of vision; then it is carried backward and downward until the uvula rests upon its posterior surface, when it is lifted boldly upward and backward until its lower edge comes entirely into view again and rests firmly against the posterior wall of the pharynx. The patient should then be directed to sound in a somewhat high key '*a*,' which lifts the larynx and at the same time the epiglottis, and exposes and brings into view the laryngeal cavity" (Bosworth).

It is important that the mirror itself should be kept in the median line, with its plane always at right angles with the field of vision, as shown in the illustration. In making a laryngoscopic examination we note any abnormalities of color-appearance (the natural being a rose-pinkish tint), of the outline of the different parts, and the deviations from the symmetrical movements of cords, if any, etc.

The laryngeal mirror brings to view a characteristic picture—a swollen, tumefied, and reddened mucosa. These changes affect the vocal cords (whose pearly-white appearance is now lacking) and the ary-epiglottidean folds. It is usual to note also redness and swelling of the epiglottis above and of the trachea below. After secretion has occurred a mucoid covering in streaks or patches is noticeable.

Diagnosis.—This is easy in the presence of marked hoarseness, dry cough, and the image afforded by the laryngeal mirror (Fig. 40). In very early life the larynx cannot be successfully examined; still, *laryngismus stridulus* (owing to the absence of fever, coryza, etc.) could hardly be mistaken, as has been supposed, for acute catarrhal laryngitis. The same is true of *membranous laryngitis*, if we bear in mind the characteristic local features and the more intense constitutional disturbances of the affection.

Treatment.—The physician must enjoin against the use of the voice. The very young and the aged should, in severe or even moderate cases, be kept in bed, and should occupy a single apartment in which the atmosphere is uniformly moist and warm, the temperature ranging from 75° to 80° F. (23.8°–26.6° C.). Inhalations of moist air or steam are of great service, and I have long been in the habit of recommending the following simple apparatus and method of carrying out this mode of treatment: An ordinary tin cup, small pitcher, or other vessel is filled with boiling water to which 1 or 2 drams (4.0–8.0) of the compound tincture of benzoin have been added; the steam is then collected by inverting over the vessel an ordinary funnel. The patient is allowed to inhale the steam by placing the mouth over the narrow neck of the funnel above, or a piece of rubber tubing may be attached to the end of the funnel that is uppermost.

Steam atomizers admirably meet the necessities of the case; and in the case of children the vapor of benzoin, eucalyptol, and other equally sedative and stimulating substances may be diffused in the air of the sick-room. Concentrated solutions or insufflations of powders are not without harmful influence, and neither the cotton-carrier nor the mop should be allowed to enter the larynx in this affection. The external application of the ice-bag or cold compress tends to mitigate the inflammatory process and to obviate spasm.

The *general* treatment differs with the special stages of the complaint. If the case is seen early, a full dose of quinin (gr. xij–xvj—0.777–1.036) may serve to successfully abort the attack, and, in conjunction Dover's powder (gr. v–x—0.324–0.648) may be prescribed. Codein sulphate may be given at prolonged intervals during the attack, and frequently at night, to allay cough; this remedy may be combined with ipecac, aconite, and liquor ammonii acetatis to facilitate secretion and render the cough humid. If we except the abortive measures, the constitutional is wholly inferior to the topical treatment of this variety, though the existence of any particular diathesis may require special internal remedies.

CHRONIC LARYNGITIS.

(*Chronic Endolaryngitis.*)

Pathology.—The laryngeal mucosa is thickened and somewhat reddened, and erosions amounting to superficial ulcerations are rarely seen. A prominence of the mucous glands, especially of the ventricles and epiglottis, is noticeable. Fine villous projections from, and nodular swellings in, the vocal cords are among the rarer morbid changes. Minute vesicles may arise upon the surface (*herpetic laryngitis*).

Etiology.—Oft-repeated acute attacks frequently cause chronic laryngitis, and the long-continued use of the voice (as in public speaking or singing), the inhalation of an atmosphere laden with mildly irritating impurities (tobacco smoke, etc.), and an immoderate indulgence in alcoholic stimulants, respectively or unitedly, predispose to, if they do not excite, the disorder.

Symptoms.—As in the acute form, *hoarseness* and *cough* are the two especially prominent symptoms. The former may be so slight as to present merely a rough tone, or it may involve an almost total loss of voice. The cough shows similar variations in severity, sometimes consisting of a short hack, and again occurring in spasmodic and ringing paroxysms, due to a sense of tickling in the larynx. There may be a small amount of mucous or muco-purulent expectoration, but for prolonged periods the cough may be dry and ineffectual. *Local pain* and discomfort sometimes supervene, and are excited generally by attempts at speaking or singing—events that aggravate all the other symptoms. To complete the diagnosis, the laryngeal mirror is required to show a swollen and slightly red membrane, with a distention of the mucous glands in the immediate vicinity of the epiglottis and ventricles, and occasionally superficial erosions.

Prognosis.—This is unpromising as to complete recovery, although it presents no grave dangers. It is incurable in those instances in which the causal influences cannot be removed, and in all cases in which the patient fails to lend hearty co-operation.

Treatment.—This is (*a*) *hygienic* and (*b*) *medicinal.* (*a*) The sanitary measures embrace preventives that are directed to the removal of all the etiologic factors, whether merely predisposing or exciting. The voice demands rest and the prohibition of smoking and the use of alcoholics in excess, and the patient must also avoid the close, contaminated air of the crowded hall, theatre, and like places. In addition, a tonic regimen, with a view to energizing the nutritive processes, is to be encouraged. In many instances the environment is best arranged with reference to the commonly associated conditions—especially the morbid processes in the nasal and naso-pharyngeal cavities. "A sea-voyage or residence at the sea-shore is, in the large majority of instances, productive of good, and the effects of surf-bathing are often magic" (Mackenzie). My own practice has been to send subjects of chronic laryngitis to pine-forest resorts at low elevations that afford a pure, equable, and somewhat stimulating atmosphere, and I have found that in many cases the selection of a proper climate constitutes the most important part of the treatment. (*b*) The *medicinal* treatment is both local and general. The latter should include creasote, cod-liver oil, and other tonics. Expectorants are of little if any value. The *local* measures, however, are important. Moderate exposure of the neck and daily ablution with cold water are to be advised, and attention to the nose and naso-pharyngeal cavity is of prime importance.[1]

A long list of applications to the larynx from within, including local astringents, disinfectants, and alcoholics, might be enumerated. Of astringent solutions, however, the best are tannic acid (1–2 per cent.) or alum (.5–1 per cent.) and zinc sulphate (3–5 per cent.). These may be sprayed into the larynx by means of a compressed-air machine with spraying-tubes, although all of the different kinds of inhaling apparatus more commonly used will answer the purpose. If the ordinary hand-atomizer be used, the patient should be taught to draw the vapor into the larynx by gentle and frequent acts of respiration. Disinfectants, such as creasote, potassium chlorate (the latter if ulcerations be present) in solutions of suitable strength, may be used in like manner. I can confidently advise as useful alteratives both iodin and silver nitrate, commencing with a weak solution of the latter (gr. v–ʒj—0.324–4.0), and the strength being gradually increased until the maximum strength that can be endured without distress is reached (gr. xx–ʒij—1.296–8.0). These topical applications should be made directly with a cotton-carrier or brush at intervals of three or four days, preceded by the use of a cleansing spray. Many astringent and sedative lozenges are to be found in the market, but they are only slightly palliative in their effects, and their prolonged use tends to excite gastric disturbance. I am unalterably opposed to the insufflation of powders, believing that they are capable of augmenting the laryngeal irritation and of adding fresh irritation in adjacent parts, particularly in the tracheo-bronchial tract.

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[1] J. C. Wilson's *American Text-book of Applied Therapeutics*, p. 791.

SPASMODIC LARYNGITIS.

(*Laryngismus Stridulus; False Croup.*)

Definition.—An affection peculiar to children, chiefly of nervous origin, though also, according to Strümpell and others, often associated with acute catarrhal laryngitis.

Etiology.—The affection is almost solely limited to children between six months and five or more years of age. It is sometimes excited by strong passion or emotion, and it may be associated with tetany. Rachitic subjects are peculiarly liable. The causes of spasmodic croup are in great part those of acute laryngitis.

The mode of action of the direct causes is unknown, but the spasm of the adductors that causes the urgent dyspnea is probably reflex and due to peripheral irritation.

Symptoms.—Two clinical varieties are to be distinguished: (1) That in which the larynx is free from catarrhal inflammation, or the *purely nervous type.* This is especially characterized by sudden brief attacks of dyspnea, either by day or night (often on awakening), that terminate in a high-pitched crowing inspiration ("child-crowing"). The face during the spasm is cyanotic. General convulsions have been noted, but there is neither cough, fever, nor hoarseness. The attacks may be frequently repeated within a single day.

(2) Spasm of the larynx, *associated with mild catarrhal laryngitis.* The attacks generally begin suddenly, about midnight or toward morning on awakening from a sound sleep. Positive evidence of the affection is afforded by the croupy, ringing cough, combined with the hard, stridulous breathing. An approaching spasm may be announced by a harsh

cough and slightly stridulous breathing in the sleeping child. During the attack the countenance may be cyanotic and the breathing most distressing, but these and the above-mentioned severer symptoms generally cease abruptly in an hour or two, and the child resumes its slumber. In my experience the attacks have been repeated for two or three nights in succession, and rarely oftener except in the severest cases. Not infrequently the child manifests the symptoms of mild catarrhal laryngitis between the attacks. A brassy, croupy cough may also attend.

Diagnosis.—*Membranous laryngitis* may be mistaken for spasmodic croup. The development of the dyspnea, however, is more gradual, is without intermission, and without relation to the period of the day. Albuminuria and a false membrane in the throat or nares are usually present in *laryngeal diphtheria.*

Prognosis.—Although the appearance of a paroxysm is alarming, the disease is practically free from danger.

Treatment.—1. The treatment of laryngismus stridulus is quite similar to that of infantile convulsions. A warm bath at a temperature of 98° to 105° F. (36.4°–40.5° C.) is the best means of breaking up the spasm. While in the bath cold sponging of the back and chest is serviceable. The finger may be passed into the fauces, and should the epiglottis "become wedged in the chink of the glottis, it must be released by the finger." After the attack active treatment should be directed at the discoverable causes, and I have been in the habit of giving

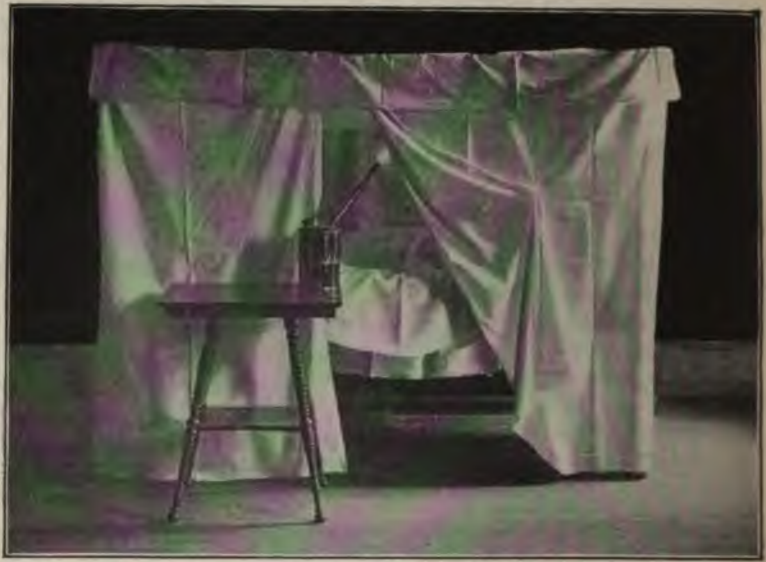

FIG. 41.—Croup-kettle in use.

Four upright rods (5.7 inches in length) are fastened to the legs of the bedstead by a wire or string. Two side-rods are tied on the uprights, and two end-rods (length dependent on width of bed) rest upon the side-rods, These rods form a complete framework for the sheets to hang upon. Four sheets are required (11-4 size)—three to cover the ends and sides, and one to be placed on top. One side should be completely closed, while the opposite is to be left open for ventilation or to be adjusted according to circumstances.

mall doses of the bromids thrice daily, together with warm cod-liver l inunctions, with striking effect.

2. In spasmodic croup an emetic is to be given at once, the best being a mixture of alum and syrup of ipecac, of which the dose is ʒj 4.0), to be followed by irritation of the fauces with the finger in order o facilitate emesis. In severe paroxysms a hot bath may be given to id the emetic. In case the dyspnea is not checked by the above measures, chloral hydrate may be exhibited by enema (gr. ij–v; 0.129–.324) or a whiff of chloroform may be given. The local application of old (ice-collar, ice-water cloths) is useful, and sinapisms placed around he throat and over the chest also tend to arrest the spasm. I am convinced that the use of steam-inhalations from the so-called croup-kettle Fig. 41) is of signal service, and should be more widely employed, particularly when it is inconvenient to use the hot-bath.

Between the paroxysms the patient should receive a mild laxative, such as calomel or castor oil, and, in addition, the treatment appropriate in acute catarrhal laryngitis. To prevent recurrences an environment calculated to increase the nervous tone of the child is to be procured, and it is especially advisable to accustom him to the outer air, though protected by suitable dress and without undue exposure to draughts.

EDEMATOUS LARYNGITIS.

Definition.—An infiltration of the mucous membrane of the larynx with serum. In most cases it is a true inflammatory edema.

Etiology.—Two chief classes of causes are operative: (1) Those that excite inflammation. The condition may complicate acute laryngitis, though oftener it appears in chronic affections of the larynx, and particularly if ulceration be associated (*e. g.* tuberculosis, syphilis); it may also appear in connection with certain infectious diseases (erysipelas, diphtheria, typhoid fever). The inflammation inducing the edema may extend from adjacent parts, as the neck, pharynx, and other organs. (2) Factors that tend to excite dropsical effusion. These may be general, as Bright's disease, heart-affections, etc.; or they may be local. Among the latter are enlargements of the cervical and mediastinal lymphatics, aneurysm of the arch of the aorta, thyroid tumors, etc.—*i. e.* conditions that exercise pressure upon the jugular veins. Rice, who studied 41 cases, thinks it doubtful whether edema of the larynx ever occurs from simple catarrhal inflammation.

Symptoms.—In acute cases the initial disturbance is both sudden and severe. There is *dyspnea* that tends to increase rapidly, accompanied by a husky, suppressed voice, with augmenting obstruction. The *respirations* may become stridulous, but there is no cough. The laryngoscope reveals marked swelling of the epiglottis and of the ary-epiglottic folds, and rarely the swelling occurs in or even wholly below the vocal cords. The inserted finger may detect the swollen epiglottis, which may also be seen if the tongue-depressor be used.

Diagnosis.—This can be made with ease from the rapidly developing dyspnea soon reaching the climax, the absence of cough and hoarseness, and by the use of the laryngoscope. In cases in which the epiglottis can be felt or seen a laryngoscopic examination is superfluous.

The **prognosis** is decidedly unfavorable except in the event of early operative interference.

Treatment.—If of inflammatory origin, the ice-bag should be applied to the larynx, and ice should be allowed to constantly dissolve in the patient's mouth. Local depletion, preferably by leeching the front of the neck, is also to be tried, and Levy and Laurens[1] record a case in which a cure followed this measure. If intense dyspnea tends to persist, scarification of the edematous parts with a curved bistoury, the point of which is covered with adhesive plaster, must be promptly instituted, and, if asphyxia threatens, tracheotomy must immediately be performed. Dropsical edema demands scarification, and, if relief does not follow, tracheotomy.

TUMORS OF THE LARYNX.

THESE may be either benign (fibroma, myxoma, lipoma, chondroma, adenoma, angioma, cyst) or malignant (sarcoma, carcinoma). Of these, papillomata or papillomatous fibromata occur most frequently, especially in infancy. Navratil[2] records 42 cases of multiple laryngeal papillomata in children whose larynges were extensively filled. These growths may also occur in chronic laryngitis, and, like other tumors of the larynx, they commonly spring from the vocal cords. Their shape, size, and tendency to pedunculation do not differ from their characteristics when noted elsewhere in the body.

Symptoms.—Small tumors may occupy the larynx without producing symptoms. The first feature then noted is *hoarseness*, which gradually grows worse and may end in complete aphonia. If situated in the upper larynx, *cough* is common, and when the tumor causes obstruction of the larynx *dyspnea* supervenes and tends to increase in severity. A mobile growth may cause sudden occlusion of the glottis, exciting orthopnea and threatening asphyxiation. To confirm the diagnosis a laryngoscopic examination is required.

The **prognosis** is favorable in the benign, but unfavorable in the malignant forms.

Treatment.—This is altogether surgical, though Delavan states that 3 cases of papilloma have been cured by frequently repeated sprays of absolute alcohol. Curetting is often followed by a recurrence, while laryngo-fissure and thorough removal of the growths restore speech and prevent recurrence.

III. DISEASES OF THE BRONCHI.

CATARRHAL BRONCHITIS.

(*Tracheo-bronchitis.*)

Definition.—A catarrhal inflammation of a part or the whole o the mucous membrane of the bronchial tubes. The mucosa of tl rachea is also involved to a greater or lesser extent, and hence the term racheo-bronchitis is quite appropriate, being descriptive of the seat and haracter of the disease. Involvement of the bronchioles may also take place, but this does not occur without an involvement of the corresponding alveolar structure, the condition being then, with propriety, termed "broncho-pneumonia." Hence the term "capillary bronchitis," which is still often employed to describe the latter condition, is not pertinent. A certain class of cases is met with, however, in which the catarrhal inflammation, as the result of downward extension, implicates the smaller bronchial tubes without involving the bronchioles; to such the term "capillary bronchitis" might be appropriately given.

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[1] *Arch. gén. de Méd.*, Dec., 1895.

[2] *Berl. klin., Woch.*, Mar. 9, 1896.

The disease may be acute or chronic, both of these forms occurring either as a primary or secondary affection.

ACUTE BRONCHITIS.

Pathology.—The portions of the mucous membrane of the trachea and bronchi that are implicated become reddened and swollen; they are covered with mucus and mingled with epithelial cells, and later muco-pus. Some of the smaller bronchial tubes are dilated. The mucous glands are swollen.

The histologic changes may be briefly stated as follows: desquamation of the ciliated epithelium, edema and swelling of the submucosa, and, in the severer grades, infiltration of the latter with leukocytes.

Etiology.—With rare exceptions tracheo-bronchitis is produced by the direct extension of a catarrhal inflammation from the nares, pharynx, and larynx. Rarely the bronchi are the seat of primary acute catarrh, and in some of the latter instances the upper air-passages are implicated secondarily, constituting a reversal of the usual direction of extension above referred to.

The immediate causes are mechanical, chemical, and biologic irritants, which act directly upon the tracheo-bronchial mucosa; and that bronchitis is frequently due to infection at a time when the resisting power of the system is reduced there can be little doubt. The circumstances disposing to bronchitis are numerous, those pertaining to the individual being—(1) Age, the old and very young being most liable; (2) Debility; (3) Occupation, as in certain trades that expose to irritating vapors and sedentary pursuits. Among the external conditions are—(1) Climatic factors, particularly variability of temperature and humidity; (2) Seasons of the year. "Catching cold" often results from exposure during the spring and autumn months. These two conditions depend substantially upon the same factors. (3) Epidemic influence, which may be independent of influenza.

Acute tracheo-bronchitis arises as a secondary condition in a great variety of diseases, as, for example, the exanthemata and other acute infectious diseases (typhoid fever, measles, whooping-cough, influenza, etc.). As shown elsewhere, among this class of diseases the bronchitis may be dependent upon the primary infectious process; but in many others it is due either to the inhalation of pathogenic irritants or to the retention of bronchial secretions that are apt to accumulate and decompose with resulting bronchitis. The accidental inhalation of particles of food and saliva may also lead to secondary bronchitis, or the condition may be secondary to chronic affections (*e. g.* Pott's disease, Bright's disease). Among the toxic causes the poison of uremia must be embraced.

Symptoms.—Bronchitis of the larger tubes, which extends down to about the second division of the bronchi, is spoken of usually as a "cold." In such cases the onset is marked by recurring sensations of *chilliness*, and by *coryza*, slight *sore throat*, and *hoarseness;* while in young and feeble children *convulsions* may occur early. Mild *febrile symptoms* may appear, the temperature ranging from 101° to 103° F. (38.3° to 39.4° C.), with slight acceleration of the pulse; and there may be langour and aching in the limbs and lumbar region. With the fully-developed attack substernal soreness, sometimes even *pain*, is experienced, especially on coughing, and the pain may be referred to the intercostal muscles and the line of insertion of the diaphragm. The respirations are increased in frequency, but there is no dyspnea. There may be thoracic oppression and discomfort until the bronchial secretions become free, and there is a *cough* which is at first dry and hard. It often manifests itself in longer or shorter paroxysms, particularly on lying down and on rising after a full night's sleep. At the end of one, two, or more days the cough is moist and attended with an expectoration which is at first *mucoid* and scanty, often viscid, then *muco-purulent* and free; later still it is sometimes distinctly *purulent*. With free expectoration comes relief to the patient. Histologically, the sputum consists mainly of pus-corpuscles with large cells, in which may be seen the so-called myelin droplets of Virchow and carbon particles.

Physical Signs.—Upon laryngoscopic examination the mucous membrane of the larynx and trachea may be seen to be reddened and covered by more or less secretion.

Inspection and *palpation* of the chest are negative, except when the finer tubes become implicated or fever is present, in which case the respirations may be observed to be slightly accelerated. In children the increased rapidity of the respirations is more common and reaches a higher degree. Bronchial fremitus may sometimes be felt. *Percussion* yields negative results, save in very rare instances, in which there occurs a decided accumulation of secretion in the tubes, when there may be found impairment of resonance posteriorly below the scapulæ. *Auscultation* usually renders audible a harsh respiratory murmur, and less frequently piping, sibilant, and sonorous râles. In the advanced stage (with relaxation of the mucosa) large and medium-sized mucous râles are frequently present, but are by no means always discernible. The râles appear in different seats from time to time, and after coughing may altogether disappear, but only to reappear later.

The **prognosis** varies with the previous constitutional state of the individual. In healthy adults, after a period ranging from a few days to two weeks, the fever subsides, but the cough, though less marked, and the expectoration usually continue for a variable length of time. In old persons, in those greatly debilitated, and in those of a gouty or tuberculous diathesis the cases pursue a more protracted course. There is also in these subjects a tendency on the part of the catarrhal process to extend downward until the finer tubes are implicated. Under these circumstances life may be endangered, and even, rarely, terminated. In the old the secretions are imperfectly expectorated; they gravitate to the most dependent parts and induce bronchiectasis. In young children this downward extension of the affection, with resulting broncho pneumonia and areas of collapse in consequence of dilatation and occlusion of the bronchioles by muco-pus, is a not uncommon and serious event (*e. g.* in measles, whooping-cough, *vide Broncho-pneumonia*).

The **diagnosis** is reached without difficulty through the symptoms (slight fever, cough, and expectoration), the acute course, and the physical signs (harsh respiratory murmur, dry followed by moist râles, heard on both sides of the chest). The recognition of the long list of cases that constitute the secondary forms will be made easily possible by noting the circumstances under which they arise.

Differential Diagnosis.—Bronchitis can readily be separated from *pneumonia* and from *pleurisy with effusion* by its history, by its lighter course, and by the different character and general distribution of the physical signs, especially by the absence of the signs of effusion and consolidation.

When *broncho-pneumonia* develops in the course of bronchitis, dyspnea and fever are increased, cyanosis is present, and the general condition becomes much more grave. There are small patches that yield dulness on percussion, and broncho-vesicular breathing with moist rales can be detected on auscultation.

Bronchitis cannot be separated from the early stage of *whooping-cough*, but when the characteristic cough of the latter is heard all doubt vanishes.

The bronchitis of *measles* before the characteristic eruption appears is distinguished by the red spots upon the anterior half-arches of the soft palate.

Localized tuberculosis of the lung and *acute miliary tuberculosis* are apt to be confounded with bronchitis. The points of difference have been given in the discussion of the former diseases.

Treatment.—Not infrequently a "cold" passes through its several stages without rendering the patient ill enough to cause him to seek the advice of a physician, and there are many instances in which but little treatment is required, apart from the usual household measures and protection against cold and damp. If seen early, while the coryza is present, the attack may often be aborted by the use at bedtime of a Dover's powder in combination with quinin (gr. iv–viij—0.259–0.518); this may be seconded by a glass of hot lemonade, with or without a portion of whiskey, and either a hot bath or a mustard foot-bath. The following morning a saline laxative should be taken. To children a mild calomel purge followed by a dose of castor oil may be administered. The patient should be kept in a warm, moist, equable atmosphere—preferably in-doors—and during this period he should take divided doses of quinin for a day or two. If the above mode of treatment fail or if the patient does not come under observation early, the main objects of treatment should be (*a*) to render the secretions free, and (*b*) to hasten the expulsion of the sputum after it has been loosened. The first leading indication is to be met by the use of diaphoretics, diuretics, and relaxants. The subjoined formula combines these classes of agents, and will be found to be highly serviceable:

℞. Potassii citrat.,	ʒvj	(23.3);
Liq. ammonii acetat.,	℥v	(148.0);
Spt. æth. nit.,	℥j	(30.0);
Vini ipecac.,	ʒij	(2.0);
Syr. pruni virg.,	q. s. ad ℥viij	(236.0).—M.

Sig. ℥ss (2.0) in water every two hours until the secretions are loosened.

If the temperature in any given case be maintained at a considerable elevation, such as 102°–103° F. (38.8°–39.4° C.) or over, tincture of aconite (♏xvj—1.065) may be added to the above mixture; and if there be present much tickling with distressing cough, due to irritability of the affected mucosa, codein (gr. ij–iij—0.129–0.194) may be added to the same. For the incessant irritative cough which is present in severe forms of catarrh opium alone is really effective. When the above prescription is not productive of free secretion and troublesome cough continues, I employ the following:

℞. Ammon. muriat.,	ʒv	(20.0);
Codeinæ,	gr. iv–vj	(0.259–0.388);
Spt. junip. co.,	℥ss	(16.0);
Mist. glycyrrh. comp.,	℥iiss	(80.0);
Syr. pruni virg.,	q. s. ad ℥iv	(120.0).—M.

Sig. ʒj (4.0) every two hours.

Apomorphin is also excellent as a soothing relaxant in doses of gr. $\frac{1}{20}$ to $\frac{1}{10}$ (0.003 to 0.006) every two hours. Mild counter-irritation by means of mustard-paste, followed by the application of iodin once daily, is also helpful. The patient should keep to his room, in which the atmosphere should be kept moist and of even temperature. (*b*) The expulsion of the sputum may demand stimulating expectorants, though rarely. It is to be recollected that when the tracheal secretion becomes copious the period of convalescence is usually reached, and stimulating expectorants are then entirely unnecessary. When, on the other hand, the cough is no longer dry, and on auscultation the râles are found to be moist, and whilst, at the same time, the expectoration is expelled with difficulty, or if the bronchitis tends to become chronic, then such stimulating expectorants as senega, squills, and ammonium muriate are to be employed. In cases in which expectoration continues to be too abundant terebene, tar syrup, and oil of sandal are to be resorted to.

Debility and secondary anemia must be speedily overcome by exhibiting quinin, bitter tonics, iron, and arsenic; and a suitable change of air often yields prompt and excellent results in protracted cases. The treatment of the various forms of secondary bronchitis will be considered in their appropriate connections in this work. In the aged the general strength must be maintained; the patient's position must be changed at short intervals and stimulants are usually needed.

Apart from the method above given, of attempting to abort the attack in children, acute bronchitis is in the main to be treated in the same manner as when it occurs in the adult. Opium, however, is to be used very sparingly, and generally in the form of paregoric. If the secretion is abundant and imperfectly raised, it is well to administer an metic, such as the wine of ipecac (ʒss–j—2.0–4.0), and repeat in ten minutes if necessary. If dyspnea be urgent and cyanosis be marked n the lips and finger-tips, a prompt emetic is then imperative in order o save life. A child suffering from acute bronchitis should be kept in bed until the fever subsides.

The diet during the dry stage should consist of liquid forms of nourishment, which should, for the greater part, be taken hot. After the "cold" has been loosened solid food should be resumed.

CHRONIC BRONCHITIS.

Pathology.—The lesions of chronic bronchitis manifest considerable variety both as regards their nature and extent. The epithelial layer is, in great part, missing, and sometimes the mucous membrane is quite thin. In consequence the longitudinal elastic fibers appear unduly prominent. The mucous glands and the muscular coat undergo atrophy in long-standing cases, and the bronchial tubes are dilated (*bronchiectasis*).

In another large group of cases the mucosa is irregularly thickened or infiltrated and granular. Small ulcers corresponding to the mucous follicles are common, and almost constantly emphysema develops in consequence of secondary changes in the vesicular structure.

Etiology.—Chronic bronchitis may either be *primary* or *secondary*. The affection is, however, almost always a secondary one, and, though sometimes the result of repeated attacks of acute bronchitis, it is oftener caused by certain chronic complaints and certain diatheses, as chronic alcoholism, rheumatism, gout, syphilis, pulmonary tuberculosis, and pulmonary emphysema. Organic valvular affections, obesity, and chronic Bright's disease cause *hypostatic bronchitis*. The primary form, which is rare, is the result of exposure to wet or cold or to the daily inhalation of some irritant that produces and maintains a low grade of catarrhal inflammation (dust, vapors). When chronic bronchitis follows the acute form we are often able to detect the operation of some favoring cause, as age, climate, or season. It is most common in the aged, though younger persons occasionally suffer, and it occurs by preference during the cold season, often recurring regularly in the cold and variable weather of autumn, winter, and spring, and disappearing in summer.

Symptoms.—The symptoms are similar to those of acute bronchitis, though rather less severe. *Pain* is rarely present, the patient complaining merely of a feeling of substernal constriction. There may be soreness at the base of the chest if the cough be frequent and severe, and occasionally in the epigastrium as a result of traction of the diaphragm on the ribs. *Cough* is not a constant accompaniment, however, but is *paroxysmal* and varies in severity and frequency. The degree of the violence of the paroxysm depends upon two factors—the character of the bronchial secretion and the seat of the catarrhal inflammation. Thus when the expectoration is tenacious and small in quantity, and when the small-sized tubes are affected, cough is most violent. It also varies both with the weather and the season, as is evident from the fact that there is often an absence of cough in summer, while it returns unfailingly with each new winter.

The *expectoration* differs widely in different cases. It is sometimes abundant and sero-mucous in character. On the other hand, there are cases of dry cough in which there is little or no expectoration. As a rule, however, it is rather copious, and either *muco-purulent* or distinctly *purulent* in character. *Fever* is usually absent, though rarely a slight rise of temperature occurs at night. The appetite is good as a rule; the bodily weight and nutrition are also well maintained.

Physical Signs.—On *inspection* we usually note undue enlargement of the thorax, with a decrease in expansile movements that is due to the associated emphysema. Hence *dyspnea* is commonly observed.

Percussion yields a clear or hyperresonant note. Dulness or impaired resonance is sometimes met with, however, during acute exacerbations, and especially over the bases, and is due to congestion and edema (Fox). On *auscultation* rhonchi of various forms and moist râles are heard, their number and size being in proportion to the extent of the swelling of the mucous membrane and the amount and fluidity of the secretory products. The respiratory murmur is enfeebled, though roughened, and the expiratory sound is prolonged and wheezy. The right heart may be dilated from increased tension in the pulmonary circuit.

Clinical Varieties.—Special forms, depending largely upon specific causal factors, remain to be described:

1. The commonest variety of chronic bronchitis has been called the "winter cough of the aged," and, as before intimated, is usually accompanied by emphysema and cardiac disease. For this form the gouty diathesis is often responsible. The cough occurs in paroxysms that are most severe at night, and during the early morning hours it is attended with free expectoration of the secretion that has accumulated during the night.

2. *Bronchorrhea.*—In this form there may be an abundant bronchial secretion, composed largely of serum (*bronchorrhœa serosa*), but more frequently perhaps the expectoration is purulent and thin, containing greenish or greenish-yellow masses. It may at times be thick and purulent. Dilatation of the tubes and resulting fetid bronchitis may be developed as secondary conditions.

3. *Fetid Bronchitis.*—In this variety the expectoration emits the characteristic odor of decomposing animal substances. The fetor may indicate gangrene of the lungs, abscesses, bronchiectasis, decomposition of matter within phthisical cavities, or empyema with perforation of the lung. It may, however, occur independently of the above-mentioned conditions, and hence these must be carefully excluded before the diagnosis of true fetid bronchitis is made. In the latter disease the expectoration is usually copious, and on standing separates into three layers, of which the uppermost is composed of frothy mucus, the intermediate of a serous liquid, and the lowest of a thick sediment, that presents a granular appearance and is made up chiefly of small yellow masses—the characteristic Dittrich's plugs. Microscopically, the Dittrich's plugs are seen to be composed of micrörganisms, chief among which is the *Leptothrix pulmonalis*; they may also contain pus-corpuscles, fat-granules, and crystals of margarin. Dèmétre found the colon bacillus and ascribes the fetor to its presence.

The condition may be a grave one, and associated with it may be observed ulceration of the bronchial tubes, with dilatation, pneumonia, abscess, gangrene, and rarely metastatic cerebral abscesses. When *putrefactive changes* take place in the bronchial secretion in the course of chronic bronchitis a new group of symptoms, as a rule, immediately appears. This comprises rigors that occur at irregular intervals and are associated with high fever and increased prostration. Cough and pain in the chest also become aggravated, but these acute symptoms may shortly subside and the usual course of chronic bronchitis be resumed. Even under the latter conditions fetor of the breath and sputum may persist.

4. *Dry Catarrh.*—The cough is both severe and paroxysmal, and there is little or no expectoration. When expectoration is present the sputum is very tenacious and is expelled with great difficulty. An asthmatic disposition is sometimes noticeable in this variety, and emphysema is commonly associated. The dry condition of the bronchial mucosa is evidenced by sibilant and sonorous râles. This form occurs in old persons, as a rule.

5. Osler has described a form of chronic bronchitis that occurs most frequently in women, and dates its onset from a comparatively early period of life. It does not undermine the general health. The cough is most pronounced in the morning, and is accompanied by a relatively small amount of muco-purulent expectoration. An examination of the chest yields negative results. The condition seems to proceed from a gouty or tuberculous diathesis in some instances. I have had under observation for several years a young woman in whom this form of bronchitis alternated with eczema of the face.

6. Teichmüller has described an eosinophilic bronchitis. The expectoration is mucoid, as a rule, though occasionally muco-purulent. It is characterized particularly by the presence of a considerable number of eosinophile cells in the sputum. It is not dependent upon adenoid disease of the naso-pharynx. Some writers doubt its existence.

Diagnosis.—The diagnosis of chronic bronchitis is rarely difficult. Since it is usually a secondary condition, it is of the utmost importance to determine the nature of the primary affection. An examination of the heart and of the urine should not be overlooked.

Pulmonary tuberculosis is to be discriminated from chronic bronchitis, and the distinctive points are—(1) A clear tuberculous history. In phthisis there are fever and loss of flesh and strength, while in chronic bronchitis fever is absent and the general health is not impaired. (2) In pulmonary tuberculosis the signs of localized consolidation (usually at one or other apex) appear early, while in chronic bronchitis the vesicular structure is not involved. (3) In phthisis the sputum, when examined microscopically, shows the presence of the tubercle bacillus.

In *acute pulmonary tuberculosis* the fever, dyspnea, cyanosis, and increased prostration constitute a group of features that should serve to avert the danger of its being confounded with chronic bronchitis. Coexisting *pulmonary emphysema* is to be recognized by the characteristic symptoms and signs of this complaint. *Primary fetid bronchitis* must be differentiated from the various other conditions previously mentioned, in which the breath as well as the sputum may emit the characteristic fetor. In *abscess* of the lung the sputum contains shreds of lung-tissue, including elastic fibers, crystals of hematoidin, cholesterin, and amorphous blood-pigment; usually localized dulness and broncho-cavernous breathing coexist. In *gangrene* there are contained in the sputum shreds of lung-tissue, but separate elastic fibers are often absent, on account of the presence of a ferment that causes a solution of the elastic tissue (v. Jaksch). *Bronchiectasis* is usually unilateral, and gives rise to dulness and other physical signs that are confined to limited areas, while in chronic bronchitis the signs are general.

Prognosis.—Recovery is the exception, though improvement may frequently be observed. The course of chronic bronchitis is exceedingly protracted, and the danger from the late development of certain complications and sequels, such as emphysema or right-sided cardiac disease, must be constantly borne in mind. Since the disease is generally a secondary affection, the prognosis in most instances depends upon the outlook in the primary disease.

Treatment.—The treatment falls naturally under two main heads —(1) Hygienic, and (2) Medicinal.

1. *Hygienic.*—This has reference, frequently, to the removal of various noxious influences. When the patient cannot make a suitable change of air during the cold season, he must keep his room during inclement weather; he should, however, be allowed to spend as much time as possible in the open air during clear and pleasant weather. The vitiated atmosphere of saloons or public halls is to be avoided. The patient should be carefully clad; he should wear flannels next the skin during all seasons of the year, but his outer clothing need not be unusually cumbersome. If the case be of an aggravated type and the circumstances of the patient permit, he should be sent to a warm latitude in the autumn, in order thus to escape the effects of a severe northern winter. It is an excellent rule to send patients in whom the bronchial secretions are abundant to a dry, warm climate or to a region whose atmosphere is impregnated with the balsamic vapors of the pine. On the other hand, patients with dry bronchial catarrh are most relieved by an equable, moist, warm climate. Among suitable resorts, those that should be mentioned are the Riviera, Cannes, San Remo, Sicily, and Algiers abroad, and Florida, Southern Georgia, and Southern California at home. Change of air becomes not only a means of relief, but also an effective means of prevention if resorted to at the proper time.

Prophylaxis also includes the removal of any diseased conditions that are causally related. The coexistence of cardiac disease, the gouty diathesis, obesity, and particularly any renal disturbance, call for the primary treatment of these conditions.

The *diet* should be generous, but not stimulating, and articles easy of digestion should be selected. Wines and liquors are to be avoided unless special indications for their use exist. Special conditions, however (*e. g.* albuminuria), may render necessary a special dietary.

2. *Medicinal.*—In this disease medicines are palliative in their effects rather than curative. Relaxing expectorants are to be avoided, owing to their depressing action, and the stimulating expectorants are, in a majority of cases, not only valueless, but hurtful, since they are liable to lessen the appetite and disorder the digestion. When, however, the sputum is muco-purulent in character and is dislodged with difficulty, expectorants of this class (squills, senega, ammonium muriate) may be

ed. I have obtained good results from the use of the following in ses attended with severe paroxysms of cough:

℞. Ol. eucalypti, ʒjss–ʒiij (6.0–12.0);
Codeinæ, gr. vj (0.388).
M. et ft. capsulæ No. xviij.
Sig. One every four hours, as required.

Occasionally potassium iodid exerts a curative influence, but its use ay be limited to cases that are due to the syphilitic, rheumatic, and outy diatheses. Five or ten grains of the iodid four times daily may e exhibited, and should there be present a syphilitic taint the remedy hould be pushed to the limit of tolerance. The balsam of copaiba is ometimes efficacious, several instances in my own experience having yielded to the following combination:

℞. Balsami copaibæ, ʒj–ʒij (4.0–8.0);
Ammon. muriat., ʒij (8.0);
Extr. glycyrrh. pulv., ʒj (4.0).
Mist. ammoniaci, q. s. ad f℥iij (96.0).—M.
Sig. ʒij (8.0) every four hours.

Other remedies that possess great value in certain cases are creasote (in ascending doses), turpentine, terpine, tar, the balsams of tolu and Peru, and sandal-wood. H. C. Wood praises sulphuretted hydrogen in cases in which there is profuse expectoration: "From two to four ounces of the saturated watery solution may be administered by the mouth four or five times a day or until the breath has a perceptible odor."

If the vital powers are poor, bitter tonics, as iron, quinin, and strychnin, and other measures calculated to invigorate the system, are indicated. When the sputum is excessive in amount, astringents (zinc sulphate and oxid) are sometimes useful. Astringents may also be used with advantage in the form of a spray when the expectoration is too free. On the other hand, sprays from properly selected solutions (*e. g.* ammonium muriate, gr. v–x ad ℥j—0.324–0.648 ad 32.0) are valuable in assisting expectoration. In fetid bronchitis sprays of antiseptic solutions are to be used, and the following will be found serviceable:

℞. Acidi carbolici, gr. ij–iv (0.129–0.259);
Olei eucalypti, ♏ij–iv (0.133–0.266);
Aquæ, ℥j (32.0).
Sig. To be inhaled from a steam- or hand-atomizer.

Pneumato-therapy has given brilliant results in certain instances, and G. Carrière obtained good results in streptococcic chronic bronchitis from the use of antistreptococcic serum.

BRONCHIECTASIS.

Definition.—The universal or circumscribed dilatation of the bronchial tubes.

Pathology.—Two main forms are recognized—the cylindrical or simple, and the saccular, and both of these may be met with in the same lung. Rarely the condition is congenital. It may be general or partial, the former variety being always unilateral, the latter sometimes bilateral. In *universal bronchiectasis* the bronchial tubes, throughout their extent, are the seat of numerous sacculi communicating with one another. These present smooth, shining walls, except in the most dependent parts, where ulcers are sometimes seen. Extreme conditions of dilatation may take the form of huge cysts, which may extend to the periphery of the lung; the lung-tissue lying between the sacculi then becomes cirrhotic as a rule. In *partial dilatation* the bronchial mucous membrane is implicated, with an occasional narrowing of the lumen. Most commonly these dilatations are cylindrical, though they may be saccular, and rarely fusiform. The partial is much more common than the general variety.

Histology.—When the walls of the larger dilatations are examined microscopically, the cylindrical epithelium is seen to be replaced by a pavement epithelium. The elastic and muscular layers are thin, and the fibers are usually separated. Contained in these dilatations are frequently found secretions that may be fetid.

Etiology.—In the majority of instances the condition doubtless arises from an involvement of the bronchial mucosa that extends to the submucous tissue and leads to muscular, fibrous, and cartilaginous atrophy. These changes render the wall of the tube unable to resist the pressure of the air in violent paroxysms of cough, and, once the process of dilatation is commenced, the accumulated secretions may tend by their weight to distend further the already weakened walls. Thus the elasticity of the latter is impaired, and finally destroyed. The etiologic factors show the affection to be *secondary* as a rule, and are—(1) Chronic bronchitis and emphysema, chronic phthisis (usually when the seat of the dilatation is at the apex), broncho-pneumonia (in children), and compression of a bronchus (*e. g.*, by aneurysm). Heubner believes that bronchiectasis in adults may be sometimes traced to whooping-cough and measles in young children. (2) Great thickening of the pleura, especially when associated with bronchitis or interstitial pneumonia, with contraction of the lung. (3) Rarely it is a congenital lesion.

Among predisposing conditions are—(*a*) *Age*, bronchiectasis being most common in adult or middle life; and (*b*) *Sex*, it being more common in males than females.

Symptoms.—There is always *cough*, and this usually occurs in prolonged and severe paroxysms. The attacks take place most generally after the dilated tubes fill in the morning, and a change of posture may excite them. Accompanying the cough there is *profuse expectoration*, which may amount to a pint or more in twenty-four hours. The sputum is grayish-brown in color and muco-purulent, emitting a sour or, more frequently, a horribly *fetid odor*. On standing, the expectoration separates into three strata—the uppermost, of brownish froth; the middle, of a thin, sero-mucous fluid; and a thick sediment, of cells and granular débris. Examined microscopically, the sputum is seen to be composed chiefly of pus-corpuscles, with which are intermingled Charcot-Leyden and fatty-acid crystals, the latter being arranged in the form of bundles; also leptothrices, vibrios, and bacteria are found. Elastic fibers may be observed if ulcers be present.

Dyspnea is noted, but is not a prominent symptom, unless some other chronic affections of the chest coexist or some complication arises. *Hemoptysis* occurs rarely, and may be due to the bronchiectatic lesion. Abscess of the brain may develop, though rarely.

Physical Signs.—These differ in character according to the size, situation, and nature of the dilatation, and also according to the condition of the surrounding lung-tissue.

On *inspection* retraction of the chest-wall may be noted when chronic pleurisy and interstitial pneumonia are associated. The tactile fremitus is usually increased, but may rarely be diminished. The *percussion* resonance is impaired or even flat, and on *auscultation* bronchial breathing is heard, with occasional râles that have a metallic quality. A *saccular dilatation* immediately beneath the pleura may give a tympanitic note, and may also give typical cavernous or amphoric respiration. A tympanitic resonance over a circumscribed area which prior to cough and expectoration presented dulness, is a significant sign (Babcock). These signs are generally discoverable at the base of one or other lung.

Diagnosis.—Simple dilatation of slight degree may exist without appreciable signs, and in other instances the breathing is broncho-vesicular over localized areas, with râles displaying increased metallic quality.

SACCULAR BRONCHIECTASIS.	PULMONARY TUBERCULOSIS.
History of chronic bronchitis, chronic pleurisy, and interstitial pneumonia, or of foreign body.	History of cough, hemoptysis, with progressive loss of flesh and strength. Family history.
Cough is paroxysmal, and sputum characteristic and copious.	Cough less paroxysmal. Sputum nummular in the stage of cavity.
Tubercle bacillus absent.	Tubercle bacillus present.
Course longer, with little impairment of the general health.	Course relatively shorter, powers of the system progressively undermined.
Physical Signs.	
The condition is persistent, but non-progressive. Usually located near base posteriorly.	Generally progressive, more frequently at one or other apex.

Circumscribed empyema with a fistulous connection with the lung may simulate bronchiectasis. There is often in such cases a clear history of an acute illness with a sudden onset, the symptoms pointing to pleural inflammation. The patient suddenly expectorates, at irregular intervals, large quantities of purulent matter. *Actinomycosis* may also cause conditions that simulate bronchiectasis. The diagnosis may be made by finding granular particles containing the actinomyces in the sputum.

Prognosis.—Apart from certain remote dangers (*e. g.* abscess, gangrene), these cases pursue a favorable but exceedingly protracted course.

Treatment.—The lesion being a permanent one, there is no known remedy that will either abridge or influence the course of the affection. Again, since the cough is protracted and attended with profuse expectoration, sedatives and ordinary expectorants are contraindicated. For the fetor, antiseptics are to be employed both topically and internally, and a solution of carbolic acid (1–3 per cent.) or thymol (1 : 1000) is to be used by inhalation. Internally, terebene (♏v–x—0.333–0.666) in capsules every four hours is valuable; also creasote in increasing doses (♏j—0.066, increasing by ♏j each day, till ♏vj—0.399—are taken three times daily) is to be persistently employed.

Should the above methods prove unavailing, intrathoracic injections of disinfectants are often resorted to with gratifying results.

In instances in which the dilatation is situated superficially and is not amenable to therapeutic measures, it may be freely opened and thoroughly drained.

BRONCHIAL STENOSIS.

Definition.—Narrowing of the bronchus, due either to constriction or to compression.

Pathology and Etiology.—(*a*) *Stenosis due to Constriction.*—This form is most frequently occasioned by the presence of foreign bodies; by new growths (polypoid) within the bronchi, or by growths without, or the cicatrices of healed ulcers, and in the case of the smaller bronchi by swelling of the mucosa. The bronchial walls also sometimes become thickened by inflammatory exudates in certain acute and chronic affections, such as syphilis, tuberculosis, and glanders.

(*b*) *Stenosis due to Compression.*—Compression of one or more bronchi may be met with in a variety of enlargements involving the organs within the thorax, among which are aneurysm, echinococcus cyst, solid tumors, enlarged glands, mediastinal and pulmonary abscesses, and extensive pleural effusion.

Symptoms.—The symptoms do not depend upon the cause of the obstruction, but their extent and character are in proportion to the size of the bronchus affected and the degree of stenosis. *Dyspnea* is the most conspicuous symptom, and when this is marked the accessory muscles of respiration are brought into active play, and still the proper filling of the lungs with air is not accomplished. Under these circumstances the air in the lungs becomes rarefied, and instead of normal expansion everywhere the lower part of the sternum and the lower ribs are *retracted* on inspiration, and expiration is accomplished only with difficulty. Obstruction of the primary bronchus on either side of the chest would naturally be followed by inspiratory retraction of the inferior part of the chest-wall and intercostal spaces upon the affected side. It is to be recollected that the movements of the larynx are slight in bronchial stenosis, while they are marked in laryngeal obstruction. *Cough* and *expectoration* are sometimes present, and *fever* of moderate severity is often noted.

Physical Signs.—*Inspection* shows defective respiratory movement upon the side involved. The local tactile fremitus is diminished or absent upon the affected side, owing to the obstruction to the passage of the vibrations of the voice to the pulmonary periphery. The *percussion-note* remains unaltered, though less influenced by forced respiration, and particularly expiration, than in health. Pulmonary atelectasis may occur as a secondary event, and is shown by dulness on percussion. The *auscultatory* signs consist of a greatly diminished vesicular murmur on inspiration, due to the diminished amount of air entering the air-cells during inspiration, and the presence of râles, sibilant and sonorous in character, at the seat of obstruction. Obstruction of a small bronchus may, however, be present without appreciable physical signs, owing to the fact that the surrounding lung-tissue may take on compensatory emphysema.

Diagnosis.—The nature and site of the affection may be determined by auscultation, and sibilant and sonorous râles will be conspicuous at the point of constriction. A clear history, together with a careful investigation of antecedent affections of the thoracic organs leading up to the stenosis, are factors that must furnish the etiological data in individual cases after the exclusion of foreign bodies as the possible cause. Tracheal or laryngeal stenosis may be eliminated by careful laryngoscopic examination.

Prognosis.—The duration is indefinite, though usually protracted, and most cases yield an unfavorable prognosis. In those instances, however, in which the narrowing is due to foreign bodies the latter may rarely be dislodged and fortunately ejected, thus averting danger to life.

Treatment.—The treatment must be addressed to the cause in individual cases. Obviously, the question of the removal of foreign bodies from the bronchi falls within the domain of surgery, though the administration of an emetic has been followed by complete success in certain instances. Obstruction due to stenosis of a main bronchus may be treated by dilatation with bougies, the treatment of course being carried out by a specialist.

ASTHMA.

(*Bronchial Asthma.*)

Definition.—A chronic affection, characterized mainly by paroxysmal dyspnea, due to contraction of the muscles of the smaller bronchi. The paroxysmal dyspnea produced by arterial contraction is also termed asthma by many writers.

Pathology.—In many cases there is hyperemia of the bronchial mucosa, due to pneumogastric or vasomotor functional disturbances, and also a characteristic exudate of mucin. In others there may be no lesions whatsoever, and the condition is a pure neurosis, often of reflex origin. Von Leyden considers asthma to be a reflex neurosis, the *primum movens* of which may be situated almost anywhere in the body. The morbid changes peculiar to chronic bronchitis, pulmonary emphysema, and right-ventricular hypertrophy with dilatation are found at autopsy.

Etiology.—There is present either a constitutional peculiarity or a singular susceptibility of the local muscular fibers to spasmodic contraction, both of which are of unknown nature. The exciting factors are very various, but may be grouped under four heads:

(1) **Acute Bronchitis.**—It must not be forgotten, however, that a bronchitis may be set up by the paroxysms. Curschmann has observed also a local croupous inflammation of the smaller bronchioles in some

of his cases, which he describes as ***bronchiolitis exfoliativa***, and which seems to have given rise to the seizures in grave cases.

(2) The inhalation of numerous and widely various ***irritants***, as chemical vapors, smoke, fog, dust, and emanations from plants or certain animals. A person may be immune in the city and suffer greatly on going into certain rural districts.

(3) *Reflex Causes.*—The causal connection between obstructive affections of the nose and asthma is a subject that is appreciated by the specialist. Gastric disturbances and, as I have observed, intestinal irritation are productive of this complaint. In dyspeptic asthma Boas found the diaphragm elevated above its normal position.

(4) Asthma may be secondary to, and most probably excited by, cardiac disease, emphysema, gout, rheumatism, syphilis, Bright's disease, emotional excitement, and irritating lesions in the region of the medulla. Possibly, some of the latter affections merely constitute predisposing factors. In this connection it is to be pointed out that individual liability to the disease depends upon the special etiologic factor.

Predisposing Causes.—*Heredity* takes first place, and is, when discoverable, well marked; it is noted in about 50 per cent. of all cases. The complaint is about twice as frequent in ***males*** as in *females*, and, if we except hay asthma, it is more prevalent in winter and early spring than during the warm season.

Clinical History.—Hyde Salter's collective statistics show that prodromal symptoms appeared in about one-half the instances (in 111 out of 226 cases). They differ widely, but are chiefly ***nervous*** in a great proportion of cases, and appear as irritability of temper, either depression or unusual buoyancy of spirits, headache, drowsiness, and vertigo. Abundant diuresis and digestive disturbances tend to appear.

The *paroxysm* usually comes on in the night during sleep, and at a definite time. It may develop, however, while awake or, again, though rarely, during the day. The onset may be sudden, but perhaps more frequently the patient first experiences a moderate grade of dyspnea and thoracic constriction. This augments with unwonted rapidity, and often attains to an inordinate degree, until the patient feels smothered, sits up, grasps his knees with his hands, or places the palms upon the bed so as to raise the shoulders and thus reinforce the accessory muscles of respiration. If the attack be severe, he rushes to an open window when able to leave his bed, or sits on a chair and places his arms on the back of another chair, so as to fix the shoulders and thus give purchase to the auxiliary muscles of respiration while frantically endeavoring to maintain the act of breathing. The *face* is pale, anxious, and soon is bedewed with cold perspiration, while the lips, eyelids, and finger-tips are livid, owing to defective oxygenation of the blood. The *temperature* becomes subnormal and the *pulse* feeble and rapid. The clinical picture wears an alarming aspect, but in uncomplicated cases death never supervenes.

Physical Signs.—*Inspection* shows enlargement of the chest, which in the advanced stage becomes barrel-shaped. The reason for this is the presence of an increased amount of air in the thorax with a total inability to expel it. The respirations are diminished in frequency to 12 or 10 per minute. The natural rhythm is also greatly disturbed, and inspiration is seen to be short and gasping, and followed immediately by expiration, which is greatly prolonged. The expansile movement of the chest is very limited, and in inverse ratio to the patient's efforts at breathing. There is lowering of the diaphragm. *Palpation* is negative in its practical results. *Percussion* yields a hyper-resonance; in advanced cases with associated emphysema semi-tympanitic resonance is common. On *auscultation* the inspiration is found to be short and feeble, and the expiration much prolonged and accompanied by a low-toned wheezing sound that may also be audible to onlookers. A great variety of dry râles are heard, chiefly high-pitched, sibilant, and sonorous, that are more marked on expiration than inspiration. They also change their character and situation frequently. At the close of the attack moist râles may be heard, and occasionally, when bronchitis complicates asthma, the moist râles may be combined throughout the paroxysms.

The *duration* of the attack is various, ranging from a few minutes to several hours, though rarely it may endure a week or two, with spontaneous remissions during the day (*e. g.* when chronic bronchitis coexists). Usually it subsides *abruptly*, with the expectoration of rounded gelatinous masses and, later still, of muco-purulent material. The former, when floated in water, are found to be composed of the so-called *Curschmann's spirals* (mucous moulds of the smaller tubes), and the spiral character of these small, ball-like pellets may even be detectable with the naked eye. When examined microscopically their spiral structure is evident. Two forms are recognized: (1) Composed of *mucin*, arranged spirally; in its meshes may be observed alveolar cells, many of which have undergone fatty degeneration. (2) A perfectly clear and translucent filament that is most probably composed of transformed mucin and occupies the center of the coiled spiral of mucin. In the early stage of the attack Curschmann's spirals (Fig. 42) are invariably present in the expectoration, and in many instances Leyden's octahedral crystals are also visible. For a time the latter were supposed, though erroneously, to excite the paroxysms by means of their irritating character. Similar crystals are found in the semen, as well as in the blood in certain conditions (*e. g.* leukemia). Müller, Fink, Leyden, and others have demonstrated extremely large numbers of *eosinophile leukocytes* in the

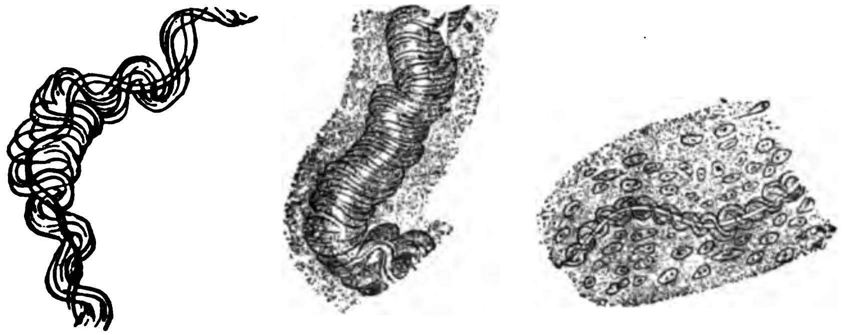

FIG. 42.—Curschmann's spirals.

sputum. Fink and Gabritchewski likewise have found a large excess (ranging from 15 to 35 per cent.) of eosinophile leukocytes in the blood. V. Noorden and Swerchewski found the same increase, but only at the times of the attacks.

Diagnosis.—A clear history, together with the physical signs and a microscopic examination of the sputum, should lead to correct results. The history alone is inadequate to put the physician upon the right track. *Laryngeal affections*, which give rise to spasm of the glottis and dyspnea, are to be eliminated by the alteration of the voice and the aphonia which are usually present, while the characteristic physical signs of asthma are absent. Again, the dyspnea is inspiratory, not expiratory as in asthma.

Emphysema may be confounded with asthma. The presence of recognized causes, of typical physical signs, and the paroxysmal dyspnea in asthma are the chief points of distinction from emphysema. The sputum should be examined microscopically if doubt remain.

Course and Prognosis.—In mild cases of asthma there may be but one or two nocturnal paroxysms, with entire freedom from cough and dyspnea during the following day. On the other hand, in severe cases there is a repetition of the paroxysms from three to five or six nights. Under these circumstances in the intervals (usually corresponding to the period of day) there are slight wheezing and some cough. In long-standing cases asthma leads constantly to the development of chronic bronchitis and emphysema, and in such these affections are invariably combined. The paroxysmal character of the affection is often partly or wholly lost, the patient rarely being entirely free from asthmatic dyspnea, combined with cough and muco-purulent expectoration. The periodicity of the attacks varies greatly; in some it recurs monthly or at even shorter intervals, and in others only annually.

There is rarely any danger to life, except when the secondary affection is emphysema and its remote consequence is dilatation of the right ventricle; but the percentage of cases in which recovery actually takes place is comparatively small, since the affection may reappear long after the paroxysms have ceased to recur in the usual manner.

Treatment.—The indications for treatment are—(1) to cut short the paroxysms, and (2) to prevent a recurrence of subsequent attacks.

(1) To bring relief during the paroxysms we should ascertain the exciting cause, and remove it promptly if possible to do so. In one of my own cases a prolonged paroxysm was cut short by a calomel purge followed by an enema. An overloaded stomach calls for an emetic, and other causal factors are sometimes removable (*e. g.* congestion of the nasal mucosa, dust, animal and vegetable emanations). If the cause is irremovable, the patient should be kept in a large and freely ventilated apartment, and everything that tends to impede respiration must be removed. The choice of posture as affording the greatest relief may usually be left to the patient.

To cut short the paroxysms: The particular mode of treatment that will afford most speedy relief differs widely in different cases, and not infrequently the patient, as the result of experience, is aware of the remedies that are most efficacious for good. As a rule, however, sedative antispasmodics, relaxants, and stimulants are the classes of medicinal agents from which a careful selection is to be made; and whilst a great variety of these have been employed, I shall content myself by adducing here only the most valuable and their mode of administration. In the hands of some observers a few whiffs of chloroform have proved highly efficacious, but in my own they have produced only momentary good effects; ether is the safer remedy and may be tried in like manner. In a certain proportion of the cases from four to six drops of amyl nitrite thrown upon cotton-wool or a handkerchief, and inhaled, bring speedy and permanent relief. Of stimulants, coffee is the best: immediately upon the appearance of the paroxysm about one pint of strong coffee is to be taken hot (without cream or sugar), and in this way the seizure may sometimes be arrested. Alcohol when given hot and in sufficiently large doses to induce mild intoxication may be found very useful; and with "hot toddy," spirits of chloroform may be combined.

The inhalation of the fumes of niter-paper[1] often gives quick, temporary, and, less frequently, permanent relief. When employed, the atmosphere of the room occupied by the patient must be well filled with the fumes.

Among depressant antispasmodics are belladonna, hyoscyamus, stramonium, and lobelia, and these seem to be of most value when used in the form of cigarets. The leaves of the plant employed are first steeped in a concentrated solution of potassium nitrate or chlorate, and a trial should be made of different sorts of cigarets or pastilles (which are similarly prepared), since all cases are not benefited by the same brand. The inhalation of tobacco-smoke is equally beneficial in rare instances.

A large number of cases, despite the use of the measures above indicated, exhibit an obstinate tendency, and for their treatment no remedy bears favorable comparison with morphin, administered hypodermically, It has occasionally led to the establishment of the morphin-habit; hence it must not be used indiscriminately. It is best given in full doses (gr. $\frac{1}{3}$–$\frac{1}{2}$—0.0216–0.0324), and may be combined with atropin or cocain. V. Noorden uses atropin in ascending doses (gr. $\frac{1}{120}$ increased to $\frac{1}{16}$ daily), then gradually diminishes the dose; this treatment is repeated every few months, though lessening the dose and shortening each course of treatment. Strychnin also has its advocates (Mays). The following formula has proved efficient in my hands:

℞. Tr. lobeliæ,	ʒj	(4.0);
Tr. nitro-glycerini (1 per cent.),	♏xvj	(1.06);
Sodii bromid.,	ʒv	(20.0);
Vini ipecac.,	ʒv	(20.0);
Ext. hyoscyami,	gr. viij	(0.518);
Elix. simplicis, q. s. ad	℥iv	(128.0).—M.

Sig. ʒj (4.0) every one or two hours in water.

S. Solis-Cohen lauds hyoscin hydrobromate (gr. $\frac{1}{200}$) administered hypodermically. Ergot and adrenal extract are among promising remedies; they probably act by promoting vascular tone. In the protracted cases of old asthmatics, associated with chronic bronchitis and emphysema, the above mixture may also be employed, though sodium iodid (gr. v—0.324) should be substituted for the bromid, and the same dose given at intervals of three or four hours.

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[1] Niter-paper is prepared by dipping bibulous paper (filter- or blotting-paper) in a solution of saltpeter.

(2) In order to prevent subsequent attacks: During the intervals the physician must ascertain whether any of the numerous causes (bronchitis, gastric disorders, dust, emanations from plants) are discoverable; and if so, efforts to remove them should be instituted. A methodical interrogation of the various organs of the body and their functions must be carried out, and the therapeutic or hygienic indications presented by them, if any, must be met judiciously. The nasal passages should be examined by a specialist, and any causal conditions found therein are to be promptly removed. If the affection be a pure neurosis or due to bronchitis, a suitable climate may often be found in which the patient will enjoy complete immunity from asthma. The choice of the locality cannot, however, be determined by any known rules. The patient must travel from place to place until he finds the climate that possesses preventive properties in his particular case. To those who cannot adopt this plan potassium iodid offers the best hope of relief, though its use must be long continued (gr. x–xx—0.648–1.296, three times daily). The systematic use of compressed air in the pneumatic cabinet, and also the inhalation of oxygen, are worthy of trial. The presence of any conditions of ill-health calls for appropriate treatment. Talma believes that the attacks may be overcome by proper, rhythmic, deep breathing.

There are also certain means of prophylaxis for impending attacks. Thus, if there be premonitory symptoms, the use of such measures as strong coffee or the "hot toddy" above mentioned, Hoffman's anodyne, stramonium or belladonna cigarets, the inhalation of the fumes of niter-paper or of a few drops of amyl nitrite, or the removal of the sources of irritation, may suffice to ward off the attack.

FIBRINOUS BRONCHITIS.

(*Plastic Bronchitis; Croupous Bronchitis: Mucous Bronchitis.*)

Definition.—A rare acute or chronic catarrhal affection of the bronchial mucosa, attended with the production of fibrinous casts (?) that are expectorated in severe paroxysms of cough and dyspnea. These casts, when unfolded, are found to be solid moulds of the bronchial tubes from which they come, being shaped like the branches of a tree, and thus proving that a bronchial tube and its subdivisions had been blocked. When the moulds are large or medium-sized they are hollow, and when from the smaller bronchi they are solid.

Pathology.—The pathology is but little understood, but in one of my own cases I found the composition of these casts to be identical with that of croupous exudates met with elsewhere, though more dense, perhaps, than the latter. Croupous bronchitis is attended with loss of epithelium in the implicated bronchi, as is the case in croupous inflammation wherever it occurs; but the answers to the questions, "Why should the affection be limited to a definite portion of the bronchial tree?" and "Why does it recur from time to time?" are obscure indeed. In fatal cases the lesions of associated or antecedent complaints, such as chronic pleurisy, pneumonia, and pulmonary tuberculosis, have been found.

Etiology.—What the irritant is that causes the condition is unknown, though streptococci have been found in the moulds and in the mucosa. Some of the predisposing causes, however, have been recognized, and are—(1) *Sex:* it being about twice as frequent in males as in females. (2) *Age:* though met with at all periods of life, it is relatively more frequent from the twentieth to the fortieth year. (3) *Season:* the seizures are most common in the spring months. (4) *Epidemic influences:* Pichini has described a group of instances that occurred in individuals in the same locality. (5) *Hereditary influence* has been traceable in a few cases. (6) Other affections, as tuberculosis (quite frequently), chronic pleurisy, and certain skin-affections, as herpes, impetigo, and pemphigus, form antecedent and associated conditions.

Symptoms.—(*a*) *The acute form* is rare. It begins with rigors and fever that are soon followed by urgent dyspnea and severe paroxysms of cough, which are usually attended, soon or late, by the expulsion of bronchial casts, and sometimes rather profuse hemorrhage. Abundant expectoration usually causes amelioration of the severer symptoms. On the other hand, urgent dyspnea, oppressiveness, and severe cough, with little expectoration, are grave symptoms, often leading to fatal asphyxia.

(*b*) *The Chronic Form.*—The attacks recur at irregular intervals and are less severe than in the acute form, the interim varying from one week to a year or more. In a case observed by myself the patient has experienced a recurrence once annually (on or about May 1st), commencing three years ago. The paroxysms may occur at regular though much briefer intervals. The cases usually manifest ordinary bronchitic symptoms, with or without fever at the onset. The cough soon becomes troublesome and is paroxysmal in character. There is expectoration in the form of rounded masses, which, when unravelled, are found to be true moulds of the affected tubes that exhibit a laminated structure. The larger casts (which are of the size of a goose-quill or even larger) may be hollow. They are of whitish or grayish-white color. When examined microscopically they are seen to consist of a fibrillated base, a few scattered leukocytes and mucous corpuscles, and, rarely, gland- and blood-cells. Curschmann's spirals are often found, and within these or associated with them the Leyden crystals. First, Beschorner and later Grandy have shown the casts to be composed of mucin. In other cases, however, similar studies show fibrin. Hemorrhage may occur.

Physical Signs.—Owing to the obstruction offered by the casts, there is a diminished amount of air entering the corresponding part of the lung. As a necessary result the tactile fremitus, local expansion, and respiratory murmur are diminished over the affected area. The note on percussion over the uninvolved portions of the lung is clear or hyper-resonant, though if the portions of the lung supplied by the affected tubes collapse, there is dulness on percussion. Dislodgement of the casts is followed by a return of the normal respiratory murmur.

Diagnosis.—From ordinary *bronchitis* it is to be distinguished by the presence of casts of mucin or fibrin, which alone are sufficient for a positive diagnosis. The fibrinous moulds met with in diphtheria and pseudomembranous croup, with extension into the bronchi, must also be eliminated. In doubtful cases, a bacteriologic examination of the membranous casts should be made. If the Klebs-Löffler bacilli are then found, all doubts as to its diphtheritic nature are set at rest. In truly diphtheritic cases the membrane does not present the laminated structure.

Prognosis.—The prognosis in the acute form is quite grave; the chronic variety, though pursuing an exceedingly long course that ranges from five to fifteen years, rarely terminates fatally.

Treatment.—This is to be conducted on the same principles as those in simple acute bronchitis. In the acute form an attempt should be made to soften and separate the casts by the topical application of steam, by inhalation, and alkaline sprays (*e. g.* lime-water). Pilocarpin was employed in one instance under my own observation with apparent good results; it tends to excite free bronchial secretion. Emetics should be resorted to without delay when the signs of cyanosis show themselves.

In the chronic form nothing can be accomplished by treatment, during the intervals between the acute exacerbations, that will tend to obviate a recurrence of the attacks or to mitigate their severity.

IV. DISEASES OF THE LUNGS.

CIRCULATORY DISTURBANCES IN THE LUNGS.

CONGESTION OF THE LUNGS.

(*Hyperemia of the Lungs.*)

Definition.—The surcharge of the pulmonary vessels with blood. Two forms are recognized: (1) Active hyperemia, and (2) Passive hyperemia.

ACTIVE HYPEREMIA.

Pathology.—The blood-vessels in the bronchial mucosa often appear intensely injected, and the capillaries in the alveolar walls are prominent, while on section a scarlet-colored, frothy liquid flows. The alveolar epithelium may become swollen and granular.

Etiology.—Active hyperemia is usually a symptomatic condition, though rarely it may arise as a distinct primary affection. Active congestion of the lungs exists as an associated condition in many pulmonary affections, as pneumonia, pleurisy, bronchitis, and tuberculosis. On the other hand, active congestion of the lungs may be engendered as an independent affection by the inhalation of hot air, highly irritative substances, as well as by violent physical exercise, the ingestion of large amounts of alcohol, and strong mental emotion. Collateral hyperemia may arise from anemia of the opposite lung.

Symptoms.—The capacity of the air-cells is diminished; hence the oxygenation of the blood is markedly interfered with. This embarrassment of the function of respiration is compensated for in part by accelerated breathing, there being a degree of *dyspnea* proportionate to the extent and intensity of the congestion. There is some *cough*, accompanied by *frothy, bloody expectoration.*

The physical signs are bilateral, as a rule, and are generally confined to the bases. *Palpation* shows increased tactile fremitus. The *percussion-note* is impaired or, rarely, dull, and it is generally exceedingly difficult to determine the pitch of the note, owing to the fact that both sides are usually involved. On the other hand, when the condition is unilateral and not associated with diseases of the opposite side, the impairment is readily appreciated. The vesicular element of the respiratory sounds is diminished, and the bronchial element relatively increased (*broncho-vesicular breathing*). Less frequently there is bronchial breathing.

Diagnosis.—In the presence of the etiologic factors the sudden development of *dyspnea*, *cough*, and a *frothy*, *bloody expectoration*, and the *physical signs* before enumerated, in the absence of fever, the diagnosis is easy.

Prognosis.—Active hyperemia is frequently followed by collateral edema. Its course is brief, and terminates either fatally in a few hours, in perfect recovery in a few days, or in pneumonia. The condition is therefore ominous.

Treatment.—Prompt measures must be instituted in order to arrest the active fluxion. The special causative factors must be actively treated, so as to diminish the quantity of blood in the pulmonary vessels; dry and wet cups over the entire seat of congestion must be tried: and in the worst cases venesection is demanded. Following the application of the cups, turpentine stupes, sinapisms, and linseed poultices may be employed. I have observed excellent results from the use of veratrum viride combined with saline purgatives. Other cardiac sedatives may also be employed, including nitroglycerin in full doses.

PASSIVE HYPEREMIA.

Passive, unlike active, hyperemia is always a secondary condition, and is quite common. Two forms are distinguishable: (*a*) Mechanical, and (*b*) Hypostatic.

(*a*) **Mechanical Hyperemia** (*Brown Induration*).—**Pathology.**—The pulmonary vessels are distended, the lungs as a whole enlarged, and the air-cells crepitate but little, owing in great part to the encroachment upon the air-spaces by the dark venous blood. The lungs are of a reddish-brown color and afford increased resistance to efforts at cutting or tearing. On section the reddish-brown tint rapidly changes to a vivid red, from oxidation of the hemoglobin when exposed to the atmosphere. The process commences at the extreme base, extends upward, and may finally become general. The interstitial connective tissue is increased, and is often edematous, while the epithelial cells of the alveoli show altered blood-pigment, usually in the form of hemosiderin and responding to the usual tests for iron.

Etiology.—Mechanical hyperemia results from the obstruction of the return of blood to the left heart, and among special causative conditions are mitral constriction, mitral regurgitation, dilatation of the right ventricle, and certain cerebral injuries and diseases. It may also be a symptom of asphyxia, and rarely it arises from pressure of tumors.

Symptoms.—The most marked feature is *dyspnea*, particularly when secondary to organic cardiac diseases with failure of the right ventricle. *Cough* is common, and an expectoration of *frothy serum* or *blood* (hemoptysis) containing pigmented alveolar epithelial cells, is the most characteristic clinical feature.

Diagnosis.—With a clear history, in addition to the dyspnea, cough, and the characteristic expectoration, the recognition of passive hyperemia of the lungs is a simple matter. The prognosis and treatment will be considered in connection with the causative affections.

(*b*) **Hypostatic Hyperemia.**—**Pathology.**—The parts of the lung that are affected are dark in color and the vesicles distended with a transudate of blood and serum. In this way the air-cells may become emptied of air (*splenization, hypostatic pneumonia*), and the resulting condition is in most instances to be regarded as a mild grade of lobular pneumonia. This view is confirmed by the fact that the same etiologic conditions that favor the development of hypostatic congestion also favor to an equal extent the development of hypostatic pneumonia.

Etiology.—Feeble cardiac action, as in long-continued fevers, debilitating chronic affections, and in old persons, combines with a prolonged dorsal position of the body (gravitation thus favoring its development) in producing the condition. This explains why the condition is found usually at the bases of the lungs, and is most marked posteriorly. It is common for the same reason in carcinoma, tuberculosis, paralysis, chronic rheumatism, typhoid fever, etc.

Symptoms.—The symptoms are wholly indefinite; indeed, none may be present. Priory has pointed out that old persons in the incipiency of the disease begin to sleep with the mouth open, so as to effect the entrance of more air. Commencing *cyanosis* may indicate the development of hypostasis, and a careful *physical examination* of the lower lobes of the lungs will show increased fremitus, slight dulness, diminished vesicular murmur, and, in the higher grades, bronchial breathing, with liquid bubbling râles.

The **prognosis** is based upon the character of the underlying affection.

Treatment.—This is an affection in which the treatment of causes alone will suffice, save in instances that are secondary to organic heart-affections, in which prompt bleedings are to be advocated. From a pint to a quart of blood should be taken, and I have seen happy results from the employment of this measure in extreme cases. Tapping the right auricle when the blood refuses to flow from an arm vein has also been successfully accomplished by competent surgeons. The patient's posture must be changed from the dorsal to the lateral, and even ventral, and as soon as possible he should be gotten out of bed.

PULMONARY EDEMA.

(*Edema of the Lungs.*)

Definition.—An effusion of serous fluid into the air-vesicles and interstitial lung-tissue. Pulmonary edema is scarcely to be regarded as an independent affection, but as a secondary condition, being in most instances associated with pulmonary congestion.

Pathology.—It consists of a transudation of serum into the alveolar walls, interstitial connective tissue, and air-cells, and rarely the process is limited to the interstitial tissue. Two forms may, for the sake of convenience, be recognized:

(a) Collateral Edema (*Inflammatory Edema*).—This is usually local in character, circumscribing an area of the lung that is affected by pneumonia, abscess, or pulmonary infarction, and is the result of a mild inflammatory process affecting the vessel-walls. When the condition follows hypostatic congestion the terms "hypostatic edema" and "splenization" have been applied.

(b) General Pulmonary Edema.—If congestion be not associated, the portions of the lungs involved by this type look pale; when pulmonary congestion or pigmentation of the tissue is present, the lung appears darker than the normal and the serum is blood-tinged. The weight of the lung-tissue, owing to the more or less airless condition of the alveoli, is increased, and yet, though heavier than the normal lung, the affected tissue does not sink in water. To the feel it is boggy, and pits on pressure, while on section a serous or sero-sanguinolent (if congestion be present) fluid of low specific gravity, and poorer in albumin than plasma, flows from the cut surface. Edema is most frequently observed at the bases of the lungs, though it may become general, and as a rule the surface of the pleura is moist; hydrothorax may be present.

The *mode of production* of pulmonary edema is not definitely known. Increased fluidity of the blood on the one hand, and increased tension in the pulmonary vessels on the other, seem to be influential factors in many cases. The heightened blood-pressure may be in great part due to a failure of cardiac power, and particularly to failure of the left ventricle (Welch). When weakness of the left is out of proportion to the weakness (paralysis) of the right ventricle, we are apt to have the tension in the pulmonary capillaries greatly increased, at least until transudation of serum is induced. Edema also occurs as a result of weakness of the right ventricle alone. Obstruction to the outflow, such as occurs in weakening of the left ventricle, or even obstruction in the aorta, leads to heightened tension and, secondarily, to paralysis of the right ventricle. The third factor entering into the production of pulmonary edema is the increased permeability of the vascular walls, due to impairment of their nutrition and "disturbance of the cardiopulmonic innervation" (Huchard). This usually arises in connection with toxic and infectious diseases, when the blood also exhibits more or less change, as in cachectic states, uremia, general septicemia, and the like. Instances are met with in which pulmonary edema, due to vasomotor relaxation, develops suddenly.

Etiology.—Pulmonary edema is secondary to pneumonia and acute and chronic affections, but not with any degree of constancy; nor is it especially liable to be associated with congestion or with low grades of inflammation of the lungs. Among the diseases of which it forms a terminal condition are—valvular affections of the heart, fatal forms of anemia, acute and chronic Bright's disease, cerebral lesions (hemorrhage, traumatism), and acute infectious fevers with failure of cardiac power.

Symptoms.—In edema of the lungs the air-space is lessened in direct proportion to the amount of serum occupying the alveoli; hence *dyspnea* is always present and is often a conspicuous symptom. There are *cough* and *bronchorrhea*. The *sputum* is usually abundant and frothy, and is expectorated with difficulty. At times, and especially in the acute forms, it is tenacious and may give rise to alarming laryngeal obstruction.

It is blood-stained if congestion be combined. The condition does not give rise to elevation of temperature, except in the inflammatory type, in which fever is constantly present. The pulse is accelerated and feeble, and cyanosis, particularly in cases of collateral edema, usually appears. The extremities are cool and often livid.

Physical Signs.—The reasons adduced to explain the dyspnea likewise render intelligible the physical signs encountered. There is dulness, though rarely complete, over the areas involved; the vesicular murmur is feeble or absent, or there may be broncho-vesicular breathing. Since the bronchioles contain serum, small râles, having a liquid character, are audible with inspiration and at the beginning of the expiration over the seat of the edema.

The **diagnosis,** with a clear history, is based upon the incomplete dulness that is usually bilateral and most marked at the bases, upon the bubbling râles heard over the corresponding area, and upon the absence of any febrile movement, except the latter be due to some underlying affection. *Hydrothorax* bears some points of resemblance to edema of the lungs, but in this condition the upper level of dulness is movable in consequence of change of position of the patient, as is not the case in edema of the lungs. On the other hand, in the latter affection moist râles are present, while they are absent in hydrothorax. *Broncho-pneumonia* may be mistaken for pulmonary edema, though it has a different mode of onset. It is also accompanied by fever, glairy, tenacious expectoration, and more sharply-localized areas of dulness then appear in edema.

The **prognosis** is governed by the pre-existing condition to which the edema is due. Thus, if secondary to a general dropsy due to renal or cardiac disease, it often destroys life with great rapidity. Inflammatory edema, following lobar pneumonia, is also grave in the extreme.

Treatment.—The treatment is that of the associated or causative affections. These must be sedulously treated, and the limitation of the transudation and the direct removal of the serous effusion from the lungs is of great importance. We should not fail frequently to change the position of the patient's body, so as to prevent the gravitation of blood to the dependent portions of the lungs. I have witnessed excellent results from the use of dry cups placed over the thorax, particularly over its posterior and lateral aspects, and renewed at intervals of six to eight hours. The number applied should range from one and a half dozen to three dozen. In aggravated forms that develop quickly prompt venesection is imperatively demanded. This is a measure which, if resorted to at the proper moment, will often rescue the patient from imminent danger. The condition of the heart and kidneys must receive attention. Tincture of strophanthus (♏iij every three hours) is effective in pulmonary edema in children.

HEMOPTYSIS.

(*Broncho-pulmonary Hemorrhage.*)

Definition.—An expectoration of blood. Its source may be the bronchial mucous membrane (usually the small bronchi), and less frequently it comes from eroded vessels in lung-cavities or their walls; rarely from the larynx, trachea, and larger bronchi. When from the bronchial tubes the term *bronchorrhagia* should be applied. The source of the hemorrhage, however, is not always easily demonstrable, even when it has resulted fatally and the lungs are minutely examined.

Pathology.—The lesions are often microscopic, and consist for the most part of ruptured capillary blood-vessels, though larger vessels may also become the seat of erosion or rupture. After death the bronchial mucosa is sometimes found to be swollen, bleeds easily, and is of a dark-red color—soon becoming decidedly pale. The lung-tissue proper may look paler than in the sound lung. When hemoptysis occurs in advanced pulmonary tuberculosis the lung-cavity may contain a ruptured aneurysm, or mere ulceration of an exposed vessel may be observed. I have witnessed small, dark-red, dense masses in the air-sacs scattered throughout the lung whence came the hemorrhage. Doubtless these are blood-coagula, which result from the clotting of the blood after the latter has been carried into the alveoli. Various associated lesions may be observed.

Etiology.—(1) **Pulmonary Affections.**—(*a*) *Pulmonary congestion* from whatever source may result in hemoptysis, although the amount of blood lost under these circumstances is usually small. There are many causes that excite congestion of the lungs, some of which reside in adjacent organs, it being common in organic disease of the heart, and particularly in disease of the mitral segments. That form of pulmonary congestion which is associated with other affections of the lungs, as well as primary active congestion due to inhalation of hot air, irritating substances, and violent physical exercise, may also result in hemorrhage. (*b*) *Hemorrhagic infarction* may lead to slight hemorrhage (*vide* Pulmonary Embolism). (*c*) *Croupous Pneumonia.*—In this disease hemorrhage is caused by the rupture of the capillaries, and the blood, when expectorated, has undergone a change, has become rusty-colored. (*d*) *Pulmonary Tuberculosis.*—This is pre-eminently the most common cause. Hemorrhage may take place early when it originates from a sharply-limited and minute tuberculous focus, and it may also be attributable to congestion. Undoubtedly its exact source is the mucosa of the small bronchi; later it is the direct consequence of the ulceration of an artery or of the rupture of an aneurysmal sac that has its seat in a branch of the pulmonary artery. After the tuberculous cavities have healed or while quiescent, calcareous masses are, from time, to time, expectorated, together with more or less blood. (*e*) *Ulcers of the Larynx, Trachea, or Bronchi.*—Rarely ulcers in adjacent structures erode the larger branches of the pulmonary artery and cause copious and speedily fatal hemorrhages. Osler observed a fatal hemorrhage in a case of chronic bronchitis with emphysema. (*f*) *Fibrinous bronchitis* induces hemoptysis by rupturing the capillaries in the bronchial mucosa at the time of separation of the bronchial casts. (*g*) *Carcinoma of the lung* produces frequent expectoration of blood. (*h*) *Gangrene of the lung.*

(2) **Diseases of Other Organs than the Lung.**—(*a*) *Affections of the heart* act as a cause, and especially advanced mitral disease, when it is due to pulmonary congestion. It not infrequently develops during the stage of adequate compensation. In a preponderating proportion of the latter instances the hemorrhage is slight, but it may be profuse and recur at intervals for many years. (*b*) *Aneurysm* of the branches of the *pulmonary* artery and of the arch of the aorta (usually with rupture of its coats) is a rare cause of hemoptysis.

(3) **Certain diseases,** such as *purpura hæmorrhagica*, *scurvy*, *anemia*, *hemophilia*, and *malignant forms of certain acute infectious diseases* (*e. g.* yellow fever), cause hemoptysis. In this class of cases the hemorrhages are due either to a diseased condition of the vessel-walls or to blood-changes.

(4) **Vicarious hemoptysis** is not uncommon during menstruation or when amenorrhea is present. Unless occurring at the time of the regular menses it is not to be regarded lightly, and is of the same significance as when taking place in the male. I cannot agree with those authors who contend that hemorrhage from the lungs in women is without the same dire significance as in the opposite sex. (*vide infra*).

(5) **Arthritic (Gouty) Endarteritis.**—According to Sir Andrew Clarke and others, this is a common cause of recurring hemorrhages in aged persons (over fifty years).

Symptoms.—Hemoptysis is so commonly a symptom of that most frequent and dread disease, phthisis, as to raise suspicions of the latter in the minds of the laity and physicians as soon as it occurs. It is appropriate, therefore, to note, first, the features of hemoptysis when dependent upon pulmonary tuberculosis, and then to point out its clinical peculiarities when due to other conditions.

In **incipient pulmonary tuberculosis** hemoptysis develops suddenly as a rule, a *warm, saline taste*, lasting but a few moments, generally preceding the expectoration of blood. The blood is *coughed* up, and the bleeding may last only a few minutes or may continue for days, the sputum being apt to remain blood-stained for a longer interval. The immediate effect of the hemorrhage, however slight, is to alarm the patient, inducing, besides mental agitation, cardiac palpitation and other nervous concomitants. A small hemorrhage is not attended with any other results, but large ones give rise to the symptoms of *shock*, combined with those of *symptomatic anemia.* When the hemorrhage is large, blood to the amount of a mouthful may be ejected with each cough, and in these instances the effect of the profuse bleeding is evidenced by such symptoms as vertigo, syncope, cold extremities, excessive pallor, perspiration, and a rapid, small, feeble pulse. This is followed, if the attack does not prove speedily fatal, by considerable restlessness, and later not infrequently by mild delirium and more or less fever.

In comparatively rare instances the same patient has a single hemorrhage; more frequently he has several at shorter or longer intervals. Large or small bleedings may precede by weeks, months, or even years any rational symptoms or physical signs of pulmonary tuberculosis. In such instances latent foci of disease may be assumed to have pre-existed.

In *quantity* the hemorrhage varies greatly: there may be less than one ounce ejected or it may amount to a pint or more before the bleeding ceases. In advanced cases in which cavities have formed large vessels may become eroded, followed by copious and dangerous hemorrhage. Fatal hemorrhage may take place into a cavity without the occurrence of hemoptysis, as in a case dissected by Osler at the Philadelphia Hospital. The distinctive characters of the blood discharged are mainly as follows: *bright color*, very *frothy* (being mixed with air), and *not clotted.* A rare exception to the rule may be noted in the case of hemorrhage proceeding from a large cavity, the blood pouring forth in a free, dark stream.

Physical Signs.—These are, for the most part, negative. Quite commonly moist bronchial râles are audible on auscultation; palpation and percussion should not be practised either during or immediately after the hemoptysis.

Hemoptysis not Due to Pulmonary Tuberculosis.—(*a*) In *affections* of the *mitral* and *aortic valves*, especially in mitral stenosis, hemorrhage from the bronchi is not uncommon, and the way in which these lesions lead to pulmonary congestion is explained in the discussion of Organic Affections of the Heart. During the progress of these cases, hemorrhages often occur at considerable intervals; they may either be slight, lasting only a few minutes, or quite free, extending over periods of a few days or a week.

(*b*) As a rule, in the beginning small hemorrhages occur for several weeks from pressure of an *aneurysmal dilatation* upon the bronchial mucosa, or there may be weeping of blood through the exposed layers of fibrin composing the walls of the sac. The bleeding point can be discovered with the laryngscope, when an aneurysm of the innominate or of the aorta impinges upon the trachea. A large and often quickly fatal hemorrhage occurs from rupture into the respiratory tract.

(*c*) "*Arthritic hemoptysis*" is undoubtedly associated with gouty, degenerative changes in the terminal blood-vessels of the lung, though no coarse pulmonary lesions are induced by the recurring hemorrhages. Although the hemorrhages may occur at intervals for years, as a rule they finally become arrested, and only rarely lead to a fatal issue. I have never observed this form of hemoptysis occurring independently of *chronic bronchitis*. In *emphysema* and *chronic bronchitis* small hemorrhages may occur, and occasionally coagula in the form of casts are formed in the bronchi and afterward ejected. It is probable that the source of the large bleedings that occur under these circumstances is an *ulcer* in the bronchial mucosa.

(*d*) The hemoptysis that is connected with the *menstrual function* is of frequent occurrence. I saw recently a patient in whom free bleeding has occurred at intervals of four weeks for a couple of years, with an absence of the menses. In another instance, a patient of Dr. Byers, recurring hemorrhages of the lungs took place instead of the regular menstrual discharge for three successive months, and a comparatively rapid and fatal form of phthisis was developed. This case typifies a large class that is especially prone to develop pulmonary tuberculosis.

(*e*) The preceding group is to be distinguished from those cases in which *trivial bronchial hemorrhages* sometimes occur, and in delicate, hysterical females. Although these bleedings are accompanied by cough, it is not uncommon to find, upon careful examination, that the blood comes from the upper air-passages.

(*f*) Hemoptysis may result from *severe injuries* inflicted upon the thorax, and last for days together.

(*g*) A person may have a single or many recurring attacks of hemoptysis *without assignable cause*, if we except severe muscular strain or intense mental excitement. Although pulmonary tuberculosis does not supervene in instances of this sort, yet not a few may be excited by a permanently limited tuberculous focus which may be indeterminable by the usual methods of examination. I have more than once seen a cure result from an active course of treatment with creosote and appropriate hygienic measures. In well-marked instances of the kind a complete arrest of the trouble resulted from a change of climate.

(*h*) *Parasitic hemoptysis* due to the paragonimus westermanii. The sputum resembles that of lobar pneumonia with intermittent free hemoptysis (for description of this condition, *vide* p. 1242).

Differential Diagnosis.—A reliable diagnosis necessitates the certain exclusion of hemorrhage from the higher air-passages, pharynx, esophagus, and stomach. In *epistaxis* the blood may directly enter the naso-pharynx, exciting cough and being discharged as in hemoptysis. An examination of the nasal chambers should be made when the symptoms are suggestive of epistaxis. Bleeding may take place from the gums, from chinks in the pharynx, or from varicose veins. If the seat of the bleeding be the *pharynx*, the hemorrhage is not free, the blood being commingled with a preponderating proportion of mucus; if from the gums, it may be more copious (as in ptyalism or scurvy), and the hemorrhage then simulates that of pulmonary hemoptysis. An inspection of the mouth will disclose whether or not the gums are the source of the hemorrhage. Strümpell distinguishes hysterical hemoptysis by the smaller bleedings, the absence of pus-elements, and the large amount of squamous epithelium, leptothrix, and the food-remnants present.

Hematemesis must be distinguished from hemoptysis, *vide* p. 783.

Prognosis.—The gravest apprehensions are constantly entertained by sufferers from hemoptysis, but immediately fatal results are of rare occurrence; and of this fact the patient should be repeatedly assured by the attending physician. In case, however, the existence of thoracic aneurysm is definitely known, the consequences of hemoptysis are certainly fatal. With reference to the effect of hemoptysis upon tuberculous pulmonary disease opinions differ widely; I am of the belief, however, that prior to the existence of cavities it exerts a favorable rather than an unfavorable influence upon the disease in most cases. On the other hand, in cases in which cavities exist at the time of the occurrence of hemoptysis an opposite effect is observed. The fact that hemoptysis often precedes by prolonged intervals of time the development of pulmonary lesions is no argument in favor of Niemeyer's view, that phthisis is caused by hemoptysis. There can be no doubt, however, that some blood finds its way into the bronchi below the point of bleeding and into the air-cells, causing, at times, irritation and even lobular inflammation. In this way hemorrhages may aid in rendering the tissues more susceptible to tuberculous infection. In cases of profuse hemorrhage, due to aneurysm or to the erosion of large branches of the pulmonary artery in phthisical cavities, death may be suddenly induced, and is caused largely by inundation of the lung and the consequent impossibility of respiration.

Treatment.—Since the hemorrhage is ascribable to (1) congestion of the bronchial mucosa, (2) erosion of the vascular walls, and (3) blood-changes, obviously the treatment of individual cases must be modified according to the character of the causative condition.

In many instances of *hemoptysis due to congestion of the bronchial mucosa* the hemorrhages are, comparatively speaking, slight; hence, apart from keeping the patient at absolute rest, little treatment is required. If not excessive, they are often salutary in their effect. If free, the physician's aim should be to decrease the power of the heart's contraction, and to accomplish this end the patient should be placed in bed, and not allowed to change his position nor to speak above a whisper. The diet should be light, nutritious, and non-stimulating, all hot drinks and alcoholics being prohibited. Among cardiac sedatives to be employed with a view to reducing the rapidity of the heart's action and lowering the blood-pressure, if the patient be neither feeble nor anemic, the ice-bag to the precordia is most valuable; if the pulse be full and strong, we may use aconite and other arterial sedatives. Arthur Foxwell[1] recommends venesection in cases in which *venous congestion* is present, and also lays stress upon measures that confine the blood to the systemic circulation—*i. e.* nutritious food, large doses of the nitrites, hot foot-baths, leeches to the anus, and ligatures applied to the thighs and arms. The pulmonary capillaries may also be effectually depleted by the use of salines. I have found dry cupping over the chest of the greatest service in cases dependent upon congestion. Eating ice and partaking freely of iced drinks are also useful measures. If the attack tends to become prolonged and exhausting, we may increase the coagulability of the blood by the use of gallic acid, acetate of lead, or calcium chlorid. Hemoptysis is usually accompanied by *cough*, that constantly disturbs the vascular serenity and excites fresh bleeding; it demands opium or morphin (hypodermically). In blood-spitting due to the gouty diathesis Mays recommends the salicylate of sodium. Tripier treats hemoptysis by using enemas of hot water (122° F.) night and morning.

When hemoptysis is associated with *organic disease of the heart*, the main indication is to strengthen that organ by bodily rest and quiet and by the use of cardiac tonics, especially digitalis. I have had under observation and treatment for several years a young physician who has been suffering from frequent, marked hemoptysis, due to mitral regurgitation, and in whose case the bleedings are readily controlled by the free use of digitalis.

When in *thoracic aneurysm* or *advanced pulmonary tuberculosis* the blood is ejected in mouthfuls, we may safely infer that erosion of a vessel or rupture of the aneurysm has taken place. Here the object is to bring about the formation of a thrombus that will arrest the hemorrhage. Perfect quiet in the horizontal position tends to allay the vascular excitement, and the induction of fainting by venesection is a measure worthy of a trial. Opium is contra-indicated in the latter class of cases, since if cough be checked inundation of the bronchial system with the blood (the chief danger) will be favored. R. H. Babcock gives an immediate injection of atropin sulphate (gr. $\frac{1}{50}$—$\frac{1}{25}$) when hemorrhage occurs from a cavity.

In all instances of hemoptysis treatment should not cease with cessation of the hemorrhage. A tendency to recurrence is manifested in many cases, and hence measures calculated to avoid this event must be brought into play. The patient should not be allowed to indulge in a stimulating diet; he should eschew tobacco and alcoholic stimulants, and avoid all physical and mental strain. Every source of bronchial irritation should be carefully avoided, and attacks of bronchitis, however mild, should receive the most careful attention. Moderate exercise is serviceable, as well as a liberal amount of nutritious food.

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[1] *British Medical Journal*, 1894, p. 194.

PNEUMORRHAGIA.

(Pulmonary Apoplexy.)

Definition.—An escape of blood into the air-cells and interstitial tissue, with or without laceration of the pulmonary parenchyma.

Pathology.—It may be, though rarely, (*a*) diffuse, when the lung-tissue is lacerated, as in cerebral apoplexy; or it may be (*b*) circumscribed, as when the blood is effused into the air-cells and the interstitial tissue, without rupture of the parenchyma. The latter form will be considered in the discussion of Pulmonary Infarction.

Etiology.—*Diffuse pulmonary apoplexy* is caused by the rupture of a thoracic aneurysm that has become adherent to the surface of the lung. Its most common cause is traumatism, especially penetrating wounds of the lung, but adult life and the male sex are to be regarded as predisposing factors. The lung-tissue is sometimes the seat of diffuse hemorrhagic infiltration in septico-pyemia and cerebral disease.

Symptoms.—These are ill-defined. *Profuse hemoptysis, urgent dyspnea,* and *cyanosis,* followed by increasing evidences of *collapse,* together with a clear history, should raise suspicions of the existence of diffuse pneumorrhagia.

The *physical signs* are indicative of extensive consolidation arising suddenly, and not of the nature of the lesion.

The **prognosis** is practically hopeless, and abscess or gangrene may result if these cases recover from the immediate effects of the hemorrhage.

Treatment.—Absolute rest of the body in the horizontal position is the one measure that offers a slight prospect of alleviation, for thus the formation of a clot, followed by arrest of the hemorrhage, is encouraged. It is unwise to use opium to allay the cough, since the action involved assists in ejecting the extravasated blood, which will, in consequence of gravitation and the effect of respiration, submerge speedily so much of the lung-tissue as to hasten the fatal termination. Ergot is not to be given hypodermically, since it raises the blood-pressure in the lesser circulation, but the internal and external use of cold has been highly recommended. With the onset of collapse cardiac stimulants become absolutely necessary, though many cases are so rapidly progressive as to reach a moribund state before remedial agents can be applied by the physician.

PULMONARY EMBOLISM.

(Hemorrhagic Infarction; Embolism of the Lungs.)

Pathology.—Embolic infarctions are firm, airless, brown or black, wedge-shaped masses, with their bases usually at the pleura, which soon becomes lustreless and covered with a delicate layer of fibrin. The infarctions may be single or multiple, and sometimes occupy the greater portion of the lobe; in the majority of cases, however, their size equals that of a walnut. Their most frequent seat is at the back of the lower lobe. The microscope shows the presence of leukocytes and red blood-corpuscles in the air-cells and in the alveolar septa. Collateral congestion and edema are frequent concomitants, and, less frequently, pneumonic consolidation appears.

Etiology.—The condition is produced by the blocking of the pulmonary arteries by an embolus or thrombus. When the circulation in the pulmonary capillaries is feeble, hemorrhagic infarction may be the result of stasis, and this is probably the most frequent form. It is met in diseases of the lungs, and also in mitral affections. The plug that occludes the blood-vessel may be composed of leukocytes, as in leukocythemia, and the chief sources of the emboli are the thrombi in the right heart, in consequence of dilatation, and in the systemic veins. Infectious emboli, resulting in abscesses, occur (*vide* Abscess of the Lungs). An embolism of placental cells in cases of eclampsia has been described. Occlusion of a branch of the pulmonary artery cuts off completely the circulation to the territory supplied by that branch, and hemorrhagic infarction occurs—venous extravasation, with expression of air.

Symptoms.—Not all infarctions give rise to symptoms; on the contrary, occlusion of a main branch of the pulmonary artery usually terminates life speedily. The latter accident occurs not infrequently in connection with organic disease of the heart, and if death be not the immediate result or if a narrower branch be occluded, alarming symptoms ensue, such as *syncope*, *dyspnea*, *pain in the side*, and *convulsions* with *unconsciousness*. The first and most distressing symptom is dyspnea, which is attended by frantic efforts at breathing and by great mental anxiety. Occasionally *hemoptysis* is an early symptom, and of primary significance if it occur in a patient suffering from mitral disease. If, together with these symptoms, loss of consciousness with convulsions occurs, the diagnosis becomes wellnigh complete. *Cough* usually supervenes, accompanied by the expectoration of dark, gelatinous, mucoid masses. Large lymph-cells containing blood-corpuscles are found in the sputum, these giant-cells being most commonly seen in instances of organic cardiac affections. They are supposed to transform the blood-corpuscles into pigment-matter.

The **physical signs** may either be negative—as, for example, when the infarctions are small or deeply located—or they may give information as to the seat and extent of the affected part. When present they are the symptoms of sharply-localized consolidation (increased fremitus, percussion-dulness, moist râles, and bronchial breathing), and it is not improbable that in many cases the physical signs are due, in great part, to associated conditions, such as bronchitis, edema, or collateral consolidation. The appearance of the friction-sound in the course of suspected cases is a great aid in diagnosis. The *heart's action* becomes enfeebled, the *pulse* is small and frequent, and the surface of the body is cool and frequently bedewed with cold sweat. *Fever* may either be present at the onset or absent throughout. The signs of embolic abscesses in the lungs will be elsewhere detailed (*vide* Pulmonary Abscess).

Diagnosis.—To establish the diagnosis of pulmonary embolism there must be a clear history of some etiologic condition, and the sudden appearance of such symptoms as dyspnea, cough, bloody expectoration (in particular), chest-pain, loss of consciousness, and convulsions, corroborated by the physical signs of a sharply-defined spot or spots of consolidation.

Prognosis.—The prognosis differs with the character of the primary condition. On the whole, it is exceedingly grave, though the absorption of an embolism, followed by the disappearance of the urgent symptoms, is not impossible. In case death does not occur soon, infarcts may give rise to abscess or gangrene, the result either of the presence of bacteria in an original embolus or of their entrance through the air-passages. In other cases an infarct may undergo fibroid change and contraction, and may even calcify.

Treatment.—Beyond procuring absolute rest of the body and a relief from the distressing symptoms, the treatment should be aimed at the affections on which this form of embolism depends. Dyspnea and pain may require the hypodermic use of atropin and morphin, preferably in combination. Heroin relieved the dyspnea in one of my cases.

CHRONIC INTERSTITIAL PNEUMONIA.

(*Fibroid Induration; Cirrhosis of the Lung.*)

Definition.—A chronic inflammation of the lungs, characterized by the formation of fibrous or connective tissue. It may occur as a primary or as a secondary affection.

Pathology.—Two leading forms of cirrhosis of the lung may be recognized: (*a*) *Local*, and (*b*) *Diffuse*, though these do not demand separate description. It is a unilateral affection, and the lung of the side involved is much shrunken, its dimensions in some cases being incredibly small. I have seen an instance in which the organ measured four inches in its longest and less than three in its shortest diameter. It lies tightly against the spine, and has frequently been overlooked. The heart occupies the affected side, being drawn in that direction during the progress of the disease, and it is enlarged, principally owing to hypertrophy of the right ventricle, and the pulmonary artery is the seat of atheromatous change. The other lung is overdistended (*compensatory emphysema*), and may encroach upon the mediastinum. Intrapleural and pleuro-pericardial adhesions may be exceedingly firm and thick on the one hand, and only moderately so on the other, though rarely the pleuræ are intact. The cut surface of the affected lung is hard, dry, airless, shiny, and usually light-gray in color (rarely, reddish-yellow), and the lung-tissue cuts with great resistance. The mouths of the blood-vessels and bronchi, which are often greatly dilated (bronchiectatic), may be observed gaping in the cut section. Cavities may be wholly or in part due to the superaddition of a tuberculous process, though even when the affection is non-tuberculous they may be quite numerous. Phthisical cavities may often be discriminated by their usual situation at the extreme apex. The lung that is unaffected by the fibroid process is also quite often the seat of tuberculous change.

Etiology.—The disease is almost invariably secondary, and very generally accompanies prolonged inflammatory and chiefly local changes in the lungs. It may also follow acute inflammatory processes. Examples of **localized interstitial pneumonia** are seen in connection with pulmonary tuberculosis, emphysema, syphilis, hydatids, and fibroid induration secondary to thickening of the pleura.

Diffuse interstitial pneumonia has a variety of causes: (*a*) It may follow *acute lobar pneumonia* in cases in which resolution is delayed, and here the fibrinous exudate filling the air-cells becomes organized into connective tissue. Fibrous tissue is also substituted for the alveolar walls. The condition is exceedingly rare, and no instance of the sort has fallen under my own observation.

(*b*) Pneumonia, appearing as a complication in influenza, is very liable to produce chronic interstitial pneumonia.

(*c*) The disease may also result from atelectasis due to compression, as by aneurysms or neoplasms.

(*d*) It most frequently, however, follows *broncho-pneumonia* in either its acute or subacute form (Charcot). The process starts in the bronchi and extends to the surrounding lung-tissue, till finally an entire lobe, or even an entire lung, may become involved. Tuberculous broncho-pneumonia also leads to the production of new fibrous tissue, but here the process is a conservative one (*vide* Pulmonary Tuberculosis), and hence is not to be classed with chronic interstitial pneumonia.

(*e*) The initial lesions may be located in the *pleura*, and the lung become involved as a sequel, and the principal lesions may be located in the adherent pleural membrane, with bands of connective tissue extending into the lung. The bronchi are inflamed and sometimes dilated.

Chronic interstitial pneumonia may, however, exist without implication of the pleura, and in view of this fact the primacy of pleural thickenings cannot be granted without reserve when they form a part of the lesions of fibroid induration.

The various forms of the disease thus far described arise *secondarily*. It may also occasionally originate as a *primary* affection (1) from the inhalation of different forms of dust (*vide* Pneumonokoniosis). (2) Delafield describes "a special form of lobar pneumonia." He contends that lobar pneumonia terminates only in resolution or in death, and that this special disease, with its production of newly-formed connective tissue, is a special form of inflammation. The variety described by Delafield runs a subacute or even chronic course, and terminates by crisis. It is an exudative inflammation, with the formation of new tissue from the onset; but the consolidated areas are not so large as in ordinary pneumonia, and cut sections lack the granular character of the latter.

Symptoms.—The patient suffers from *cough*, which increases in intensity with the progress of the affection. There is a mucous, sero-mucous, or rarely bloody expectoration; *dyspnea* occurs early, and frequently is present only on ascending heights; and uneasiness, or even *pain*, over the side of the chest involved may be experienced. In cases in which the bronchi become dilated the characteristic symptoms of bronchiectasis are superinduced. The *general symptoms* consist merely in a loss of flesh and of strength. *Fever* is altogether absent.

Physical Signs.—*Inspection.*—The chest-wall of the affected side is retracted, while the healthy lung is enlarged (*compensatory emphysema*). The spinal column is curved laterally. The affected side is fixed during respiration, and the heart is displaced by traction toward the affected side. If the left lung be involved, the apex-beat will be displaced to the left and slightly upward; if the right, the apex-beat will be observed to the right of the sternum. The ribs approximate, thus obliterating the interspaces, and the shoulder droops over the shrunken chest-wall.

Palpation.—The tactile fremitus is usually increased; if the pleura be much implicated or thickened, however, fremitus may be decreased. Palpation discovers no expansile motion.

Percussion.—The percussion-note varies. Dulness is common, owing to consolidation of the lung, but flatness is sometimes met with, and a tympanitic or amphoric note is occasionally elicited over a dilated bronchus.

Auscultation.—The breathing is bronchial or more or less sonorous as a rule, and over bronchiectatic cavities it is cavernous or, rarely, amphoric. Near the base it is frequently feeble, distant, or even altogether suppressed. Subcrepitant, sonorous, sibilant, or gurgling râles may be audible, and dry, creaking, or leathery friction-sounds may also be heard.

Prognosis.—The course of the complaint is exceedingly chronic, and lasts over many years. Death may result from an intercurrent attack of acute pneumonia affecting the other lung. The disease always shortens the duration of life, and it may be the direct cause of death. Rarely a fatal issue is due to dilatation of the right heart, followed by tricuspid regurgitation.

Treatment.—The condition is incurable. The patient should, however, be placed under the best sanitary conditions, and if practicable he should make a permanent change of climate. A suitable resort should be selected in accordance with the rules indicated in the treatment of Pulmonary Tuberculosis, and every effort should be put forth to improve the general nutrition of the patient. Due attention should be given to the associated bronchitis, as well as to any symptoms that may arise during acute exacerbations.

BRONCHO-PNEUMONIA.

(*Capillary Bronchitis; Catarrhal Pneumonia.*)

Definition.—An inflammation of the minute bronchi and air-vesicles, due either to the extension of inflammation from the capillary bronchi to the air-vesicles or to an inflammatory process set up in atelectatic lobules.

Pathology.—Macroscopically, the lungs present decided variations in persons who have died of broncho-pneumonia. On the pleural surface may be noticed purplish or slaty patches, often sunken (atelectasis), intermingled with the more elevated patches of healthy lung and grayish consolidation, and smoother and more moist than croupous pneumonia. Similar appearances are presented by the cut surface. On pressure fluid exudes—edematous from the healthier areas, and grayish and puriform from the consolidated areas. The mucosa of the large bronchi may look natural, though frequently it is congested, while the small bronchi usually contain more or less muco-purulent material. Their walls are greatly thickened, and on section the cut surface presents a nodular appearance. Dilatation of the smaller bronchi may be observed, and minute consolidated areas, varying in size from that of a pin's head to that of a pea, may be seen surrounding the thickened walls of the bronchi. When, as frequently happens, they become confluent, large areas—an entire lobe and even an entire lung—of lung-tissue may become consolidated. The solidified zones are firm to the touch, being destitute of air, and at first they contain blood; hence their color is a dark-red, but later it presents a grayish hue. The condition is usually bilateral. As a rule, the bronchial glands are swollen and inflamed. In the non-consolidated portions of the lung the air-cells are found to be considerably dilated.

The essential lesion is a productive inflammation of the bronchi and of the immediately surrounding air-spaces. The inflammation is from the first not exudative, but productive; that is, with the formation of new tissue (Delafield). This form of inflammation may merge into sclerosis of the lung or chronic thickening of the pleura. *Microscopically*, the walls of the bronchioles and alveolar passages are seen swollen and infiltrated with cells; they likewise contain plugs of mucous exudate, most marked near the centre of the process. The air-cells toward the periphery show much less exudate. The latter consists of serum, some mucus, and many swollen cells from the alveoli (soon showing fatty degeneration), leukocytes, and also red blood-cells in small numbers. Fibrin is seen in small quantity if at all.

In deglutition- and aspiration-pneumonia the leukocytes are present in much larger numbers, and the exudate tends to suppuration, while in the hemorrhagic forms the red blood-cells are relatively increased.

Kikodse[1] found the blood in broncho-pneumonia to contain an increased number of white corpuscles, except in fatal or very severe cases. The cause of this increase appears to be the return into the circulation of the corpuscles that have passed into the alveolar spaces; hence it ceases after the fever declines.

Among the associated lesions that remain to be mentioned are—(*a*) Catarrhal inflammation of the mucous membrane of the bronchi; and (*b*) Exudative inflammation of the air-cells, which become filled with epithelium, fibrin, and pus, with resulting consolidation of the lung. The epithelial cells lining the air-sacs, since they are more numerous in young children than in adults, form a larger part of the inflammatory exudate in the former than in the latter. (*c*) The pulmonary pleura is often coated with fibrin, but less regularly than in croupous pneumonia.

Etiology.—(1) A marked predisposing influence is *age*, the disease being most prevalent amongst young children. In them it may appear in association with measles, whooping-cough, scarlet fever, and diphtheria, but not infrequently it is entirely independent of these diseases. Infants are especially susceptible to the affection, most instances of pneumonia at this period of life being of the lobular form. Other conditions that act as predisposing factors in children are improper exposure to cold, unsanitary surroundings (especially impure air), rickets, and chronic diarrhea. Broncho-pneumonia is also frequent in the aged, often being occasioned by certain debilitating causes and chronic diseases that are common to advancing years (emphysema, gout, chronic valvulitis).

(2) *Season.*—The affection prevails especially in the winter and spring months; particularly is this the case in those forms that are unassociated with the acute infectious group of diseases.

(3) It also supervenes as a complication in such acute infectious diseases as influenza, typhoid fever, erysipelas, and small-pox, and is of

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[1] *Annual of the Universal Medical Sciences*, 1892, vol. i. sec. A.

serious import. According to my own observations, it is more commonly met with in the diseases above mentioned than is lobar pneumonia.

(4) The *inhalation of food-particles and other substances* often serves to convey the agents of inflammation to the lobules of the lungs. A long-continued recumbent posture predisposes the patient to broncho-pneumonia. It is, however, in conditions in which the larynx and bronchi have totally or in part lost their sensitiveness—as in coma due to apoplexy, uremia, and allied cerebral states—that retention of bronchial secretions occurs, and that, owing to gravitation, these secretions reach the minute bronchi. Pneumonia is similarly produced when we cut the vagus nerves, the paralyzed structures permitting irritants to be carried to the lung by inspiration. *Inhalation pneumonia* may follow operations upon the nose, mouth, larynx (tracheotomy particularly), and is often secondary to carcinoma of the larynx and esophagus. It is also the pneumonia of new-born children.

(5) It must not be forgotten that quite commonly broncho-pneumonia is caused by the *tubercle bacillus* (*vide* Pulmonary Tuberculosis).

Bacteriology.—Weichselbaum has shown the presence of streptococci with the greatest frequency in the usual, secondary form. The pneumococcus is often found, and in a goodly number of cases the staphylococcus aureus (Neumann), while in influenza the specific organism may itself cause broncho-pneumonia (Pfeiffer). Numerous other organisms have been found (typhoid bacillus, bacillus coli communis). Mixed infection with the Diplococcus pneumonia is almost the rule when lobular pneumonia is secondary to the acute infections (diphtheria, typhoid fever, influenza).

Symptoms.—Two clinical forms may be distinguished:

(*a*) **Primary broncho-pneumonia** is met with generally in adults, and presents, in great part, the symptoms of an acute bronchitis of severe grade (*cough*, *dyspnea*, *pain*, *fever*). When occurring in weakly subjects the onset may be gradual. The cough is attended with *expectoration* (glairy and tenacious) that may be blood-tinged in the form of droplets or points. The fever is moderate, the temperature ranging from 101° to 104° F. (38.3°–40° C.), and is of irregular type; in severe cases, however, continued high temperature may occur. Physical examination gives the same result as in the secondary form. The *duration* is from two to four weeks, the fever terminating by *lysis*. West holds that primary broncho-pneumonia in children is of pneumococcus origin.

(*b*) **Secondary broncho-pneumonia** is the variety usually met with. The symptoms are frequently veiled by those of the primary affection, and, indeed, a moderate grade of lobular pneumonia is frequently unsuspected during life when arising in the course of other grave diseases.

It is usually preceded by bronchitis affecting the larger bronchi, and in this common event the first symptom that directs attention to the disease is the *sudden increase* in the frequency of the *respirations*, which rise as high as 60 or even 80 per minute. An initial chill is rare. *Fever* develops suddenly, or, if previously present, increases rapidly. An early symptom is the *cough*, which is usually hard, harassing, frequently painful, and accompanied by *expectoration*. The *pulse-rate* is abnormally frequent, and in the later stages may be quite rapid, feeble, and irregular. The type of the fever is similar to that of the primary form.

Physical Signs.—At the beginning of the attack the only sign is the presence of subcrepitant and sibilant râles, pointing to a general capillary bronchitis. Shortly larger or smaller areas of consolidation become manifest. At first rapid breathing, and soon cyanosis, affecting first the lips and conjunctivæ, may be observed; later, the face becomes dusky and the finger-tips blue. *Palpation* shows defective expansion and increased tactile fremitus over the consolidated areas. The *percussion-note* is dull or, less frequently, hyperresonant if the area be small. *Auscultation* reveals numerous fine, subcrepitant râles, corresponding to the consolidated portions. The respiratory murmur may be bronchial, though more often broncho-vesicular. The signs are usually noted in both lungs.

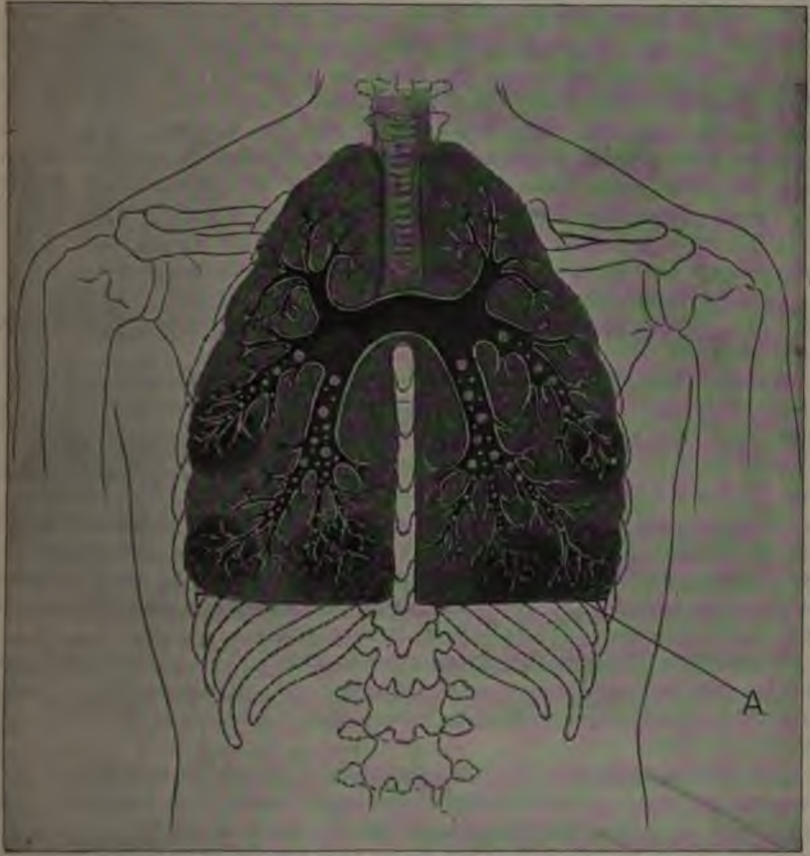

FIG. 43.—Illustrating broncho-pneumonia. The dark spots represent the consolidated areas; the white dots indicate râles; *A*, coalescence of two areas of consolidation.

Duration.—(1) In children this varies considerably in different cases. Rarely do fatal instances last more than two or three weeks, while they may be as brief as two or three days. On the other hand, cases in which recovery ensues frequently last from six to eight weeks, though in some instances from one to three weeks only. Two special forms demand brief description:

(*a*) The *cerebral*, in which restlessness, convulsions, and delirium become so marked as to overshadow entirely the pulmonary symptoms. Not infrequently the onset is characterized by convulsions, high fever, prostration, and alternating stupor and delirium. After such symptoms have continued for from two to five days, pulmonary symptoms appear, while the cerebral decline.

(*b*) Other cases may manifest a *subacute onset*, in which there is anorexia and occasional vomiting, with the nervous symptoms before noted.

(2) The *protracted forms* are those in which (*a*) the symptoms of acute broncho-pneumonia give place to those of a similar though chronic state. The general disturbances may not be marked in some instances, but usually there are cough, loss of appetite, or inability to gain in flesh and strength, and the signs of consolidation persist. (*b*) Those presenting fever of an irregular type, together with decided prostration, in addition to the symptoms of the preceding variety. In many cases belonging to this form the lesions are tuberculous.

In adolescence the cerebral symptoms are not as well marked as in children. Two anomalous varieties are met with in practice that demand brief separate description:

General Broncho-pneumonia.—The attack develops suddenly and is severe. There are chills, high fever, marked prostration, headache, chest and loin pains, a rapid pulse (soon becoming feeble), rapid and labored respirations, cyanosis, restlessness, delirium, and cough that is at first dry, and followed by mucous, muco-purulent, blood-tinged sputum.

The physical signs are defective expansion and an increased tactile fremitus. The percussion-note may be either normal, tympanitic, or dull; the auscultatory signs are large moist, subcrepitant, crepitant, sibilant, and sonorous râles over both lungs, and a harsh or broncho-vesicular respiratory murmur. The affection is very grave.

Resembling Tuberculous Broncho-pneumonia.—The symptoms appear slowly, and the case pursues a subacute or even chronic course. Cough, catarrhal expectoration, moderate fever (often of a hectic type), and night-sweats are noted.

Physical examination discloses generalized bronchitis, coupled with circumscribed areas of consolidated lung-tissue. Resolution may take place at the end of eight or ten weeks, and complete recovery ensue; when, however, this favorable event does not occur, the case drags on for an indefinite period, and finally terminates fatally. There are no bacilli in the sputum.

Diagnosis.—This can be arrived at by considering—

(*a*) The nature of the antecedent affections and their etiologic circumstances;

(*b*) The distribution of the consolidated areas in both lungs;

(*c*) The fact that the physical signs of consolidation are subsidiary to those of generalized bronchitis;

(*d*) The intense dyspnea and cyanosis;

(*e*) The type of the fever, irregular as a rule, and its gradual decline;

(*f*) The frequent long duration.

Differential Diagnosis.—Doubtless, *lobar pneumonia* is constantly mistaken for broncho-pneumonia, and particularly when, in the latter disease, a large portion of one or both lungs becomes inflamed in consequence of the coalescence of small areas of consolidation. The points of distinction may be tabulated as follows:

BRONCHO-PNEUMONIA.	LOBAR PNEUMONIA.
Etiology.	
Presence of pathogenic organisms [1] (streptococci).	Presence of the Diplococcus pneumoniæ.
Usually secondary to bronchitis and acute infectious diseases (*e. g.* measles, whooping-cough).	Usually a primary disease.
Clinical History.	
Onset gradual.	Onset abrupt; previous health generally good.
Fever is, in proportion to the extent of inflammation, of irregular type, and declines by lysis after a variable duration.	Fever is high, of continued type, and falls between the fifth and ninth days by crisis.
Sputum glairy, tenacious, and in adults may be blood-tinged.	Sputum characteristic (rusty or prune-juice).
Dyspnea and evidence of carbon-dioxid poisoning prominent.	Respiration panting, but dyspnea and cyanosis relatively less marked.
Physical signs of generalized bronchitis always marked, and usually preponderating over those of consolidation.	Signs of bronchitis generally absent, those of lobar consolidation always preponderating.
Consolidation commonly bilateral.	Commonly unilateral.
Duration indefinite, often extending over many weeks.	Duration definite as a rule, convalescence following crisis.
Consolidated areas liable to become the seat of tuberculous infection.	Far less likely to become the seat of tuberculous infection.

It may be difficult to distinguish *tuberculous broncho-pneumonia* from the disease under consideration. Indeed, a non-tuberculous broncho-pneumonia may be located at the apex of the lung and accurately simulate the symptoms and signs of the tuberculous form. The differentiation is to be based upon the presence or absence of the signs of softening, and upon a microscopic examination of the sputum (which in a child may be vomited). The softening in tuberculous pneumonia does not, however, begin very promptly; but if elastic fibers and tubercle bacilli be found, the diagnosis is at once set at rest.

Prognosis.—In broncho-pneumonia the severity and gravity of the symptoms and the extent of the involvement of lung-tissue are proportionate to one another; hence it follows that the disease may either be devoid of serious import or it may be fraught with great danger to life. Its course is subject to decided fluctuations, the periods of exacerbation in the symptoms often marking the time of the development of the gravest features. Apart from the extent of the lung-tissue involved, however, we must consider especially the condition of the patient at the time of invasion. If the constitution have been previously undermined, as is frequently the case in children, broncho-pneumonia is very apt to be fatal. The disease is less dangerous when it develops in the course of, or follows, measles than when secondary to whooping-cough, influenza, or diphtheria. Wiry, thin children seem to stand broncho-pneumonia better than fat, flabby ones (Osler). *Deglutition* and *inspiration* lobular pneumonia, especially when occurring after operations upon the larynx or trachea, are frequently fatal. The mortality-rate in this disease varies from 25 to 50 per cent.

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[1] The diagnostic value of the discovery of streptococci is not pronounced. Numerous other organisms have been found in broncho-pneumonia in their absence, and a similar organism (*Streptococcus pneumoniæ*, Weichselbaum) has been found in a number of cases of croupous pneumonia.

Treatment.—Prophylaxis.—There are few diseases that can be so effectually prevented as can broncho-pneumonia. In the first place, proper attention to the mouth as well as to the position of the patient (which should be changed frequently) during attacks of acute infectious diseases will prevent its development in a great proportion of this large class of cases. Adequate protection against exposure to cold during convalescence from measles, whooping-cough, etc. is also a potent factor in preventing the disease, as is the timely handling of catarrhal affections of the nose, pharynx, larynx, and larger bronchi.

Treatment of the Attack.—Certain *sanitary arrangements* are of the utmost practical importance. The sick-room should be well ventilated and its atmosphere kept at a uniform temperature—68° to 70° F. (20°–21.1° C.). The air of the room should also be well laden with moisture, which may be generated from a croup-kettle or other suitable vessel.

Local Measures.—In young children the chest should be enveloped in a jacket-poultice of linseed meal, which should be covered with a layer of oiled silk or waxed paper so as to prevent its growing cool. This should be renewed at intervals of about six hours. After the more active symptoms have subsided the linseed jacket-poultice may be replaced by one of absorbent cotton, which should also be covered with oiled silk or wax paper. In older subjects the application of iced poultices to the chest exercises a most favorable influence, not only upon the local inflammation, but also upon the fever and the nervous symptoms.

General Measures.—High fever calls for tub-baths, the temperature of the water at first being set at 95° F. (35° C.), and then gradually cooled to 75° or 80° F. (26.6° C.). The gradually cooled bath or the cold pack may be used two or three times daily. The effects are to reduce temperature, to promote refreshing sleep, and to improve the character of the respiration. This mode of treatment is especially effective in cases that begin abruptly. In such the tincture of aconite or veratrum viride may be employed temporarily. In cases presenting moderate pyrexia cold spongings, combined with the use of the ice-bag to the head, may suffice. The following fever-mixture may be employed, though it is not to be regarded as a substitute for the cold-water method of treatment, but is merely supplemental to the latter:

℞. Potassii citrat., ʒijss (10.0);
Spts. ammon. aromat., fʒij (8.0);
Spts. æther. nitrosi, f℥ss (16.0);
Liq. ammon. acetat., f℥iij (96.0);
Glycerini, q. s. ad f℥iv (128.0).—M.
Sig. ʒj (4.0) every two hours for a child of five years.

In *children* a mild mercurial purge at the outset is advantageous, and subsequently by the use of salines or glycerin suppositories a daily evacuation of the bowels is to be secured.

The Diet.—The bodily strength is to be maintained by careful, methodical feeding, milk, eggs, albumin, and broths being the best forms of food. The milk should be predigested if there be marked pyrexia, and egg-white may be given in cold water or as egg-lemonade. The cough is often wellnigh constant and very distressing. Frequently the use of remedies that promote secretion, combined with a small dose of opium, will, under these circumstances, afford relief. A useful formula is the following:

℞. Vini antimonii, ʒj (4.0);
Spts. æth. nit., ʒijss (10.0);
Tr. opii camph., ʒijss (10.0);
Liq. ammon. acetat., q. s. ad ℥ij (64.0).—M.
Sig. ʒj (4.0) every two hours, diluted, for a child of from three to five years.

Dover's powder is also of value in relieving the cough. When the expulsion of the sputum is attended with great difficulty the preparations of ammonium often meet the indications. Of these the muriate is the most effective, but, unfortunately, this is often objected to, and we must then rely upon the carbonate or the aromatic spirits. The bronchi may contain an abundance of secretion that cannot be expelled, despite the use of the above measures. Under these circumstances an emetic may be given, composed of the wine of ipecac (ʒj—4.0), combined with alum (gr. xx to xxx—1.296–1.944), and administered to a child every ten or fifteen minutes until emesis occurs.

Cardiac stimulants (alcohol and strychnin) are required if the pulse fails. The preparations of ammonium owe much of their reputation in this disease to their stimulating properties. These agents when boldly used may suffice to re-establish the cardio-pulmonary circulation; but if they fail in this and cyanosis supervenes, oxygen by inhalation should be used also. Sudden heart-exhaustion may occur, associated with mucous râles in the larger bronchi and rapidly increasing cyanosis. Alternating douching with hot and cold water and electricity should be given a trial. Injections of salt solution increase arterial tension and act as a "whip" to all emunctories; they may also stimulate phagocytosis, and should be used in serious cases. In streptococcic bronchopneumonia anti-streptococcus serum may be tried.

PULMONARY ATELECTASIS.

(*Collapse of the Lungs; Compression of the Lungs.*)

Definition.—Atelectasis of the lungs is a condition occasioned by the removal of the air from the air-cells—a state directly the opposite of emphysema. The air disappears largely in consequence of the process of absorption.

Pathology.—The affected lung-spots sink in water, being non-crepitant. They also present through the pleura a bluish-red tint, and on cross-section a brownish-red color. The surface of the affected areas is smooth and depressed below the level of the adjacent lung-structure. The bronchi supplying the collapsed parts are frequently occluded by inflammatory products, but in all cases, as shown by Legendre and Bailly, the latter may be inflated by means of a blowpipe.

Apart from more or less distention of the pulmonary capillaries with blood, there are no ***histologic*** changes in the atelectatic areas, though they are of firm consistence (splenization, carnification). There can be no longer any doubt as to the entire propriety of the pathologic distinction between lobular pneumonia and atelectasis.

Etiology.—The condition occurs most frequently in the new-born, and is then due to defective respiration. Thus in children dying soon after birth the lower lobes may be found to be atelectatic. When acquired, however, there are three modes of production: (1) The first step consists in a more or less complete plugging of the smaller bronchi with muco-pus and other products of bronchial inflammation. If complete, air can no longer enter on inspiration, and as the contained air gradually becomes absorbed atelectasis is the natural result. This condition is very commonly associated with broncho-pneumonia, especially in children. (2) A frequent mode of origin is through compression of the lungs, resulting from positive intrathoracic pressure, after the normal contractility of the lung has been overcome. Instances of this may be produced by pleural effusion, hydrothorax, pneumothorax, pericardial effusion, great cardiac hypertrophy, a solid tumor, or an aneurysm of the arch. Not infrequently abdominal tumors, excessive meteorism, and ascites make sufficient upward pressure against the diaphragm to cause compression of the lower lobes of the lungs. (3) Conditions that weaken and obstruct the inspiration may produce this disease, such as certain brain-affections, paralysis of the pneumogastric, and paralysis of the chest-walls. Thoracic deformities may produce pulmonary atelectasis, and in extreme grades of kyphoscoliosis the lung occupying the side corresponding to the convexity of the spinal column is small. Whilst the lung-expansion and the growth of the organ are greatly interfered with, however, and particularly if the condition arises in youth, true atelectasis rarely occurs from this cause, owing to the natural retractility of the lung. Among conditions arising from deformities of the chest is the so-called aplasia of the lungs.

Symptoms.—Atelectasis is a secondary condition, and its symptoms are very generally veiled by those of the primary disease. It arises frequently in the course of broncho-pneumonia, but passes unnoticed unless it becomes very extensive. *Respiration* is carried on by the upper and anterior portions of the lungs, is increased in frequency, and is laborious. The *pulse* is small, rapid, and feeble; the *skin-surface*, especially that of the extremities, is cool.

The form presenting the most typical symptoms is that occurring in the new-born. It is evidenced by *shallow*, *rapid breathing*, *lividity*, *cold extremities*, a *faint whining cry*, *drowsiness*, and sometimes by evidences of *motor irritation*, such as muscular twitching and convulsions. Congenital anomalies of the circulatory organs are associated.

Physical Signs.—When it involves a goodly portion of the lower lobes posteriorly, as frequently happens, there is marked retraction during inspiration over the lower portion of the thorax, due partly to external atmospheric pressure, and partly to the contractile efforts of the diaphragm. Dulness on percussion is revealed, though only when the atelectasis is extensive, and the tactile fremitus, though very various, is generally decreased. Localized compensatory emphysema may present semi-tympanitic resonance over small areas of collapse.

Auscultation shows a greatly diminished or absent vesicular murmur, and, if the area of collapse be large, bronchial breathing. Among associated sounds is the subcrepitant râle, due to broncho-pneumonia, and, indeed, capillary bronchitis and atelectasis are often combined, there being, moreover, no reliable signs that will separate them clinically.

The *aplasia* of the lung that is produced by spinal curvature (*kyphoscoliosis*) richly deserves brief separate description, owing to its clinical importance. In many instances the chest is more or less twisted on its own axis, shortened in the vertical diameter, and thoroughly fixed. Under these circumstances lung-expansion is impossible, and hence respiration is purely diaphragmatic. In many other patients life may be prolonged for an indefinite period, nothing more being observed than slightly labored breathing. Such persons, however, upon great physical exertion suffer from urgent dyspnea, and the development of an ordinary bronchitis may lead to similar results, and even to speedy death.

The **physical signs** are those of localized emphysema, combined with those of more or less compression of the lungs. There is an extension of the cardiac dulness to the right, and other evidence of right ventricular enlargement, to which may succeed dilatation with the usual clinical events produced by the latter condition. Death is not rarely due to this failure of compensation.

Autopsies have shown the lungs to be small and more or less compressed, some portions being almost airless. Areas of emphysema of the lungs are often associated. The right ventricle may be found to be hypertrophied merely, or dilatation may also have taken place. Congenital atelectasis, by keeping up high pulmonary pressure, may lead to a persistence of the ductus Botalli and of the foramen ovale.

Diagnosis.—Atelectasis may be distinguished from *lobar pneumonia* by the absence of an initial rigor, fever, crepitant râles, and the pain of the latter disease, and by the characteristic inspiratory retraction of the lower portions of the chest and the smaller areas of dulness.

Pleuritic effusion gives a flat percussion-note, the upper level of which varies with a change in the position of the patient—a sign that is wanting in atelectasis.

Prognosis.—When the condition is limited to small areas it is rarely serious, but equally seldom does extensive atelectasis lead to recovery. The outlook depends to some extent upon the nature of the associated affections; thus, when it is secondary to whooping-cough and widespread broncho-pneumonia, it is very fatal. Other diseases that may complicate and increase the gravity of the atelectasis are pleurisy and pulmonary tuberculosis. On the other hand, compensating emphysema often coexists, and is to be regarded as salutary in its effects. When due to *compression* by pyo-pneumothorax, tumors, and the like, the prognosis is especially gloomy.

Treatment.—The treatment corresponds with that of the primary disease. *Capillary bronchitis*, which is so apt to be followed by collapse of the lobules, must receive active treatment, and prophylactic measures are of the utmost practical importance. The patient should be instructed to practise full inspiration at regular intervals; he should not be allowed to lie continuously in the dorsal decubitus, but should be told to change his position frequently. Another measure that may effectually prevent the development of atelectasis is the use of cold shower-baths (*i. e.* a stream of cold water poured over the region of the neck), and this can sometimes be depended upon as a curative agency when the condition already exists. Tonics and the judicious use of stimulants, together with a nourishing diet, are invariably required. I have also seen good results follow the inhalation of compressed air and of oxygen.

In *kyphoscoliosis* tepid baths are indicated. The heart-condition demands careful attention, and cardiac stimulants are to be resorted to at the first loss of compensation or when compensation fails to become established.

EMPHYSEMA.

Definition.—In general this term implies the presence of air in the interstitial alveolar tissue. As applied to the lungs, however, two forms are recognized: (1) Interlobular; and (2) Vesicular, an abnormal dilatation of the alveoli.

INTERLOBULAR EMPHYSEMA.

This is produced by the rupture of the air-cells, the air contained in the lung escaping into the interlobular connective tissue. Among its causes are—(*a*) Injuries of the lung (usually by a fractured rib) and perforating wounds of the chest; (*b*) Violent paroxysms of coughing, as in whooping-cough; and rarely defecation, parturition, and hysterical convulsions. When arising in this way its favorite situation is the anterior margin of the upper lobe.

Pathology.—In the interlobular septa immediately beneath the pleura air-bubbles are sometimes seen to be arranged in well-defined rows. The pulmonary pleura may become detached, and the air-tumors may then become as large as an English walnut or even of greater size. Unlike the condition in vesicular emphysema, these sacs are freely movable, and the air may find its way from the root of the lung into the mediastinal connective tissue, and thence into the subcutaneous tissue of the neck and the wall of the thorax. Rarely these air-sacs perforate the pleura, setting up pneumothorax, with or without pleuritis.

Interlobular emphysema is sometimes associated with advanced vesicular emphysema.

VESICULAR EMPHYSEMA.

(*Alveolar Ectasis.*)

Definition.—Dilatation or enlargement of the alveoli and infundibular passages.

Varieties.—The cases are classified into—(1) Compensating, (2) Hypertrophic, and (3) Atrophic forms.

COMPENSATING EMPHYSEMA.

This variety is limited to certain parts of the lung, and arises in consequence of pathologic changes in other parts of the same organ that prevent full expansion of the lung on inspiration. Hence a vicarious increase in the volume of the air-cells is observed in circumscribed morbid processes such as occur in pulmonary tuberculosis, lobular pneumonia, cirrhosis, and pleurisy with adhesions (particularly when the latter is situated at the inferior border of the lung). An entire lung, unaffected by the primary disease, may be the seat of compensating emphysema when the causal disease invades the whole or a greater portion of the other lung, as in cirrhosis, extensive pleurisy with effusion, lobar pneumonia, and pyo-pneumothorax. When, however, the latter conditions are confined to a portion of one lung, the remainder of the same organ becomes distended also. The term *acute emphysema* is applicable to many of the cases.

As a rule, this pulmonary change is physiologic and beneficial: only rarely secondary atrophy of the walls of the air-cells develops.

Symptoms are not presented by the lungs in consequence of the changes met with in compensating emphysema. The condition is sometimes recognizable by means of the usual physical signs, but even these are not always to be relied upon. Fortunately, its existence may be safely inferred when there is conclusive evidence of the presence of the local causative diseases (broncho-pneumonia, pulmonary tuberculosis, pleurisy, lobar pneumonia).

HYPERTROPHIC EMPHYSEMA.

Nature of Emphysema.—The symptoms are dependent upon a loss of elasticity in the lungs, and, the latter condition being the result of overstretching, the contractile energy of the lungs is in great part destroyed; hence they become permanently enlarged. Nor do the emphysematous lungs contract when the thorax is opened, as they do ordinarily. We may in some cases account for the loss of elasticity in the lungs by the operation of causes that produce an abnormal degree of stretching, either temporarily or constantly; but under these circumstances emphysema would be developed despite the pre-existence of normal contractility of the lung. In true emphysema, however, which develops at a comparatively early period in life, we may safely assume that the retractile energy is defective (probably a congenital condition), and hence in such cases the action of the usual causal factors will speedily engender over-distention, or emphysema may develop even in the absence of causative influences. In these instances there is probably a quantitative as well as a qualitative defect in the elastic-tissue element of the lungs.

Pathology.—The thorax is enlarged (barrel-shaped), and upon removing the sternum the lungs are found completely to fill the mediastinum, and do not retract as in health. They present a pale, anemic appearance, although pigmented patches and streaks may be noted, while to the feel they appear soft and feathery, though dry. They readily pit on pressure (a leading characteristic).

Immediately beneath the pleura enlarged air-cells can be distinguished macroscopically, and air-sacs as large as a walnut or even larger may project above the lung-surface. Occasionally they may be so far detached as to be pedunculated. At the anterior borders a series of air-blebs, resembling a frog's lung, may be observed. Here, as well as near the root of the lung, distention is usually more marked than elsewhere, due to the direction taken by the distending force. The pleura is pale, and in patches the pigment may be absent (*Virchow's albinism*).

Upon microscopic examination it is observed that the dilatation starts in the infundibular and alveolar passages. The septa are partially obliterated, the alveolar walls thinned and, lastly, perforated, while in consequence of these changes the air-cells communicate with one another, and thus form larger or smaller air-sacs. The process is an atrophic one, in which the smaller elastic fibers at first disappear, while the larger become less prominent and often ruptured. After the latter changes have begun the capillaries likewise disappear, and the epithelium of the air-cells undergoes fatty degeneration, though in the larger bullæ a pavement layer is retained. The smooth muscular element may also occasionally be seen to be hypertrophied (Rindfleisch). The clinical phenomena probably arise from the loss of the capillary blood-vessel system and collateral hyperemia of the larger bronchial vessels.

The bronchial mucous membrane is usually the seat of chronic inflammation. It may be roughened and thickened, or the submucous elastic tissue may present prominent longitudinal lines, while the bronchial mucosa is covered with muco-pus. The smaller tubes may be dilated (*bronchiectasis*), and this condition may be associated with hyperplasia of the peribronchial connective tissue. The diaphragm is lowered and the subjacent viscera correspondingly depressed.

Physiologic Pathology.—The heart is pushed downward and somewhat backward, the right side showing well-marked changes; the cavities are dilated and hypertrophied, due to obstruction in the pulmonary circulation; and in long-standing cases hypertrophy of the left chambers may also develop. The pulmonary artery and its branches are enlarged and the seat of atheromatous degeneration. The liver, kidneys, and other viscera present the changes that belong to long-continued venous engorgement.

Etiology.—The affection is often secondary to, and develops in consequence of, other affections of the lung—notably, *chronic bronchitis* and *whooping-cough*. The dry form of chronic bronchitis, in particular, is apt to generate pulmonary emphysema. Under these circumstances the disease is directly attributable to the mechanical influences to which the alveolar walls are subjected during respiration. This abnormal strain attends inspiration to some extent, but mainly expiration, owing to the obstruction to the egress of the air in the smaller bronchi, with increased *intra-alveolar air-pressure*. The increased tension in the air-cells may be accounted for, partly, by the severe and persistent cough, the air being forced during violent coughing into the apices of the lungs, forcibly expanding them and causing emphysema.

Bronchial asthma, on account of the obstruction of the exit of the air from the lungs, produces during the attacks an acute emphysema that may result finally in a condition of permanent over-distention. *Certain occupations*, such as blowing wind-instruments, or those that entail severe muscular strain (*e. g.* blacksmithing), act as predisposing causes, and hence, emphysema is of common occurrence among the working classes, and is more common in males than females. The constant straining in certain pelvic disorders may induce emphysema. The disease is often *hereditary*. During *advanced years* the lung-elasticity often diminishes, and as a consequence a disposition to emphysema is engendered. On the other hand, emphysema is not infrequently met with in children, and in such there may be a temporary respite, with a recurrence at a later period. An emphysematous tendency also results from congestion of the lungs associated with mitral valvular disease.

Clinical History.—In nearly all cases the disease develops insidiously, the symptoms being gradually added to those of the primary affections (chronic bronchitis, asthma, etc.). When due to the occupation of the patient its development is also slow, and not infrequently its origin dates back to childhood or beyond the recollection of the patient. Rarely it may exhibit a more acute development, as, for example, after whooping-cough.

The first symptom is a variable degree of *dyspnea*, and to this may be added temporary *cyanosis* and *cough*. The severity of the dyspnea varies with the degree of distention of the air-cells, even though additionally aggravated by the coexistence of the primary disease. In moderate emphysema the dyspnea is only apparent on going up stairs, running, walking rapidly, or after a hearty meal; on the other hand, in advanced grades of the affection it is constant, and is intensified by the slightest exertion, even to orthopnea. Speech is interfered with, the patient's utterances taking the form of fragmentary sentences or syllables. The labored breathing is shown particularly in expiration, and, as in asthma, in which the alveolar spaces are acutely distended, so in emphysema the rhythm of the respiration is changed. The inspiration is shortened, and the expiration is greatly prolonged and accompanied by wheezing when chronic bronchitis coexists.

In the later stages cyanosis becomes more marked, and is noticeable in proportion to the loss of compensation and interference with the cardio-pulmonary circulation. It often attains to an extreme degree, and the patient's alarming appearance may be in striking contrast with his apparent degree of comfort. In mild forms the cyanotic tint is confined to the lips, lobes of the ears, and the extremities. Any increase in the degree of dyspnea after exertion results in an increased blueness of the surface.

The *cough* is dependent upon the presence of chronic bronchitis, and the latter disease is frequently found in combination, particularly during the winter. There is also an expectoration that is identical with that of chronic bronchitis, and when this disease reaches an advanced stage the cough persists throughout the year (*vide* Chronic Bronchitis). Intercurrent acute attacks of bronchitis are often followed by temporary attacks of asthma; and since chronic bronchitis in its highest grades is met with at an advanced period of life, so, as would be expected, the cases of advanced emphysema are also met with at the same period. Osler has described a group of cases occurring in patients "from twenty-five to forty years of age who, winter after winter, have had attacks of intense cyanosis in consequence of an aggravated bronchial catarrh."

These patients are short-breathed from infancy, and their condition is attributed to a primary defect of structure in the lung-tissue.

General Symptoms.—There is an absence of febrile movement; the pulse is not increased in frequency, though sometimes feeble; and the temperature of the body is generally subnormal. There is a very gradual loss of flesh and strength, and the patient is stoop-shouldered, presenting a peculiar cachectic appearance—a condition that is in strong contrast with the dusky appearance of the face, the swollen neck, and the enlarged chest.

Finally, other symptoms may be mentioned that are for the most part secondary to hypertrophy, followed by dilatation, of the right ventricle. This hypertrophy is the result of pulmonary congestion and obliteration of the pulmonary capillaries induced by the emphysema. Under these circumstances severe attacks of cough occur, attended with extreme dyspnea and lividity, and later the conditions that usually succeed a moderate grade of tricuspid insufficiency supervene, such as congestion of various viscera and edema of the feet. Anasarca is rare.

Physical Signs.—The shape of the chest is characteristic: owing to the increased antero-posterior diameter, it becomes barrel-shaped (Fig. 44), and the sternum bulges, as do also the costal cartilages. The infraclavicular and mammary regions are also prominent, and give the thorax an abnormally rounded appearance. The episternal notch is deeper than the normal, the clavicles and muscles of the neck are unduly prominent, and the neck itself appears to be shortened, owing to the elevated position of the clavicles and the sternum. There is an antero-posterior curvature of the spine and a winged condition of the scapulæ—changes to which the stooping posture is ascribable. Below, the thorax appears contracted. The intercostal spaces are widened and depressed, and a network of dilated venules frequently extends laterally above the inferior costal border, but is by no means characteristic of the affection.

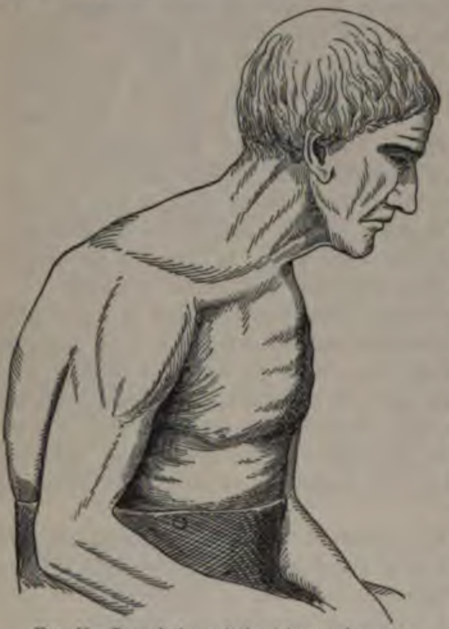

FIG. 44.—Barrel-shaped chest in emphysema.

The movements of the chest are vertical rather than expansile, and the lungs are constantly in a state of extreme expansion; in the lower thoracic and upper abdominal regions there may be observed retraction rather than expansion during the act of inspiration. The respiratory acts, as a whole, are labored, and the diaphragm and abdominal muscles are seen working with considerable violence. The heart's apex-beat is invisible, but marked epigastric pulsation is frequently noticeable. A transverse linear depression across the abdomen, on a level with the lower ribs, may also be present during inspiration. Venous pulsation may be seen in the neck after failure of the right ventricle has occurred.

On *palpation* the character and direction of the chest-movements may be accurately appreciated. The tactile fremitus is decreased, but not absent. In advanced cases the apex-beat cannot be felt, and even in the earlier stages it becomes more and more enfeebled. Owing to displacement of the heart and engorgement of the right ventricle there is a distinct systolic shock over the ensiform cartilage, and also a pulsation in the epigastrium.

Percussion yields a characteristic hyper-resonance. This may be distinctly "Skodaic" or semi-tympanitic, and in extreme dilatation of the air-cells the tone may be woodeny. The area of cardiac dulness, owing to the fact that the lungs overlap the heart, becomes lessened and finally obliterated; while the upper limit of liver-dulness, both anteriorly and posteriorly, is found to be one or two interspaces lower than normal, owing to the fact that the diaphragm is depressed. The upper level of splenic dulness is also lowered, and the area of percussion-hyper-resonance extends higher above the clavicles than naturally.

On *auscultation* the inspiration is short and feeble, while the expiration is greatly lengthened, the ratio of these sounds as to duration being reversed as compared with the normal. Their pitch is somewhat lowered, particularly that of expiration; and when râles are present the respiratory murmur (particularly the inspiratory) may be scarcely audible. In well-marked instances of emphysema inspiration and expiration may rarely be of equal length. It is a fact worthy of emphasis that the parts of the lungs that are not so markedly emphysematous as others give a harsh, exaggerated vesicular murmur, owing to the great efforts of breathing. Râles of various sorts are frequently audible, due to bronchitis, which, it must be recollected, accompanies emphysema in a majority of instances; less frequently the auscultatory signs of asthma, pleuritis, and phthisis are encountered. Rarely, rubbing sounds, that have been attributed to the friction of enlarged air-cells against the pleura, are audible, and when the interlobular variety supervenes upon vesicular emphysema a *crumpling* sound is heard. The so-called "*Laennec's râle*," which resembles somewhat the subcrepitant râle, is not infrequently present. The vocal resonance varies from an almost total absence to a greatly increased intensity. The tricuspid insufficiency that develops late in this affection is betrayed by its characteristic murmur.

Diagnosis.—A positive diagnosis may be arrived at from a consideration of the history, including such points as heredity, occupation, the long duration of the condition, coupled with the most characteristic symptoms (dyspnea, cyanosis, signs of chronic bronchitis), and from the physical signs. In a case of beginning emphysema, particularly among children, a certain diagnosis is not to be attempted.

Differential Diagnosis.—*Pneumothorax* is the disease most apt to be confounded with emphysema. It develops suddenly, however, while emphysema is of slow development, and the rational symptoms of pneumothorax are more constant and urgently distressing than those of emphysema. Pneumothorax is unilateral, and gives a purely tympanitic percussion-note, while hypertrophic emphysema is bilateral and its percussion-note is hyper-resonant. Auscultation in pneumothorax usually gives amphoric breathing, metallic tinkling, the characteristic succussion splash, and an absence of the vesicular murmur; all of which auscultatory signs are very unlike those of emphysema.

Another affection giving rise to dyspnea, cough, and cyanosis is *pleurisy with effusion*, but the slow course, the absence of fever, and the universal hyper-resonance that characterize emphysema do not belong to pleurisy. The latter affection is usually unilateral, and over its seat a flat percussion-note is obtained.

Prognosis.—Hypertrophic emphysema of acute form (*e. g.* resulting from whooping-cough) is often curable; but the usual slowly-generated variety, so far as recovery is concerned, gives a totally unfavorable prognosis. In many cases, however, life is not materially shortened. Temporary improvement is possible when the lesion consists merely of a distention of the air-cells, and this is shown by a corresponding improvement in the physical signs. The effect of frequently recurring attacks of bronchitis is only to intensify the symptoms of a disease that is innately progressive. Intercurrent affections, however, such as pneumonia (lobar and lobular) and pulmonary tuberculosis, may prove fatal. Dropsy, following broken compensation, is often a late and dangerous complication; other late accidents of the disease are hemoptysis and sudden dilatation of the right heart.

Individual circumstances, such as the patient's social condition, the stage of the affection in which he comes under proper treatment, and the degree of care he is willing to exercise, greatly influence the outcome of the case.

Treatment.—The treatment is to be directed toward the removal of the causes of emphysema, and chiefly of the chronic bronchitis. From personal observation I am firmly convinced of the fact that the progress of the disease can be arrested, and that the condition is sometimes improved, by relieving the chronic bronchitis. The iodids (potassium, sodium, and ammonium) will sometimes produce effects that are truly remarkable, and the syrup of hydriodic acid may be employed when the iodids are not well borne by the stomach. If the occupation of the patient tends to aggravate the disease, it must be forsaken for one that is less harmful. Violent paroxysms of cough also contribute to the production of alveolar distention, and hence must be alleviated promptly. Intercurrent attacks of asthma have a similar effect, and must be relieved as speedily as possible by a resort to appropriate therapeutic measures. Attacks of acute bronchitis are to be prevented, if possible, by suitable clothing, by avoidance of exposure to inclement weather, dust, and the vitiated atmosphere of overcrowded halls, churches, and the like; whenever practicable the result can be most successfully obtained by a residence in an equable climate. Since a severe bronchitis is apt to increase the severity of the emphysematous symptoms, it must be relieved as speedily as possible.

As soon as passive congestion, flatulence, and constipation, with other gastro-intestinal symptoms, appear, the diet will demand careful regulation, and especially a restriction in the use of carbohydrates.

The bowels must also be moved regularly with a view to obviating the flatulence and portal engorgement.

The heart needs to be carefully watched, and as soon as signs of broken compensation appear digitalis and strychnin will be found highly useful. Diuretics and cathartics may also become necessary. The sudden development of urgent dyspnea (or orthopnea) and extreme lividity, especially if associated with weak cardiac action and a rapid, feeble, irregular pulse, calls for free bleedings, and more than once in the course of my hospital practice have I seen the lives of patients suffering from emphysema saved by timely venesection.

Besides meeting the pathologic and symptomatic indications, we should aim to assist the patient in expiration, and Gerhardt has suggested systematic *mechanical compression* of the thorax during expiration as a useful measure. This external pressure must be made by an attendant, who places his hands flat on the lower lateral portions of the thorax, and the manipulation is to be continued for from ten to fifteen minutes daily. The results obtained by certain German authors have been encouraging, but in my own hands the method has failed, except in two instances occurring in young adults with yielding chest-walls, in whom it was of the greatest service. The *pneumatic treatment,* comprising the inhalation of compressed air and the breathing into rarefied air, richly deserves further trial,[1] its use having been productive of permanent improvement in a number of cases, as shown by physical examination (including mensuration).

SENILE EMPHYSEMA.

This variety is in reality a senile atrophy of the lungs, and has been appropriately termed "small-lunged emphysema" by Sir Wm. Jenner. In consequence of the complete atrophy of the alveolar walls, coalition of the air-cells takes place, with the production of large air-sacs. The lungs contain less than the normal volume of air, instead of an abnormal quantity as in true hypertrophic emphysema, and as a result occupy less space in the chest-cavity than do healthy lungs. The pulmonary tissue elements are deeply pigmented. The condition does not produce right ventricular hypertrophy.

The **symptoms** are negative, although subjects in whom senile emphysema develops may have previously had chronic bronchitis with more or less dyspnea. They quite frequently present a withered appearance, and the chest on inspection is seen to be contracted, owing to the fact that the ribs approximate more closely and take a more oblique direction than in health.

Treatment is unavailing.

GANGRENE OF THE LUNGS.

Pathology.—The affection presents itself in two forms—as a (*a*) diffuse, and a (*b*) circumscribed process.

(*a*) The **diffuse** variety is rare. It may, however, be met with in lobar pneumonia, and very rarely in consequence of occlusion of the large branch of the pulmonary artery; it may also be secondary to the circumscribed form. The greater part of the lobe, or even an entire lung, may be involved, the pulmonary parenchyma degenerating into a putrid, greenish-black, pulpy mass, with no obvious line of demarcation.

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[1] Waldenburg's portable apparatus is convenient for use.

(*b*) The **circumscribed** form may involve either one or both lungs, though the right is affected somewhat oftener than the left. To this category belongs the so-called *embolic gangrene*, the nodules of which have their favorite seat in close proximity to the pulmonary pleura. All etiologic varieties of the circumscribed form more frequently implicate the lower than the upper lobe of the lung, occurring in sharply defined areas, which may either be single or multiple. The affected area first presents a greenish-brown appearance; its central portion soon undergoes softening, and a cavity is thus formed whose walls are ragged and irregular and contain a foul-smelling, dark, greenish liquid. The surrounding lung is inflamed, and the air-sacs contain inflammatory products (fibrin, epithelium, pus), while the highly-irritating and putrid material sets up an intense bronchitis. These gangrenous foci may increase in size by a peripheral extension, and thus the adjacent veins may become plugged with infectious thrombi or the vessels may become eroded. Emboli may then be detached from the infectious thrombi, and, entering the circulation, may set up foci of septic inflammation in remote organs. A truly remarkable connection exists between circumscribed gangrene of the lung and cerebral abscess. When the gangrenous spot is situated near the pleura, simple or gangrenous pleurisy may arise as a complication, or the pulmonary pleura may be perforated and pyo-pneumothorax result. When recovery ensues the cavities formed as the result of the conversion of lung-tissue present a limiting wall of dense connective tissue. Such cavities may remain permanently or may slowly become contracted.

Etiology.—Gangrene of the lungs is caused by the bacteria of putrefaction (probably the staphylococcus albus or aureus). The disease is rare, even though the opportunity for inhaling the bacteria that cause it is a constant one. It is only when the lung-tissue has become impaired or peculiarly altered that the specific bacteria are capable of producing gangrene. It may occur in several ways:

(1) Secondary to lobar pneumonia, hemorrhagic infarctions, cavities in the lungs, bronchiectasis, wounds of the lung, contusions of the thorax, carcinoma of the esophagus, or to compression or embolism of the pulmonary artery or of the bronchial vessels.

(2) As an embolus, derived from a gangrenous area in some other organ of the body, it may lodge in the lung and set up putrefactive changes.

(3) Pressure from a thoracic aneurysm may give rise to gangrene.

(4) The most important causal factor, however, is the entrance of foreign bodies, especially bits of food, into the bronchi and lungs. Whether or not the specific bacteria of putrefaction enter the lungs with the foreign bodies, the latter render the tissue-soil receptive to the former, and once the process has been initiated it is apt to extend itself. There are several ways in which these foreign particles gain entrance into the bronchi and lungs: (*a*) By a faulty swallowing of the food; (*b*)

By inhalation; (*c*) By a carcinomatous perforation of the esophagus into the bronchus or into the lung.

(5) In the course of debilitated states of the system, as during convalescence from protracted fever (rarely), and in diabetes mellitus (frequently).

Symptoms.—These are *local* and *general*, the former alone being diagnostic.

Local Symptoms.—There is *cough* accompanied by an exceedingly *fetid expectoration* that is usually quite profuse. When abundant, and when expectorated into a conical glass and allowed to stand for a time, it separates into three layers: (*a*) the uppermost, being frothy, opaque, and of a grayish-yellow color; (*b*) the middle, clear and watery; and (*c*) the lowest, appearing as a greenish-brown sedimentary layer containing shreds of lung-tissue and sometimes blood. The microscope shows it to consist of numerous elastic fibers, bacteria, fat-crystals, muco-pus, granular matter, and leptothrices. Small quantities of blood in the sputum are very common. Kannenburg and Streng have also described ciliated monads as occurring in the sputum. The patient's breath is, as a rule, intensely fetid, even though there be no expectoration, but this fetor of breath may be absent, as in a case of my own (which came to autopsy), in which the localized gangrenous process had no fistulous connection with the bronchus. It is to be recollected that if any of the large branches of the pulmonary artery be eroded, free and even fatal hemoptysis will result.

Physical Signs.—The physical signs are sometimes obscure, as when the areas involved are smaller and deeply situated, and in such instances signs of bronchitis only may be detectable. When large and favorably situated, however, the affected spots usually give signs of consolidation, rapidly followed by those of cavity. In addition bronchial râles—usually moist—and coarse cavernous râles are usually audible. It is obvious that when the pleura is implicated the signs of pleurisy are added, and if pneumothorax be present those belonging to the latter condition also.

The chief **general symptoms** are irregular fever, emaciation, and profound prostration. A septic condition of the system is commonly developed, and the patient sinks from exhaustion. Rarely there may be an almost total absence of constitutional disturbances, and such instances terminate in recovery.

Diagnosis.—The distinctive feature is the intense fetor both of the sputum and the breath. The physical signs may readily determine the existence of the pulmonary lesion, but it is difficult to eliminate *abscess* and *fetid bronchitis* associated with bronchiectasis. The results of a careful examination of the sputum, together with the less horribly fetid odor of the breath, in *abscess* will usually suffice to eliminate the latter affection. In *fetid bronchitis* the fetor of the breath and sputum is also less marked than in gangrene, while its course is slower and more favorable than that of the latter affection.

Prognosis.—The prognosis is always grave, though rarely recovery in circumscribed gangrene of the lungs ensues. The chief dangers are exhaustion and hemorrhage. Improved methods of surgical treatment, however, have saved life in a few instances, and promise to reduce still further the mortality-rate of this serious affection.

Treatment.—The leading indications are—

(*a*) The disinfection of the gangrenous focus or foci in the lungs. This may be accomplished by the internal administration of creasote or carbolic acid or by the use of an antiseptic spray. In a recent case the employment of Robinson's inhaler, charged with equal parts of creasote, alcohol, and chloroform, gave encouraging results.

(*b*) The patient's nutrition must be maintained, if possible, by a concentrated liquid diet, administered in fixed quantities and at regular intervals; also by the judicious cultivation of the digestive functions, together with the use of stimulants and tonics. For a description of the surgical treatment of gangrenous cavities of the lungs the reader is referred to special works on surgery. It is the physician's duty, however, to determine whether or not the patient's general condition will admit of surgical intervention, and also to localize as nearly as may be the affected zones for the surgeon's guidance.

ABSCESS OF THE LUNGS.

(*Suppurative Pneumonitis.*)

Pathology.—This affection is characterized by the formation of pus and the degeneration of lung-tissue. It may be (*a*) a mere infiltration of the blood-vessels, bronchi, or interstitial tissue, but more frequently purulent inflammation of the lungs takes the form of (*b*) an ordinary abscess. In size the abscesses range from that of a walnut to an apple, and I have observed in one case inflammation of the whole of the middle lobe of the right lung. The abscess-walls are irregular and decidedly ragged; and in the case of old lesions there is a dense fibrous wall; the contents are purulent and rarely necrotic. If the contour of an abscess touches the pleura, empyema is the usual result, though serofibrinous pleurisy may rarely follow. Rupture of the abscess into the pleura may also occur.

Etiology.—Streptococci are found, though they are not the only direct causes of abscess of the lung. The diplococcus pneumoniæ and Friedländer's bacillus have been found, as well as certain other pyogenic organisms. *Predisposition* is noted in certain conditions, as (1) during or following the occurrence of inflammation, as in lobar and lobular pneumonia. Suppurative infiltration, however, more frequently arises under these circumstances than abscess, and in the rare instances in which the latter occurs it is apt to be comparatively small and multiple. In all forms of inhalation and deglutition broncho-pneumonia, however, abscess of the lung is a frequent sequela.

(2) Perforation of the lung from without or from adjacent organs, as in carcinoma of the esophagus, abscess of the liver, or suppurating hydatid cyst.

(3) Infectious emboli, found in connection with septico-pyemia, frequently cause metastatic abscesses in the lungs. In a mechanical manner they may produce hemorrhagic infarctions, followed by suppuration, or the latter process may occur independently of the former. The abscesses

are usually situated close to the pleura, and are frequently wedge-shaped; they vary in number from one to several hundred, and in size from a pin's head to an orange.

(4) Abscess of the lung may result from inward extension of a purulent pleurisy; and, oppositely, purulent pleurisy may result from an extension of abscess of the lung.

(5) As elsewhere stated (*vide* Pulmonary Tuberculosis), suppuration is quite generally associated with chronic pulmonary tuberculosis.

Symptoms and Diagnosis.—The examination of the *sputum* is of the greatest value in the diagnosis of this disease, since, being purulent, it usually presents a yellow, or less frequently a greenish- or brownish-yellow, color. It emits a fetor that is less pronounced than that of either gangrene or putrid bronchitis. Particles of lung-tissue may be visible in the pus, and on microscopic examination of the latter, elastic fibers, the presence of which is of the utmost importance in the diagnosis, may be found in profusion. Next to the investigation of the sputum, the *physical signs* of cavity are of the greatest assistance in distinguishing abscess of the lung; these, however, are wanting unless the abscess is of a considerable size. By themselves, the signs of cavity do not suffice for the recognition of abscess, but when combined with the characteristic sputum leave no room for doubt. Chills and suppurative fever often attend. The history is of considerable importance, as confirming the more characteristic features. Thus antecedent pneumonia or septico-pyemia would be strongly corroborative.

Prognosis.—The prognosis is often hopeless, as, for example, when the disease is associated with pyemic processes in other parts of the body. On the other hand, those rare instances in which it is secondary to pneumonia give a comparatively favorable prognosis.

Treatment.—The chief aim in the therapeusis should be to support the system by the administration of tonics, stimulants, and antiseptics, as well as by methodic feeding with light and concentrated forms of nourishment. Inhalation of antiseptic sprays (creasote, thymol) should be tried. When the abscess is situated near the periphery of the lung, surgical interference is to be advised as soon as the first indications of increasing weakness appear. For the details of the operation of pneumonotomy for pulmonary abscess the reader is referred to special works on surgery. The statistics of Eichhorst,[1] showing its favorable results, may, however, be mentioned, as follows: in 13 operations recovery or improvement was noted in 6, while fatal terminations occurred in 7.

PNEUMONOKONIOSIS.

(*Anthracosis*, *Chalicosis*, *etc.*)

Definition.—A form of chronic interstitial pneumonia that arises from the inhalation of dust-like particles. Different terms have been applied to the condition according to the nature of the dusts inhaled, the chief among these being—(1) Anthracosis (coal-miners' disease), due to the inhalation of coal-dust; (2) Chalicosis (stone-cutters' phthisis), caused by the inhalation of mineral dusts; and (3) Siderosis, caused by inhaling metallic particles, particularly iron oxid.

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[1] *Specielle pathologie*, Bd. 1, S. 519.

(1) **Anthracosis.**—Among dwellers in cities a moderate degree of pigmentation of the lung-tissue with coal-dust is the rule, while in those residing in rural districts the condition is decidedly less common. True anthracosis, however, has reference to such an accumulation of the carbon particles as can be due only to the inhalation of a well-laden atmosphere, or under circumstances when the mucous membrane is unhealthy or without perfect ciliary action. Under such circumstances the normal scavengers of the respiratory organs—the mucous corpuscles lining the trachea, the bronchi, and the alveolar cells—fail to deal successfully with the numerous dust-particles that gain entrance along with the inspired air; hence some of the latter pierce the mucosa and reach the lymph-spaces and lymph-vessels. On reaching the bronchial mucosa they become enclosed in leukocytes, mucous corpuscles, and alveolar cells, and are conveyed by the latter to a more remote destination. Arnold shows that after the particles enter the lymph-system they are carried "(*a*) to the lymph-nodules surrounding the bronchi and blood-vessels; (*b*) to the interlobular septa beneath the pleura, where they lodge in and between the tissue-element; and (*c*) along the larger lymph-channels to the substernal, bronchial, and tracheal glands, in which the stroma-cells in the follicular cord dispose of them permanently," with resulting indurative enlargement of these structures. Rarely the carbon particles may find their way into the general circulation; this may occur, as shown by Weigert, when the pigmented bronchial glands become adherent to the pulmonary veins, thus giving opportunity for the escape of the carbon granules into the blood.

Anthracosis leads, primarily, to chronic bronchitis, to be soon followed by emphysema; but it must be recollected that extensive anthracosis may be present without any other changes in the lung than the presence of carbon particles stored in the protoplasmic cells. The lung-tissue presents great variations in its degree of susceptibility to these foreign particles. Sooner or later, there is usually produced, as the result of their irritant action,[1] a proliferation of the connective-tissue elements—*i. e.* a chronic interstitial inflammation. This fibroid change usually starts in the peribronchial lymph-structures, though the bronchial and tracheal glands are, as a rule, similarly involved at a comparatively early period. The affected lung-tissue is frequently coal-black, dense, and airless. The pneumonokoniotic areas vary greatly in size and numbers, and not infrequently they coalesce, in which case large portions of the lung-tissue may become the seat of fibroid change. The alveolar walls are observed to be much thickened in some instances, and firm pleuritic adhesions exist. Bronchiectatic cavities may be present, and later necrotic softening of the indurated areas occurs, leading to the formation of small cavities that contain a dark fluid. When the latter communicate with the bronchi their walls are prone to ulcerate. I have noticed that the process almost invariably terminates in pulmonary tuberculosis, and particularly is this true of cases that follow the inhalation of mineral and vegetable dusts (*vide infra*).

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[1] Cohnheim contends that coal particles do not produce irritative changes in the lung, and that the latter are due to irritating substances inhaled with the particles of coal.

(2) **Chalicosis.**—Changes similar to those previously described are induced in the pulmonary connective tissue by the inhalation of stone-dust by those who follow certain occupations, such as stone-cutting, knife- and axe-grinding, and millstone-making. The irritating properties of this form of dust cannot be denied, as shown by the great disposition in this subvariety of pneumonokoniosis to the formation of fibrous nodules and diffuse areas of sclerosis in the lungs. The nodules have a gray center and a darker periphery; they are exceedingly dense, and sections are made with much difficulty. The cut surface may present a grayish and distinctly glistening appearance.

(3) **Siderosis.**—This term implies a collection of iron oxid in the lungs, also due to the pursuit of certain occupations (dyeing, iron-smithing, etc.). Cases of much the same nature are caused by the inhalation of vegetable dusts by grain-shovellers, cotton-spinners, cigar-makers, etc. The *pathologic* changes are identical with those in anthracosis, though the color-appearance is red instead of black.

Symptoms.—Rarely the *onset* is marked by the symptoms of acute, followed by those of chronic, bronchitis; but in a vast majority of instances chronic bronchitis gradually develops after long exposure to the action of the exciting cause. The symptoms of emphysema are soon superadded, the patient now suffering from dyspnea, and less frequently from asthma. The *sputum* is diagnostic in anthracosis, being quite dark; in chalicosis a microscopic examination is essential to show the particles of silica; while in siderosis the expectoration presents a reddish color. Apart from the foreign particles, the sputum is for a long period of years muco-purulent in character, and later it often contains the tubercle bacillus.

The *physical signs* are not distinctive, being identical with those met with in chronic bronchitis associated with emphysema, and followed by those of interstitial pneumonia, and sometimes by those of cavity.

The **diagnosis** is to be made both from the history and from a gross or microscopic examination of the sputum. It may be confirmed by the invariable presence of the signs of bronchitis and emphysema, as well as by the effect of removal to an atmosphere free from dust. In the later stages the detection of infallible evidences of phthisis only serves to corroborate the earlier diagnosis of pneumonokoniosis.

The **prognosis** is favorable in hygienic surroundings until the more advanced stage is reached. The condition favors the invasion of new growths (lympho-sarcoma, or cobalt-miners' disease; *vide infra*).

Treatment.—A change of occupation or several hours of exercise in the open air daily for those who are exposed to dust in work-rooms should be advocated. Dusty work-rooms must be properly ventilated.

The active treatment is the same as for chronic bronchitis and emphysema from other causes, and is to be appropriately modified when pulmonary tuberculosis develops.

NEW GROWTHS OF THE LUNGS.

CARCINOMA OF THE LUNG.

ALL varieties of carcinoma have been met with in the lung, but, with rare exceptions, carcinoma of this organ is secondary to similar growths in other parts of the body. To explain its origin it may safely be assumed that the primary new growth involves a vein or lymph-channel, and that the latter carries the germ of the disease to the lung. It is also to be recollected that it may result from extension, or by contiguity from neighboring organs (as the esophagus, mamma, pleura, or mediastinum).

Etiology.—The causes of primary carcinoma of the lung must be, in the main, identical with those of carcinoma in general, and are as yet unknown. Most cases occur in middle-aged persons, and, while sex has no influence upon the appearance of the primary form of the disease, the secondary form is more frequent in the female than in the male. In the female secondary carcinoma of the lung is often preceded by carcinoma of the breast. We may also regard hereditary influence as a potent predisposing factor. Secondary carcinoma of the lung is most commonly consecutive to primary carcinoma of the bones, and of the digestive and urinary tracts.

Pathology.—The pathologic varieties of the primary form are scirrhous, encephaloid, and epithelioma, and of these the latter is the most common. *Primary carcinoma* is usually unilateral, the tumors attaining to a massive size and frequently involving the greater part of one lung. Their favorite seat is in the upper part of the right lung, though the pleura is quite often invaded by the carcinomatous process. Less frequently there is pleurisy with sero-fibrinous exudate, which may be hemorrhagic. Carcinomatous involvement of the cervical, bronchial, and tracheal lymph-glands is quite usual, and rarely even the inguinal glands become implicated. *Secondary carcinomata* are, as a rule, multiple, and may be miliary in size. They are disseminated widely throughout both lungs, though in the rarest instances they may be unilateral. In the softer varieties the central portion of the tumor-mass may undergo fatty degeneration, with subsequent discharge through adjacent bronchi.

Symptoms.—The symptoms of carcinoma of the lung vary according to the location and extent of the disease. Among the most marked symptoms belongs *pain*, particularly when the pleura is implicated. As a rule, for a considerable period of time the symptoms of *bronchitis* obtain, and later the breathing-space is diminished sufficiently to excite dyspnea and cyanosis. With the increase in size of the new growth compression of the heart, aorta, and large veins may result, whereupon *disturbances of the circulation* will arise. The new growth may exert pressure on the esophagus, causing *dysphagia;* or upon the recurrent laryngeal nerve, causing *aphonia* and *hoarseness;* or on the trachea or a main bronchus, followed by the symptoms of *stenosis* of those organs. There are *cough* and *expectoration*, the latter frequently containing blood-corpuscles with mucus, and resembling in appearance currant-jelly; the sputa may also rarely exhibit a grass-green color, due to transformation of the blood-pigment. In carcinomatous lungs putrefactive changes sometimes take place, and if so the expectoration and breath emit an offensive odor, while a microscopic examination of the sputum frequently discloses the presence of carcinomatous elements. The well-known cancerous cachexia invariably develops.

Physical Signs.—These will naturally depend upon the extent and location of the new growth. *Inspection.*—If the lung-tissue be extensively involved, the walls of the thorax become unduly prominent and fixed over the seat of the tumor. Indeed, the tumor may, though rarely, protrude between the ribs. The intercostal spaces are widened, and the superficial veins, in view of the fact that they cannot empty themselves into the internal veins, appear engorged; from the same cause edema affecting the thorax, neck, face, and arms may be noted. Swelling of the lymph-glands in the neck or axilla is a symptom of importance. On *palpation* the tactile fremitus may be found to be diminished or absent. The *percussion-note* will be flat, since the air-vesicles and smaller bronchi are replaced by the solid growth. On *auscultation* friction-sounds are the rule. The respiratory sounds may be greatly enfeebled or absent; but if the carcinomatous tumor communicates with a wide-mouthed bronchus, bronchial breathing may be audible, and the physical signs of lung-cavity may be developed. The signs of general bronchitis are present in most instances, especially in the disseminated form of the disease; in the latter variety the lung may shrink, with retraction of the chest-walls on the affected side. If secondary pleurisy with effusion occurs the detection of the characteristic cancer-cells in the contents of the pleural cavity will show the precise nature of the thoracic affection.

Diagnosis.—The following symptom-group will pretty well establish a diagnosis: A peculiarly shaped dull area (as when it extends under the sternum), perhaps a marked prominence over the site of the tumor, enlarged and hard lymphatic glands in the vicinage, and more or less of the compression-symptoms—circulatory, nervous, bronchial, or trachial stenosis. In rarer instances the diagnosis may be made by the occurrence of metastasis to the chest-wall. Again, the discovery of cancer-tissue in masses accidentally detached gives reliable indication of the disease. An exact diagnosis can often be made from the examination of the particles obtained on aspiration of the tumor itself.

The **differential diagnosis** between pulmonary carcinoma and *pulmonary tuberculosis* can be made with positiveness only by a careful microscopic examination of the sputum. From *fibroid induration* of the lung it is easily discriminated, owing to the history and slower course of the latter affection.

Prognosis.—This is bad, as death may occur suddenly from abundant hemorrhage or more frequently from either exhaustion or asphyxia. The duration of the affection varies from six months to a year, or, rarely, even two years.

Treatment.—The treatment must be addressed solely to the relief of pain and other subjective symptoms.

SARCOMA OF THE LUNG.

Primary sarcoma of the lung is rare, but in instances of generalized sarcomatosis the lungs show larger or smaller nodules "in almost every case" (Birch-Hirschfeld), occurring in connection with osteo-sarcoma of other organs or in lympho-sarcoma of the cervical glands.

Secondary sarcoma, occurring in consequence of invasion of the root of the lung by sarcomatous disease of the post-bronchial glands, is more common than secondary carcinoma. The ***diagnosis*** is reached as in carcinoma (*vide* p. 543).

Neoplasms occurring among the cobalt-miners of Schneeberg were described by Hesse and Tragner as lympho-sarcomata—slowly growing masses that attained to a large size and gave metastasis to lymph-glands, pleura, liver, and spleen. In most cases there was an associated pneumonokoniosis, which had probably predisposed to the new growth.

HYDATID CYST OF THE LUNG.

Hydatids in the lungs may either be primary or secondary, the former variety being exceedingly rare and the latter somewhat less so. Almost invariably the echinococci are developed in other organs—the liver in particular—and find their way to the lungs, either by direct perforation through the diaphragm or by entering through the blood-current. The lungs are involved in about 12 per cent. of hydatid disease.

For **etiology** and **pathology** see Hydatid Cysts of the Liver.

Symptoms.—The clinical manifestations are quite varied, even though the cyst may entirely conceal itself. It is important to recollect that similar involvement of the liver usually coexists; and in addition to the symptoms of the latter affection there may be *pain* in the chest, *dyspnea*, considerable *cough*, and, rarely, blood-stained *expectoration*.

The **physical signs**, when present, are as follows: Diminished vocal fremitus, defective expansion, dulness on percussion with an absence of the respiratory murmur—all signs pointing to pleural effusion. The cysts are more common in the right lung and frequently cause marked bulging over the base. Later signs of cavity-formation may appear. In other cases, the signs of consolidation may preponderate.

A positive **diagnosis** of hydatid cyst of the lung can be made only when the scolices, pieces of membrane, or the hooklets of the echinococcus are demonstrable in the sputum. Besides being evacuated into the bronchi, the cysts may rupture into the adjacent serous sacs (pleura, peritoneum, percardium), or externally, the latter being the most favorable mode of termination. Unless they are discharged early by ulceration into the bronchi or externally, they are apt to excite inflammation of the adjacent lung-tissue and tubes, accompanied by an active febrile movement and an aggravation of the symptoms before detailed: these complications (pneumonia, gangrene) may assume a dangerous form, or the patient may, if the growth attains large dimensions, become asphyxiated. From *gangrene*, *pleurisy*, and *phthisis* echinococci are distinguished by the sputum-test or by an examination of the aspirated fluid.

Prognosis.—The affection is always attended with great danger, and is of more serious import when secondary to involvement of the liver than when primary.

Treatment.—When it can be shown that the growths are situated at the periphery of the lung operation should be carefully considered. The physician stands powerless to do more than to relieve urgent symptoms in special cases and to support the vital functions.

V. DISEASES OF THE PLEURA.

PLEURISY.

(*Pleuritis.*)

Definition.—An inflammation, either local or general, of one or both pleural membranes. The disease, as shown by postmortem examinations, is of great frequency.

Varieties.—Pleurisy has been variously classified. *Etiologically*, the distinction between primary and secondary forms of the disease should be made, as well as a division into tuberculous, carcinomatous, septic, etc. *Pathologically*, all cases may be summarized under the following heads: Localized and generalized and dry (plastic) pleurisy and pleurisy with effusion (sero-fibrinous, purulent, hemorrhagic). They may also be classified according to their duration into acute, subacute, and chronic pleurisies. I shall describe the following forms, which are based partly upon their etiology and clinical course, though mainly upon their pathologic manifestations—viz. (*a*) acute plastic pleurisy; (*b*) sero-fibrinous pleurisy; (*c*) purulent pleurisy (empyema); and (*d*) chronic adhesive pleurisy.

Bacteriology.—In all forms of the disease the *direct* causes are various micro-organisms or their irritating chemical products. Conspicuous among these is the *bacillus of tuberculosis*, even, though rarely, found in the pleuritic exudate. Inoculation of guinea-pigs with the latter by Eichhorst gave positive results in 15 out of 23 cases, and by La Damany in 47 out of 55 cases. Although rarely containing bacteria, Netter, Prudden, and others have found in the exudation of **fibrino-serous pleurisy** the *streptococcus pyogenes*, the *staphylococcus*, the *typhoid bacillus*, and the *diplococcus of pneumonia*. The micro-organisms most commonly present in **empyema** are the *micrococcus lanceolatus* and the *streptococcus*, the former especially in the pleurisy associated with pneumonia (in two-thirds of the cases occurring in children—Levy), and the latter in those independent of pneumonia, particularly in adults. Among other bacteria that have been found rarely in the effusion are the *colon bacillus*, the *proteus vulgaris*, the *gonococcus*, the *Ameba-coli*, *Friedländer's bacillus*, *anthrax bacillus*, *influenza bacillus*, and various saprophytic bacteria. Except in the case of the pleuritic exudation (usually purulent) in pneumonia, in which the diplococcus is alone present in about one-half of the cases, the afore-mentioned microörganisms are generally found in association.

ACUTE PLASTIC PLEURISY.

(*Dry, Fibrinous Pleurisy.*)

Pathology.—The lesions are usually circumscribed, the part inflamed being intensely injected. It has lost its natural lustre, and instead has a dull, non-glistening surface "like a tarnished mirror," due to a slight fibrinous exudate. Minute ecchymoses are seen at different points. Later the exudate may become more copious, when the pleura presents a rough, shaggy appearance. On account of the friction between the two pleural membranes in high grades of dry plastic pleurisy, the exudate may be very thick, and its color-appearance is then yellowish- or reddish-gray. This sheeting of fibrinous exudate entangles in its meshes numerous embryonic round cells, out of which blood-vessels and connective tissue are developed. The opposing surfaces of the pleura adhere. Occasionally, in the lighter grades, the disease does not advance to firm adhesion, and in such instances the products of the exudate undergo fatty degeneration and are absorbed. The respiratory movements are but little disturbed in these cases.

Etiology.—The affection may be (*a*) primary or (*b*) secondary. (*a*) By the *primary form* is meant an inflammation of the pleura occurring in previously healthy persons. It is exceedingly rare, and doubtless many instances of true secondary pleurisy are regarded as belonging to this category. Aschoff's studies of 200 cases of pleurisy showed 41 to be idiopathic. Of great etiologic prominence is exposure to cold and wet, and next to this stands mechanical injury. It is more common in men than in women, and especially during the time of active life, on account of the greater degree of exposure of the former than the latter sex. In almost all instances a careful search will disclose the existence of some diathesis (tuberculous, gouty, rheumatic) that may be properly regarded as the favoring cause. The changeable weather of the winter and spring augments the proportion of cases during these seasons as compared with summer and autumn.

(*b*) The *secondary form* of dry plastic pleurisy arises from extension of acute and chronic inflammatory affections of the lungs and other neighboring organs. Hence it frequently follows croupous pneumonia, somewhat less frequently broncho-pneumonia, and more rarely still hemorrhagic infarct, abscesses, and pulmonary carcinoma and gangrene. When pleurisy occurs on the right side it must be recollected that it may have originated in hepatitis. Plastic pleurisy sometimes arises in acute articular rheumatism, to which it may essentially belong. It is an almost constant accompaniment of chronic pulmonary tuberculosis, and may, though rarely, even constitute the primary lesion (primary tuberculous pleurisy). The disease may appear as a complication in chronic alcoholism and in chronic Bright's disease. Finally, inflammation of other serous membranes, as of the pericardium and peritoneum, by direct extension through the lymphatics of the diaphragm, may invade the pleura.

Symptoms.—The affection may vary in intensity between the extremes of mildness and great severity, though, as a rule, well-marked local symptoms attend the onset. Among the latter a ***sharp* "*stitch*" *in the side***, that is usually referred to the nipple, is the most prominent. The pleural *pain* is increased by inspiration as well as by voluntary motion of the affected side, and hence the patient assumes a fixed position in which he favors the affected side by leaning toward it. There is a dry, distressing *cough* that is restrained for obvious reasons, and the respiration is somewhat hurried, painful, and jerking in character until the exudation is poured out, when relief from this and other local symptoms ensues.

The general symptoms are not pronounced, and, save in comparatively rare instances, do not correspond with the local signs. The temperature is not typical, rarely exceeding 103° F. (39.4° C.), and more often it is below 101° F. (38.3° C.). The pulse is usually small and tense or soft in character, registering from 90 to 120 beats per minute. Not infrequently the cases are so mild as to be attended by few, if any, subjective symptoms. The patient may complain of ill-defined, uneasy sensations in the affected side, but does not discontinue his usual occupation. On the other hand, the worst cases of acute plastic pleurisy—which, fortunately, are rare—manifest violent symptoms: there is a distinct chill, a speedy development of high fever (104° F.—4.0° C.), and profound prostration, and the general and local symptoms are proportionately aggravated. The illness then is often a fatal one.

Physical Signs.—On *inspection* the movements of the chest-wall on the affected side are observed to be much restricted, particularly during the first day of the affection. *Palpation* confirms the results of inspection, while *percussion* yields a normal note. *Auscultation* renders audible a grazing friction-sound, most intense at the end of inspiration. These signs are not uncommonly situated at the apices.

With the occurrence of fibrinous exudation *palpation* detects over the corresponding area a diminution of the tactile fremitus. On *percussion* there is, as a rule, a slight though variable degree of dulness; and on *auscultation* the rubbing friction-sounds or a rustling sound due to fine râles are heard both on inspiration and expiration, being intensified by deep breathing. These sounds frequently endure for a day or two after the other symptoms have disappeared. Rarely the plastic exudation may be so extensive as to cause compression of the lung, in which instance the breath-sounds may become bronchial in character; and I have known a case of this sort to be mistaken for lobar pneumonia.

Diagnosis.—By exercising ordinary care the clinician can scarcely mistake other thoracic affections for dry pleurisy, the latter being diagnosticated to a certainty by the presence of the characteristic friction-murmur. *Intercostal neuralgia* may present features not unlike those of acute pleurisy. In both affections there is frequently a history of exposure, followed by severe chest-pains that are excited by coughing and deep breathing. In neuralgia, however, there are painful pressure-points, and the pleuritic friction-sound does not occur. *Pleurodynia* may also give a history very similar to that of acute pleurisy, but the presence of the characteristic physical signs of pleurisy are absent.

Prognosis.—The duration of the affection varies from a few days to three weeks, and the immediate outcome is favorable as a rule. It cannot be doubted, however, that a primary attack predisposes to subsequent attacks, and thus, as a result of repeated seizures, pleural thickening and intrapleural adhesions often arise. Lung-expansion may in this manner be restricted, with the gradual development of interstitial pneumonia as a consequence. Acute plastic pleurisy is not infrequently a terminal condition in serious forms of illness (*e. g.* septicopyemia and chronic nephritis).

Treatment.—The first object in the treatment is to relieve the pain, and this can best be accomplished by the hypodermic use of morphin. The inflammatory process is best controlled by absolute *rest* in the recumbent posture, allowing the patient to assume that position which gives him most comfort. I am also in the habit of administering moderate-sized doses of quinin (gr. iv—0.259—three times daily). After the exudation has appeared, the iodids of iron and potassium, in combination, may be employed. *Locally*, nothing is so effective as cold in the form of the ice-water bag or Leiter's coil, preceded, in robust patients, by the local abstraction of blood (℥iij to vj—96.0–192.0) by leeches. At the end of one week the morphin may usually be discontinued. During *convalescence* the patient should be instructed to take deep inspirations several times in succession, not less than a dozen times each day, with a view to obviating so far as possible the pleural adhesions and other unfavorable consequences. Symptomatic anemia may be present at this time, and should be met by iron given internally. At this time iodin may be used locally with great benefit; I have not, however, seen any favorable results from blisters. For the pain which continues in the side after all detectable physical signs have disappeared the use of the constant current over the seat of the pleurisy for twenty minutes at a time gives almost instantaneous relief (Loomis).

SERO-FIBRINOUS PLEURISY (PLEURISY WITH EFFUSION, SUBACUTE PLEURISY).

Pathology.—During the first stage of sero-fibrinous pleurisy the changes are the same in character as those met with in dry pleurisy, though of severer grade, and usually involving the greater portion of the pleura on the side affected. There is an abundant exudation of serum, and usually the entire pleura becomes coated with a fibrinous exudate, that varies greatly in thickness and arrangement. The latter is thin and smooth in some instances, though more frequently it forms a thick layer, presenting a shaggy surface on the one hand or an irregular, honeycombed surface on the other. Lymph in the form of flocculi is rather abundant in the serous effusion. The interlobular pleural surfaces are also invaded as a rule, in consequence of which they become adherent. The fluid exudate varies greatly in quantity (½ to 8 pints—4 liters), is often of a citron color, and is, in the majority of instances, clear or slightly turbid. Rarely it is of a dark-brown color.

Unless adhesions between the pleural surfaces have previously existed the effusion gravitates to the most dependent portion of the pleural cavity. Microscopically, there are found leukocytes, red blood-corpuscles, endothelial cells, threads of fibrin, and, rarely, crystals of cholesterin and uric acid. The composition of the fluid is almost identical with that of blood-serum, and on boiling it is found to be rich in albumin. Spontaneous coagulation may take place on standing.

Changes in the Neighboring Organs.—So long as the normal retractility of the lung is not overcome by the fluid that collects in the pleural cavity, the latter does not produce positive intrathoracic pressure, and hence does not produce displacement of adjacent organs. It may be assumed that until the pleural sac is at least one-half filled with sero-fibrinous exudate the natural contractility of the lung is not destroyed. At this period there may be a slight displacement of the mediastinum toward the opposite side, due to traction exerted by the normal retractility of the sound lung. Obviously, large effusions must in a mechanical manner displace the pleural membranes, thus causing compression of the pulmonary structures lying above the effusion. A very copious effusion may push the lung up and back against the vertebral column and convert it into a small, flat, bloodless, and airless mass (atelectasis). While a total absence of air in the collapsed lung is due chiefly to compression by the fluid, to some extent, however, the air may be absorbed by the vessels or even by the effusion (Strümpell).

Together with compression of the lung by the effusion, pressure is also exerted by the latter against the mediastinum, causing displacement of the heart. The mediastinum also loses the normal traction-force of the lung upon the affected side, and hence the lung on the sound side draws the mediastinum toward itself by its own retractile energy. Osler shows that even in the most extensive left-sided effusion the heart's apex is not rotated, but that the normal relative position of the apex and base obtain, though the apex is in some instances lifted, and in others the heart lies more transversely. The right chambers of the heart occupy most of the anterior part of the organ, showing that the displacement of the mediastinum with the pericardium and its contents to the right involves no appreciable twisting of the heart itself.

Downward displacement of the diaphragm takes place in extensive effusion, and shows itself on the right side by the lowering of the liver to a variable distance below the inferior costal border; on the left side large effusions produce pressure-displacement of the stomach and the transverse colon, and, to a slighter extent, of the spleen. It must be recollected that adhesions may prevent displacement of any of the adjacent organs.

Etiology.—In the present state of our knowledge the causal factors are identical in nature with those producing dry plastic pleurisy. It is highly probable that the degree of severity is dependent upon the previous condition of the patient, whether he be suffering from some other affection or not, and upon the amount of specific poison gaining access to the pleura.

The affection may be *primary*, but is much more often *secondary*; and this fact may be explained by reference to any of the specific micro-organisms producing the affection.

Direct Causes.—Many of the cases follow quickly upon exposure to cold or wet or an injury to the thorax. I thoroughly agree with those authors who contend that about three-fourths of the cases of sero-fibrinous pleurisy are induced by tuberculous infection of the pleura. The tuberculous process may invade the pleura primarily, but more often it is secondary to tuberculosis of the lungs; less frequently, though oftener than is generally supposed, it is secondary to tuberculous peritonitis. In these instances the tubercle bacilli probably find their way from the peritoneum to the pleura by traversing the lymphatics in the diaphragm. Resinelli believes that pleurisy with effusion may be the direct result of neoplasms of the ovaries. I am convinced that a large percentage of apparently primary cases of tuberculous pleurisy have their origin in a circumscribed and more or less latent tuberculous focus in the lungs. It is not improbable also that tuberculous processes in other viscera may furnish the tubercle bacilli for secondary pleural infection. Moreover, the fact that many cases of sero-fibrinous pleurisy recover does not disprove their tuberculous nature.

The affection is not infrequently secondary to acute articular rheumatism, which is itself most probably a microbic affection. It also arises as a complicating condition in the course of various acute and chronic affections of the chest, as pericarditis and catarrhal pneumonia, and may develop in acute infectious diseases, as typhoid fever or lobar pneumonia. The typhoid bacillus of Eberth has also been known to provoke pleurisy (Bozzolo, Fernet, and others).[1] It may occur as a complication in the chronic affections of various viscera (chronic nephritis, cirrhosis and carcinoma of the liver). The *predisposing causes* are the same as for the dry plastic form.

Symptoms.—The description here refers particularly to primary sero-fibrinous pleurisy, and it is important to recollect that when secondary to other acute and chronic affections characterized by great bodily weakness the pleuritic symptoms may be in abeyance.

With few exceptions the onset is *insidious*, the symptoms being quite mild; but rarely there is a *sudden onset* with active symptoms (rigor, high fever). In the majority of instances the patient first complains of a *stitch-like pain* in the side; this is rarely pronounced, but is aggravated upon deep breathing and upon any muscular exertion. *Dyspnea* soon arises and gradually increases in intensity. *Cough* may be present or absent, and in some instances is attended by a scanty mucoid expectoration that may rarely be blood-streaked.

The *constitutional symptoms* are of correspondingly slow and gradual development. From the commencement of the attack a moderate febrile movement at night may be observed, and the pulse will be found to be frequent, small, and compressible, or, more rarely, tense. At the time of the patient's first visit to his physician he may give a history of having gradually lost flesh and strength for a period of weeks together, though he may not have been obliged to abandon his vocation. He looks pale, his countenance wears an anxious expression, and he is without appetite. These cases frequently drag on from two to four weeks before consulting a physician, the local symptoms going unnoticed, and the patient making complaint only of weakness, anorexia, headache, etc.

Sometimes the period of invasion develops acutely and after lasting a few days the symptoms exhibit a decided remission; but at another subsequent period there may be a sudden recurrence of the local and general phenomena, and particularly of the dyspnea. The pleural cavity, which may have been one-half or two-thirds full, now becomes completely filled.

Special Symptoms.—*Pain.*—Chest-pain is an almost constant but not highly characteristic symptom, and, though usually among the earliest symptoms, it may not be present until a few hours or a day after the commencement of the affection. It may be described as a sharp, shooting pain, and is popularly termed a "stitch in the side." It may, however, be tearing or dragging in character. Its intensity is not a safe indication of the severity of the disease. It is usually referred to a small spot below the nipple or to the mid-axillary region; exceptionally, however, it is more diffuse, and in my experience it has not infrequently been retrosternal or referred to limited areas below the inferior costal border. When absent it may be excited by coughing, sneezing, deep inspiration, and stooping. With the appearance of the effusion the pain diminishes, and, as a rule, soon disappears.

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[1] *Annual of the Universal Medical Sciences*, 1892, vol. ii. p. 12.

Dyspnea.—The breathing is shallow, "catching," inspiration being made up of a series of gasps, and it is hurried in consequence of the severe pleural pain; in copious effusions, that render one lung functionless, the dyspnea may become intense, even attaining to orthopnea. It reaches its most pronounced form in persons who have previously been robust, and in those in whom the effusion has developed rapidly. On the other hand, when the pleural sac fills slowly dyspnea may be absent except on exertion. This symptom often appears before the effusion takes place, and is then due partly to the fever and partly to the pleuritic pain. Following marked disturbances in the respiration, *cyanosis* appears and may become quite marked.

Cough and Expectoration.—Little need be added to what has already been stated. When there is present much expectoration it is not uncommonly due to associated bronchitis or to pulmonary tuberculosis; there may, however, be a total absence of expectoration, and in such instances the exciting cause of the cough is probably the pleuritis. Both the cough and expectoration are apt to be increased during the process of resorption of the exudate as the result of a catarrhal bronchitis that is prone to develop in the re-expanding lung.

Fever.—The rise of temperature is not rapid as a rule, nor does it reach a high point (101.5° to 103° F.—38.6°-39.4° C.). At the end of a variable period—usually one to three weeks—the temperature falls by lysis, and soon touches the normal. The temperature may be of the continued type in many acute cases. In subacute forms the temperature rarely rises above 101° F. (38.3° C.), or the fever may, finally, become hectic. The surface-temperature of the affected side is from one-half to two degrees (0.4°-1.6° C.) higher than that of the normal side.

Pulse.—The pulse is quickened, beating 100 or more per minute, and its volume and tension are diminished. Irregularity both of the volume and rhythm of the pulse may also be observed. These pulse-characteristics are to be attributed to the pressure of the effusion upon the heart and great vessels.

Gastro-intestinal Symptoms.—Loss of appetite is commonly present, and more rarely nausea and occasional vomiting may arise at the outset. Constipation is the rule.

Renal Symptoms.—The amount of urine is diminished both during exudation and while the exudate remains at its maximum level. The daily quantity may not exceed eight or ten ounces, but the specific gravity is increased, ranging from 1018 to 1028. Rarely, the quantity is increased with existing effusion. An increase in the daily amount of urine excreted is frequently the first sign of commencing absorption of the exudate, and the rapid resorption of the copious effusion may greatly augment the flow of urine to 80 or 100 ounces (2.5 to 3 liters) daily (Strümpell). The cause of the diminished secretion of urine is, in the main, diminished arterial pressure.

Physical Signs.—The physical signs of sero-fibrinous pleurisy differ with the amount of effusion present, and also with the particular stage of the affection: those of the first stage, however, are identical with the signs pointed out in connection with dry plastic pleurisy, and need not be restated here. We will note the physical signs (1) during the stage of effusion, as well as (2) those presented when absorption of the effusion has taken place.

(1) *Stage of Effusion.*—When the pleural sac is only partly filled there is noted, on *inspection*, but little change in the thoracic contour. The respiratory movements are, however, restricted, owing to mechanical hindrance to the lung-expansion. In the majority of instances the effusion increases until positive intrathoracic pressure and noticeable bulging in the middle and lower third of the chest-wall on the affected side take place; the intercostal spaces below are shallow, widened, and sometimes even effaced. The apex-beat of the heart is displaced, being visible in right-sided pleurisy to the left of the vertical mammary line in the fourth and fifth interspaces, and in left-sided pleurisy to the right of the right mammary line, or even beyond, in the third and fourth interspaces. The apex of the heart may take a position behind the sternum, when no impulse will be visible.

Palpation.—The limited range of expansion is readily appreciated on palpation, and in large effusions the chest-wall is practically fixed. The separation of the ribs and the obliteration of the intercostal spaces are easily made out in the same manner. Edema of the chest-wall is rarely present, and fluctuation almost never. An important and early physical sign is the diminished tactile fremitus, which is soon abolished, except in infants, in whom it may be excited on crying. This is a less valuable sign in women than in men, owing to the differences in the vocal vibrations in the two sexes. In copious effusions tactile fremitus may sometimes be obtained when bands of adhesion, which serve as a medium for the transmission of vocal fremitus, connect the pulmonary with the costal pleura. The apical impulse can also be readily located by palpation. The displaced spleen or liver can be readily felt through the abdominal wall, and must not be mistaken for an actual enlargement of these organs.

Mensuration.—It must not be forgotten that in right-handed adults the right side is, normally, slightly larger than the left; and it is only after the effusion is considerable in amount that the cyrtometer shows any alteration in the thoracic contour or an enlargement of the affected side. The tape, however, exhibits the difference in expansive motion of the two sides early, or when there is a moderate amount of fluid. At the end of expiration the circumference of the affected side will be found to be one or two inches greater than that of the left side, while at the end of inspiration the difference will be but slight. The cyrtometric tracing also shows a discrepancy between the horizontal outlines of the two sides.

Percussion.—At first the percussion-note is impaired, either posteriorly or in the infra-axillary region, and a little later there is dulness, tending toward flatness (deadness), with increasing effusion. The resistance to the pleximeter-finger becomes greatly augmented. In cases in which the effusion rises to the fourth rib anteriorly there is dulness over the fluid above and absolute flatness below. Since both the flatness and dulness are due to the free fluid, it is obvious that the line of demarcation must change with the posture of the patient; hence the limit of dulness will be higher in the sitting than in the recumbent position. When the pleural sac is filled or when the effusion is confined by adhesions, movable dulness is not obtainable. Displacement of the pleuritic fluid when the patient's position is changed and also with the movements of the diaphragm has been noted with the fluoroscope (Bergoné and Carrière). When the exudate rises to the lower border of the third rib, the percussion-note above the line of dulness is tympanitic or vesiculo-tympanitic (*Skoda's resonance*); this holds also in more moderate effusions, and is attributable to mediate relaxation of the lung. In copious exudations the cracked-pot sound may be elicited immediately below the clavicle, and "*Williams's tracheal tone*" may sometimes be obtained. This may also be obtained at a point corresponding to the seat of the compressed lung. When the patient is sitting or in the erect posture the upper limit of dulness in large effusions is not a horizontal line, but is highest at the spine and falls as we proceed to the front, which is its lowest point. The upper line of dulness in moderate effusions begins "relatively low down in the back, passes upward from the vertebral column, and soon turns upward and proceeds obliquely across the back to the axillary region, where it reaches its highest point; thence it advances in a straight line, but with a slight descent, to the sternum" (Ellis). This curved line resembles the italic letter *S* (Garland). On the right side the flatness is continuous with that of the displaced liver; on the left it passes into and may obliterate Traube's semilunar space.

Auscultation—The signs of the first stage have already been described (*vide* Plastic Pleurisy). With the appearance of the effusion the breath-sounds become weak, distant, and have a bronchial quality. Soon the respiratory sounds over the affected side will be entirely absent, except near the upper level of the fluid posteriorly, where distant bronchial breathing is audible. The latter sounds may exhibit a metallic or amphoric quality, and may be accompanied by râles (pseudo-cavernous signs). The latter are more frequently met in children than in adults, and often give rise to a false diagnosis. Above the level of the fluid there is broncho-vesicular breathing, and on the opposite side intensified breath-sounds may usually be noted. In pneumonia with pleural effusion there may be loud and persistent bronchial respiration over the exudate. The vocal resonance is diminished and may manifest a nasal quality, simulating somewhat the bleating of a goat (*Laennec's egophony*). This is best obtained near the upper level of the fluid in large effusions, and at or above the angle of the scapula when the effusion is moderate.

(2) *Stage of Resorption.*—With resorption of the fluid there is a decrease in the size of the affected side, together with a return of the normal appearance of the intercostal spaces and the respiratory movements. In many instances there is positive retraction, leading to thoracic deformity with displacement of neighboring organs toward the affected side; and this retraction may be either general or circumscribed. The inferior intercostal spaces are more or less narrowed; the shoulder droops; the nipple approaches the median line; the spine may be curved, the convexity being directed toward the sound side (quite rarely toward the

affected side); and the scapula projects from the chest-wall on the affected side. In children, and even in adults, the lungs and thorax gradually expand in order to overcome this chronic deformity. On the other hand, permanent shrinkage of the thorax may occur.

Palpation.—The tactile fremitus closely follows the fluid as it subsides from above downward without any extreme degree of thickening of the pleural membranes, though cohesion of their surfaces may prevent its return over the lower segment. The inspiratory movement of the chest-wall gradually returns, but not to its former limit.

Mensuration shows a steady diminution in the size of the side involved, which finally becomes smaller than its fellow.

Percussion.—The dull or flat note gives way to normal percussion-resonance, proceeding from above downward in a gradual manner; but the latter is not renewed over the lower portion of the pleural cavity for a long period after the exudation has disappeared. The abnormal areas of flatness due to displacement of organs (liver, spleen, heart) also disappear.

Auscultation discloses most important signs during the stage of absorption. The breath-sounds reappear at first above, and then lower down, until the base is reached. With commencing subsidence of the fluid the respiratory sounds are feeble and distant, but later they resume their natural distinctness; and partly as a result of the revival of the natural muscular tonicity, and partly in consequence of the disappearance of the fluid, the two roughened pleural surfaces come in contact and play upon one another, giving rise to a rubbing, creaking friction-sound on auscultation. These friction-murmurs may persist for months after the effusion has been absorbed. Occasionally the lower portion of the compressed lung remains permanently inexpansile; the upper portion of the lung is now the seat of compensatory emphysema.

X-rays.—Williams[1] states when the effusion is large no more rays pass through it than through the liver, and the outlines of the diaphragm, ribs, and heart are obliterated on the side of the effusion. The fluoroscope also shows the direction and extent of cardiac displacements due to pleural effusions. Williams affirms that displacement of the heart to the right may not be recognized by percussion, even when it has been pushed much beyond its normal place. The heart-sounds return to their normal position.

Special Clinical Forms of Acute Sero-fibrinous Pleurisy.—(1) **Tuberculous Pleurisy.**—This is, in the majority of instances, secondary to pulmonary tuberculosis. On the other hand, the primary lesions may be situated in the pleural sac and give rise to (1) *Acute sero-fibrinous pleurisy* (with the usual course); (2) *Subacute pleurisy* (with insidious course), leading to tuberculous invasion of the lungs; and (3) *Chronic adhesive pleurisy*, in which the course and physical signs correspond with those that will be depicted in a special section on Chronic Pleurisy.

The morbid lesions are similar to those met with in other forms, plus the specific tubercles, which may be exceedingly numerous (miliary tubercles) on the one hand, or confined to a few circumscribed areas on the other. This variety of pleurisy has no special etiologic connection with empyema, and the effusion is usually sero-fibrinous and often blood-stained.

Brief reference should be made to those instances in which tuberculous pleurisy is followed by tuberculous pericarditis or peritonitis, or both. The two latter affections will be considered elsewhere. Suffice it to state here that tuberculosis of the serous membranes usually pursues a chronic course, lasting a year or more, and exhibits widely varying degrees of intensity in its symptoms in different cases, and from time to time in the same sufferer. We must grant that tuberculous pleurisy may pursue a favorable course with apparent recovery, though too often, after a variable interval of time, tuberculous symptoms are manifested.

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[1] *Philadelphia Medical Journal*, January 6, 1900.

(2) **Diaphragmatic Pleurisy.**—This term is applied to those instances in which the diaphragmatic portion of the pleura is involved, either alone or in part. There occurs an exudate that may be either plastic or sero-fibrinous, though rarely large in amount. The *symptoms* are acute, and the pain, which is lancinating in character and situated in the epigastric region, is the most prominent feature. Geuneau de Mussy[1] holds that pain along the tenth rib, extending from the anterior extremity to the sternum and xiphoid cartilage, is pathognomonic. It is increased by deep inspiration and by pressure over the insertion of the diaphragm at the tenth rib, and often abates when effusion takes place. Dyspnea is a marked symptom in most cases, and the patient may be forced to assume a stooping or sitting posture, the respirations being superficial, purely thoracic, and "catching." Cough, nausea, and even vomiting, may occur. In a case under my own care vomiting, due most probably to associated peritonitis, was a troublesome symptom.

The *constitutional features* are quite pronounced, and particularly the fever, which exceeds that met with in other forms of pleurisy. The patient's anxiety is extreme. The effusion may be purulent, and if so bulging of the lower intercostal spaces, followed by edema, may occur. The physical signs are for the most part negative.

(3) **Encysted Pleurisy.**—This term has reference to effusions that are circumscribed in consequence of adhesions between the pleural membranes. There may be two or more pouches, with or without communication. This so-called encapsulated pleurisy may occupy any part of the chest, and is exceedingly variable in extent. The symptoms and physical signs are rarely trustworthy for diagnosis, but should usually afford ground for suspicion, and hence should lead in every instance to the employment of the exploratory puncture.

(4) **Interlobar Pleurisy.**—This variety is usually secondary to, or associated with, the ordinary type of acute sero-fibrinous pleurisy. The serous surfaces, dipping between the lobes, are involved in the inflammatory process, and the fluid becomes encapsulated in this position in consequence of interlobar pleural adhesions. It is more frequent on the right than on the left side, and its favorite seat is near the root of the lung, between the upper and middle lobes. Osler[2] met with a case following pneumonia in which there was between the lower and upper and middle lobes of the right side an enormous purulent collection that looked at first like a large abscess of the lung. Fistulous connection with a bronchus often occurs, and the purulent expectoration that follows may be the first symptom to attract the attention to the process of suppuration in the thorax. Prior to the occurrence of this accident the patient gives evidence of indisposition without definite symptoms. The

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[1] *Arch. gén. de Méd.*, 1853, vol. xi., quoted by Fox.

[2] *Practice of Medicine*, p. 567.

patient may or may not give a clear history of antecedent pleu
These cysts contain, as a rule, but a small amount of fluid, and do
cause much bulging of the intercostal spaces. Indeed, in a case of
own at the Philadelphia Hospital there was actual retraction, thoug
aspirating needle showed the presence of effusion.[1]

(5) **Hemorrhagic Pleurisy.**—By this term is meant an admixtur

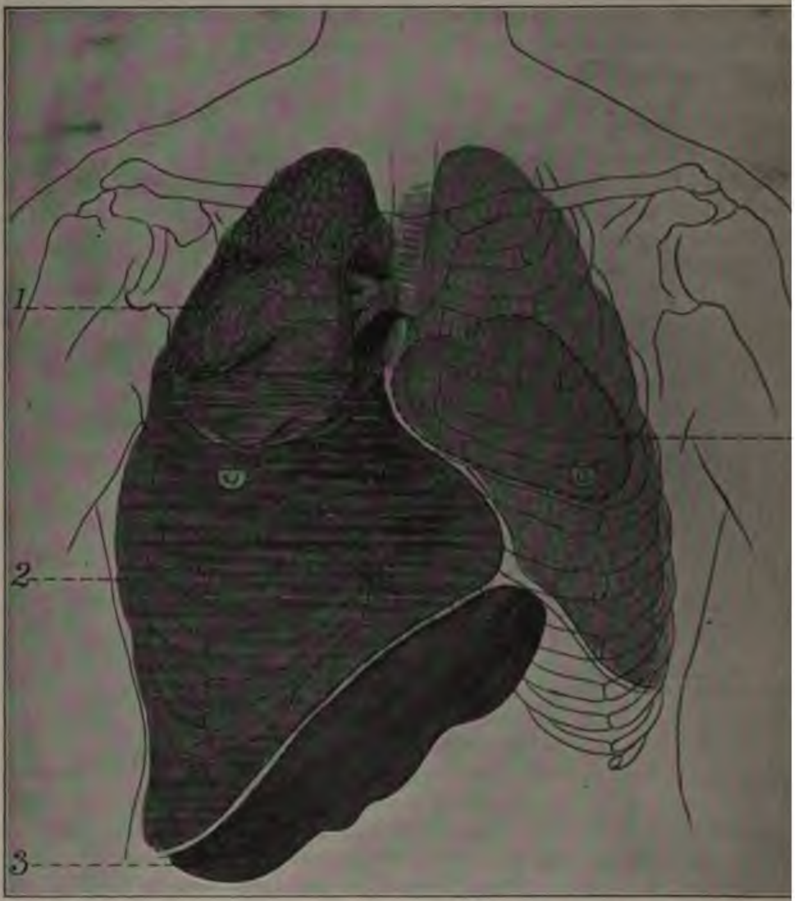

FIG. 45.—Illustrating pleurisy with effusion: 1, compressed lung-tissue, giving dull tympany o
cussion; 2, fluid exudation obliterating intercostal spaces; 3, depressed liver; 4, displaced h

blood with the exudate in acute sero-fibrinous pleurisy, in quantities
ficient to be detectable by the unaided eye. The condition must be
arated from *hemothorax*. The *causes* of hemorrhagic pleurisy are—(1)
berculous infection, either of the miliary or the chronic (circumscri
form, following tuberculous disease of the lung; (2) Carcinoma of
pleura; (3) Bright's disease and cirrhosis of the liver; (4) Adynamic s
of the system associated with malignant forms of acute infectious dis
(pneumonia). (5) Advanced age and alcoholism are among the con
uting conditions.

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[1] *International Clinics* (1894), vol. i. p. 39.

In a certain proportion of the cases no assignable cause can be found, and if the condition be observed for the first time after aspiration, the fact that it may have been engendered by an accidental wound of the lung must be remembered.

Diagnosis.—In diagnosticating pleurisy our attention must be directed chiefly to the physical signs. Unfortunately, in view of the fact that the rational symptoms are often ambiguous, a physical exploration of the chest is apt to be neglected. The chief difficulties are encountered in distinguishing this affection from conditions in which the lung is either consolidated, retracted, or compressed by solid new growths, etc. Chief among the former is *croupous pneumonia*, and I have tabulated below the most important distinctions between it and pleurisy. The reader will be further aided by comparing Fig. 45 (which shows the physical conditions in pleurisy) with Fig. 15, on page 152, which shows the physical conditions in pneumonia.

Pleurisy with Effusion.	Primary Lobar Pneumonia.
Rational Symptoms.	
Onset marked by chilliness, persisting for a few days.	*Onset* acute, rigor, lasting one hour.
The pain is sharp, "stitch-like," and strictly localized.	Acute pain (similar), but soreness more diffused.
Cough irritating; no expectoration, or catarrhal.	Cough more marked and accompanied by rusty or bloody expectoration.
Absent.	Sputum shows presence of pneumococcus.
Moderate fever of continuous type; decline by lysis.	Intense fever; decline by crisis from the fifth to the ninth day.
Systemic prostration (moderate).	Prostration marked.
Countenance pale and anxious.	Countenance congested; mahogany flush on the cheeks.
Herpes does not appear.	Herpes quite common.
Physical Signs.	
Inspection.	
Distention of the thorax.	None.
Palpation.	
Diminished or absent tactile fremitus.	Marked tactile fremitus (absent only when a bronchus is plugged).
Percussion.	
Flatness, with great resistance to the pleximeter-finger.	Dulness less complete, less resistance, and sometimes a tympanitic note.
Shows displacement of neighboring organs.	No displacement of neighboring organs, if uncomplicated.
If the sac be partly filled, there is a change in the line of flatness on change of position.	Absent.
Auscultation.	
Diminished or absent breath-sounds, bronchial breathing frequent, but diffused and distant and unaccompanied by râles, as a rule.	Harsh bronchial breathing and presence of râles in first and third stages, unless a bronchus be plugged.
Vocal resonance diminished or absent; egophony.	Bronchophony (loud), unless a bronchus be blocked.
Friction-sound in early and late stages.	No friction-sound, except crepitant râles in the first stage.
Aspiration.	
Yields serum.	Yields a few drops of thick blood.

Consolidation of the lung, due to tuberculous infection, may be differentiated from pleurisy with effusion by means of the physical signs contrasted in the foregoing table, by the history of the case, and by the discovery of the tubercle bacillus in the sputum.

Hydrothorax presents physical signs that simulate strongly those of pleural effusion. Hydrothorax, however, gives the history of cardiac or renal disease, is oftener bilateral, and is unassociated with a rise in temperature or with the pain or friction-sounds peculiar to pleurisy. In hydrothorax the withdrawn fluid has a specific gravity below 1015, while that of the pleural exudate is above 1017.

Tumors and *cysts* of the thorax will give complete dulness, will displace the heart, and compress the lung on the affected side, thus causing an absence of the respiratory murmur, etc. But the history of the case, the situation of the dulness (usually over the upper or middle parts of the lung), the absence of uniform distention extending to the base, and the exaggerated tactile fremitus and vocal resonance will serve to distinguish these affections from pleurisy with effusion.

Echinococcus cyst of the liver, or *abscess* of this organ, pushing upward, will cause retraction or even compression of the lung, and hence will also produce most of the physical signs of pleurisy with effusion. The former affections can be discriminated only by a correct appreciation of the history, by the presence not infrequently of a friction-sound on auscultation, and by immovably fixed upper convex boundary of dulness. If doubt remains, an exploratory puncture should be made, and the fluid withdrawn should be subjected to a chemical, microscopic, and bacteriologic investigation.

An *enormous pericardial effusion* may be mistaken for a pleural effusion on the left side. In the former, however, there is commonly a history of rheumatism, and dyspnea is the most urgent symptom, while the heart-sounds are greatly enfeebled; moreover, the heart is not displaced to the right as in pleural effusion. Again, flat tympany is obtained in the posterior portion of the axilla and good pulmonary resonance at the base in the postero-lateral region of the chest, differing from the results of percussion in pleuritis.

For practical purposes it is desirable to distinguish the *tuberculous* from the rarer forms of pleurisy. This is sometimes possible by paying due regard to the previous history of the patient, including hereditary taint, by noting certain clinical peculiarities (such as associated disease of other serous membranes and of the lung and bilateral inflammation of the pleura), and by the results of an examination of the exudate. In a dubious case the guinea-pig should be inoculated with the exudate, and if the patient be tuberculous positive results may be confidently expected.

Duration and Prognosis.—This depends largely upon the cause. The course of acute sero-fibrinous pleurisy is not definite, but is made up of two parts—the febrile followed by the non-febrile stage. The fever lasts from one to three weeks, and is due to an infectious inflammation; it corresponds to the period when the effusion occurs, and the appearance of a non-febrile period indicates the subsidence of the inflammation. The exudate may be poured out rapidly, and may be absorbed not less rapidly; more commonly, however, the effusion takes place rather gradually, and the same is true of resolution. Again, large effusions may persist in consequence of a purely mechanical hindrance to resorption; and finally, the course may become subacute or chronic in consequence of the development of empyema. The continued absence of bacteria in the pus speaks for tuberculosis. Such facts as these constitute an explanation for the great differences in the duration of the cases. Simple sero-fibrinous pleurisy, including the hemorrhagic variety, unless it appears as a complication in the later stages of some other grave disease, has a comparatively favorable prognosis. Death rarely ensues suddenly, without adequate lesions to explain its occurrence. Moreover, the superaddition of symptoms of empyema renders the prognosis far less hopeful. Again, the crippling influence upon the lung-tissue of previous attacks, owing to resulting adhesions, must be borne in mind, since chronic bronchitis and emphysema often supervene.

Treatment.—In the first stage the treatment is the same as for dry or plastic pleurisy. During the second stage, that of effusion, the objects of treatment are threefold: (1) To limit the extent and intensity of the inflammatory process; (2) To accomplish the removal of the effusion; and (3) To support the strength of the patient.

(1) **To Limit the Extent and Intensity of the Inflammatory Process.**—To this end two classes of agents are employed—namely, (*a*) *Internal*, and (*b*) *External*.

Among the latter are counter-irritants, as sinapisms and iodin, by means of which gentle but constant counter-irritation is to be maintained. Another agent of great worth is cold, applied by means of the ice-bag or ice-water bag, and if the temperature rises to 102° F. (38.8° C.) cool spongings of the surface of the body, together with the use of the ice-cap, will be found highly useful. Roberts recommended keeping the affected structures at complete rest by fixing in a mechanical manner the side affected. For this purpose strips of adhesive plaster must be firmly and evenly applied to the chest, and by this means the pain is relieved and the amount of inflammatory product poured out is greatly limited.

The *internal* remedies embrace quinin, the salicylates, and opium. Opium and quinin are potent in controlling inflammation of serous membranes; the former being given preferably either in the form of suppositories or hypodermically, and the latter in divided doses, in capsule, followed by a few drops of mineral acid, administering gr. xvj to xx (1.036–1.296) daily.[1] I have observed good results from the salicylates (ʒj–ij—4.0–8.0, daily), which have been warmly advocated by Fiedler, Koester,[2] and others, as valuable in mitigating or even aborting the inflammation of the pleuræ, and thus in limiting the amount of effusion. It must not be forgotten that the effusion is due to an inflammation, and not to a simple transudation. The use of mild diaphoretics and diuretics, coupled with repeated small doses of salines, also aids in reducing the inflammation in the pleura. With a subsidence of the inflammatory process the temperature falls, and, when the latter reaches a point near to the normal, our efforts should be directed toward the fulfilment of the second leading indication, (2) the **removal of the effusion.**

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[1] *International Clinics* (1892), vol. i., second series.

[2] *Annual of the Universal Medical Sciences* (1893), vol. i. (A–31).

Little is to be accomplished by local means, though iodin, persistently employed, sometimes does good. The following ointment may also be tried:

℞. Ung. ichthyol. (12 per cent.),	
Ung. iodinii comp.,	*āā.* ʒvj (24.0);
Ung. belladonnæ,	q. s. ad ℥ij (64.0).—M.

Sig. Apply twice daily.

Blisters are not admissible.

Mild hydragogue cathartics, and especially the salines, after the Matthew Hay method (*i. e.* ʒij to ℥ss—8.0–16.0, in the smallest possible amount of water, on rising in the morning), stimulate absorption from the pleural cavities by draining the blood of a certain amount of serum. Unirritating diuretics may also be employed, but I have found no appreciable advantage from their use. Free diaphoresis (from the use of pilocarpin) sometimes assists in the absorption of the exudate, but it should not be employed in the presence of feeble heart-action or marked displacement of the organ. Among measures to promote absorption, the best, in my own experience, is the following combination:

℞. Potassii iodidi,	ʒj (4.0);
Syr. ferri iodidi,	ʒij (8.0);
Syr. sarsap. comp.,	℥j (32.0);
Ess. pepsinæ,	q. s. ad ℥ij (64.0).—M.

Sig. ʒj (4.0) every four hours, diluted; the dose to be doubled at the end of four days if well borne by the stomach.[1]

The patient should be put upon a dry *diet* in order to increase the plasticity of the blood, which is thus induced to absorb the liquid exudate from the pleural cavity. The *modus operandi* of this treatment is different, but the effect aimed at is the same as when saline purgatives are given. The exudation, however, defies all efforts at removal in about 33 per cent. of the cases, and in such the withdrawal of the liquid by aspiration (thoracentesis) must be practised. The indications for thoracentesis arise at two different periods in the course of pleurisy with effusion:

(1) During the febrile stage, while efforts are being directed to combating the inflammatory process. The object during this stage is to avert imminent danger to life, and not merely to remove the fluid. The conditions demanding immediate thoracentesis are—(*a*) when one pleural sac is completely filled or when Skoda's resonance extends from the clavicle downward no farther than the second interspace; (*b*) in double pleurisies, when both sides are half filled, since death may occur from rapid filling of one or the other side; (*c*) in cases of copious effusions, upon the first signs of involvement of the unaffected side, such as moist râles, broncho-vesicular breathing, and impaired resonance; (*d*) the appearance of serious symptoms, such as orthopnea or syncopal attacks with cyanosis; (*e*) marked displacement of the heart, especially if one or more murmurs develop in the organ.

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[1] The author has employed this formula in more than 60 cases with very good results.

(2) The indications for aspiration during the second or afebrile period, when the main object is to remove the exudate, are—(*a*) if no diminution in the quantity of liquid effusion has taken place one week after the temperature has reached the normal; (*b*) in subacute cases, in which there is little, if any, temperature from the beginning; aspiration should not then be withheld longer than three weeks.

The operation is free from danger if carried out under antiseptic precautions and if a modern aspirator is employed. The instrument should always be tested before it is used. The patient rests in bed in the semi-recumbent posture, the arm of the affected side being brought forward with the hand placed on the opposite shoulder, so as to separate the ribs from one another. The point of puncture is in the sixth interspace on the right-hand side and the seventh interspace on the left, in the mid-axilla, or just below the outer angle of the scapula in the seventh right and eighth left interspaces, respectively. An assistant draws up the skin from the interspace, while the operator uses the fore finger of his free hand as a director. The needle should be introduced with a quick thrust, hugging the rib below the interspace, but endeavoring to avoid striking its periosteal covering. The fluid may not be obtained at the first operation, and the reasons for this failure are several. The costal pleura may be excessively thickened, or we may meet with a much-thickened fibrous band. Again, the fluid may be encapsulated; and, lastly, the needle may become blocked. Under these circumstances repeated trials should be made.

The amount of fluid withdrawn at one time should never be large (℥xij to xxiv—384.0–768.0), though a relatively larger quantity may be taken during the febrile stage than during the afebrile, since in the latter instance the lung has been compressed for a longer period of time. The fluid is allowed to drain away slowly, a small needle being used, so as to invite the lung to expand in a gradual manner. If this precaution be not taken, the paretic pulmonary capillaries are apt to become the seat of sudden fresh congestion, followed by edema, and often by a speedily fatal termination. Thoracentesis is to be repeated at intervals of several days if nature does not take up the work of absorption, following the first operations. If during the operation incessant cough, dyspnea, a tendency to syncope, marked thoracic constriction, or sudden intense pain be developed, the needle must be withdrawn instantly.

Thoracentesis should not be resorted to in cases in which croupous pneumonia is associated, and never in very aged and excessively feeble persons.

(3) **To Support the Strength of the Patient.**—The powers of the system are to be maintained by a nutritious diet, bodily rest, and other hygienic measures. The lighter forms of solid food may be allowed whenever they are found to agree, and it is important to promote the digestive power, should the latter be weak, by the administration of suitable remedies. During the stage of *convalescence*, therefore, tonics (strychnin, quinin, and arsenic) are to be administered. The dietary should be liberal, though composed of wholesome articles. Gentle exercise in the open air is to be encouraged, and massage of the muscles of the affected side tends to re-establish their usual vigor. To bring about the best possible chest-expansion nothing is so good as light gymnastic

exercises, together with the methodical practice of deep inspirations for a minute or two at intervals of three or four hours. I am of opinion that the management of the third stage, or that of convalescence, is similar to that of tuberculosis.

EMPYEMA (PURULENT PLEURITIS).

Definition.—A suppurative inflammation of the pleura.

Pathology.—On opening the pleural sac after death we may find a thick, creamy pus, though more frequently it is sero-purulent and separated into two layers—an upper, clear, greenish-yellow serous, and a lower, thick, purulent layer. In a smaller proportion of cases the exudate is fibrino-purulent. The odor emitted from the purulent collection is either sweetish or fetid (*e. g.* when due to wounds), and, when the condition is associated with gangrene of the lung or pleura, horribly offensive. Microscopic examination shows that the inflammatory products are identical with those of purulent inflammation in general. The pleural membranes are the seat of a more intense inflammation than in acute sero-fibrinous pleurisy, and are greatly thickened (1 to 2 mm.). They present a granular suppurating surface, and both visceral and costal pleuræ exhibit perforations, and the latter, often erosions.

Histologically, the altered membranes consist of new connective tissue, new blood-vessels, and numerous leukocytes.

Etiology.—The following are the chief circumstances under which empyema arises: (1) As a sequel of the acute, sero-fibrinous variety. However clear the effusion may be, it always contains corpuscular elements, which in the further progress of certain cases undergo coincident increase in numbers until the effusion presents a milky aspect, when it is said to be purulent. Thoracentesis may be responsible for this change, though never if performed under rigid aseptic precautions.

(2) In children the effusion early becomes purulent in many instances.

(3) Secondary to the acute and chronic infectious diseases—*blood metastasis*—(pyemia, scarlatina, pneumonia, tuberculosis, and dysentery most frequently; typhoid fever, measles, whooping-cough rarely).

(4) Secondary to malignant affections of contiguous organs (lungs, esophagus), or tuberculous cavities which perforate the pleura. Rarely, carious ribs and vertebræ may cause empyema.

(5) Lymphatic metastasis is probably an important means by which bacteria reach the pleura from neighboring but not contiguous tissues (McFarland).

(6) Injuries to the chest may set up empyema (fracture of the ribs, stab or other penetrating wounds).

Bacteriologic investigation has shown that the organisms most frequently present are the micrococcus lanceolatus (*meta-pneumonia*), streptococcus, staphylococcus, and tubercle bacillus. The cases due to pneumococci usually pursue a favorable course. The leptothrix pulmonalis is often found in putrid effusions.

Clinical History.—The symptoms vary with the cause. The *onset* may be characterized by acute symptoms, such as rigor, followed by high temperature and signal prostration, and in the affected side there may be severe pains, aggravated by deep breathing and bodily movements.

If the exudate becomes gangrenous, a *typhoid state* develops early, and the case is apt to prove fatal in the course of a few weeks. It is quite a common event for the acute symptoms that characterize the invasion to be replaced at the end of a week or more by the more obscure rational symptoms of chronic empyema. The latter, however, may develop very insidiously as a secondary affection. The rational symptoms in a well-marked case should always excite a suspicion of the presence of the affection, but cannot set the question of diagnosis at rest. The *local symptoms* (pain cough, and expectoration) are of a mild character; the dyspnea, however, that is usually present may be more or less intense. I have on more than one occasion found an utter absence of these symptoms. The *general symptoms* are those of septic infection—diurnal chills occurring at irregular intervals, followed by intense paroxysms of fever and profuse sweating—and such patients lose flesh and grow pale and weak. The temperature is higher than in pleurisy with effusion and is intermittently, though irregularly, elevated.

Peptonuria is a symptom of purulent pleurisy that is not without diagnostic value. It, however, also occurs in suppuration associated with the third stage of pulmonary tuberculosis, and in suppuration due to other causes. While not indicative of empyema, it nevertheless serves sometimes to eliminate sero-fibrinous pleurisy. The urine also contains *indican* in excess in the various suppurations, at least from time to time, if not constantly. *Blood-examination* invariably shows leukocytosis.

If the pus is not removed artificially, it frequently breaks into the lung, penetrates it, and finally discharges through a bronchus. Pneumothorax now tends to supervene. Traube contends that necrosis of the pulmonary pleura may allow of the soaking of the pus through the spongy lung-tissue into the bronchi, without the establishment of a fistulous connection between the latter and the pleural sac, and hence without the formation of pneumothorax. Besides rupture into the lung and external rupture, empyema may perforate neighboring organs, as the esophagus, pericardium, stomach, and peritoneum. In rare instances the pus burrows along the spine behind the peritoneum and the psoas muscle, reaching, finally, the iliac fossa and simulating psoas or lumbar abscess.

Physical Signs.—These are, for the greater part, identical with those of pleurisy with effusion. Attention will therefore be called only to such as are more or less distinctive of the affection. Slight *edema* of the chest-wall over the seat of effusion, especially in children, is often present, and if the pleural sac be not aspirated, the abscess may point externally and evacuate itself spontaneously. In the latter event a *protrusion* between the ribs shows itself: this may be the seat of fluctuation, and present an inflammatory appearance prior to its rupture, with subsequent discharge of its contents. The opening is usually found in the fifth interspace in front, and less frequently in the third and fourth interspaces or below the angle of the scapula behind. The upper level of the fluid does not change so readily on changing the posture of the patient, but requires a longer period of time.

Baccelli's sign, or the transmission through a serous exudate of the whispered voice, is sometimes an aid in the discrimination of pleurisy with effusion from empyema. According to my own observation, though it is not invariably propagated by large serous exudations of the pleura, it is yet detectable in a large majority of instances, whilst I have never observed it in empyema.

Pulsating Pleurisy.—Pulsation synchronous with the cardiac beat in pleural effusion has received various designations (*pulsating empyema*, *empyema necessitatis*, *pulsating pleurisy*). The latter term is the most appropriate one, in view of the fact that it occurs not only in empyema necessitatis, but also in empyema (which manifests no tendency to point externally) and rarely in sero-fibrinous pleurisy.

Its *etiology* is not definitely known. The principal causal factors, however, seem to be—(1) a copious effusion; (2) paresis of the intercostal muscles, inducing relaxation of the thoracic wall; (3) a somewhat forcible heart-beat (Henry). The rational *symptoms* of empyema are present. The *physical signs* are also identical with those of the latter affection, with the pulsation superadded. There are instances in which palpation alone detects the systolic pulse in the pleural effusion. With rare exceptions the effusion occupies the left pleural sac. The pulsation may be limited to two or three interspaces or it may be visible over the entire antero-lateral aspect of the chest; pulsation at the back, however, is rare.

Differential Diagnosis.—An absolute distinction between empyema and *pleurisy with effusion* rests solely upon the results of an aseptic exploratory puncture. For this purpose the needle attached to the ordinary hypodermic syringe, or, preferably, the surgeon's exploring-needle, may be employed, withdrawing but a very small quantity of the fluid, which, if purulent in character should be examined bacteriologically.

Pulsating pleural effusion simulates closely *aneurysm of the thoracic aorta*. When pulsation occurs in empyema, however, it is seen to be to the left of the normal course of the aorta: the rational symptoms and usual physical signs of purulent pleural effusion are usually present also, while the vascular symptoms and signs of aneurysm of the aorta (thrill, bruit) are absent.

Prognosis.—Empyema is a serious disease, but, obviously, the outlook will be modified by the special etiology. Spontaneous absorption may occur, though it is extremely rare. The discharge of the contents of the pleural sac through the bronchial tubes is a comparatively favorable event, some cases in which this occurs recovering, while in others death follows in consequence of the sudden inundation of the bronchi. An empyema may, in rarer cases, empty itself externally with favorable issue (*empyema necessitatis*). Evacuation of the pleural cavity is often followed by a continuous discharge of pus for an indefinite period. As a result of the long-continued suppurative process, death may take place by slow asthenia. It must not be forgotten, however, that an unfavorable termination may be, in part at least, ascribable to certain associated affections (phthisis, pericarditis). Double empyema, fortunately a rare condition, is exceedingly grave.

Among children the outlook is much more favorable than among adults. The prognosis has been rendered less serious by the application of surgical principles in the treatment of the disease. In all cases in which recovery ensues there is a progressive obliteration of the pleural cavity, owing to adhesions, which finally become universal and lead to marked retraction of the affected side (*pleuritis retrahens*).

Treatment.—The treatment of empyema is chiefly surgical. In a child the condition may terminate in recovery without operation, and hence may, at this period of life, be allowed to run for two or three weeks, thoracentesis being resorted to if suffocation be threatened. In an adult, however, if the purulent effusion be copious, aspiration should be performed at once as a temporary means of relief. Empyema following pneumonia may terminate favorably after one or more tappings; but unless contraindicated by an unfavorable general condition of the patient, such as is met with in the closing stages of pulmonary tuberculosis, free incision should be made without delay. The pleural sac should be opened in the fifth or sixth interspace outside of the mammary line, the incision being from 2 to 3 cm. in length. Resection of a rib (Estlander's operation) is advocated, but if the drainage afforded by free incision be complete resection is unnecessary. It is only indicated when, by approximation of the ribs, the free exit of the pus is hindered (Verebeyli[1]). Opinions are divided as regards the value of irrigation of the pleural cavity. When the pus emits an offensive odor irrigation with a disinfecting solution is imperative. Carbolic acid should, however, not be used. In rare instances accidents arise during irrigation (sudden collapse, convulsions), and I have repeatedly observed a dangerous, and in one instance a fatal, collapse as the result of irrigation in children. The careful insertion of a roll of iodoform gauze is a method to be preferred to irrigation, except when the effusion is stinking. For further details in the operative treatment of empyema the reader is referred to textbooks on surgery. Every effort should be made to favor obliteration of the cavity during post-operative treatment. The indication is to bring about the best possible degree of re-expansion of the compressed lung, and in order to accomplish this the method advised by Ralston James has been practised with great success in the surgical wards of the Johns Hopkins Hospital. The patient daily for a certain length of time, increasing gradually with the increase of his strength, transfers water by air-pressure from one bottle to another. The bottles should be large, holding at least a gallon each, and by an arrangement of tubes, as in the Wolff bottle, an expiratory effort of the patient forces the water from one bottle into the other. In this way expansion of the compressed lung is systematically practised. The abscess-cavity is gradually closed, partly by the falling in of the chest-wall and partly by the expansion of the lung.[2] In long-standing cases, in which the lung cannot expand on account of thick bands of adhesion, the pleural layers cannot be brought into juxtaposition without more or less sinking in of the chest-wall. De Lorme's operation (stripping the pseudo-membrane from the compressed lung) may be advisable. This retraction of the thorax is probably hastened by timely resection of one or more ribs, the amount of bone to be removed depending upon the "expansive power of the lung and elasticity of the thorax."

The duration of empyema is longer than in pleurisy with effusion, and the former affection tends to exhaust to a greater degree the powers of the system than the latter; hence the physician's attention should be directed to the support of the vital forces by all possible agencies, modified to some extent by the special etiology of the case.

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[1] Quoted in *Annual of the Universal Med. Sciences*, 1892, vol. i. sec. A.

[2] Osler's *Text-book of Medicine*, p. 605.

CHRONIC PLEURISY (ADHESIVE PLEURISY).

Definition.—Chronic inflammation of the pleural layers —(*a*) with effusion, and (*b*) without effusion.

(*a*) **Chronic Pleurisy with Effusion.**—This sub-variety may follow acute sero-fibrinous pleurisy, and less frequently it has an insidious development. The morbid lesions, including the character of the exudate, may also be identical with those of the acute or subacute forms of the affection. Fibrin and serum are present in varying relative proportions, the latter, however, as a rule, in preponderating proportion when compared with the composition of the exudate in acute pleurisy. The secondary consequences of copious acute effusions also are met with—*i. e.* displacement of adjacent organs (liver, spleen, heart) and unilateral dilatation of the chest. When the fluid is either absorbed or removed and the case ends in recovery, marked contraction of the affected side results, since the lung, which is covered by thick, organized bands of adhesion, cannot re-expand. *Symptoms.*—But for slight dyspnea upon muscular exercise the subjective symptoms are frequently wanting. The pulse is compressible and accelerated, as a rule, and there is a trifling rise of temperature in the evening hours. If the effusion becomes purulent, hectic fever develops, leading to asthenia, and the latter condition eventually terminates life. Death may also be due to secondary suppurations (abscess of brain, etc.). In most cases occurring in children the effusion early changes to pus. The physical signs do not differ from those in acute sero-fibrinous pleurisy. The *duration* of the cases varies from three months to several years, or intercurrent pulmonary tuberculosis may shorten the course of the affection.

(*b*) **Chronic Dry or Adhesive Pleurisy.**—(1) This may succeed to the acute or chronic sero-fibrinous pleurisy. If the liquid portion of the exudate is absorbed, the pleural membranes come into more or less close apposition, being separated only by fibrinous elements that become organized into a layer of firm connective tissue. Hence the two layers of the pleura, that are greatly thickened, cannot be separated, owing to the firmness of the adhesions. In most cases the autopsy shows the latter condition to be most pronounced at the base, while the lung is found to be compressed and the seat of fibroid change. If it follows the acute form, the extent of retraction is slight, since there are no dense fibrous bands to prevent a fair degree of lung-expansion; if it succeed the chronic form, however, or empyema, the extent of retraction and flattening will be quite marked. The exudate may undergo calcareous degeneration, and occasionally little pouches of fluid may be found between the false bands.

There is a large class of cases that are dry from the onset (*idiopathic dry chronic pleurisy*), and this variety may either be a sequel of acute plastic pleurisy or primarily tuberculous. The condition is very commonly met with at autopsy in subjects who during life had never presented symptoms of pleurisy with effusion. The plastic exudate, however slight, invariably tends to become organized, with resulting fibrinous adhesion of the two layers of the pleura. Most generally the adhesions are circumscribed, and if tuberculous in origin are most frequently apical and often bilateral. Under these circumstances small caseous masses and little tubercles may be found embodied in the somewhat thickened pleura. General synechia is, however, not rare, particularly unilateral.

Symptoms.—Definite rational symptoms are rarely present, and the physical signs lack uniformity or may be entirely negative. In other cases of a mild grade the main characteristics are restrained mobility of the affected side and feebleness of the respiratory murmur. In rarer cases the weakness of the breath-sounds is out of all proportion to the expansive motion of the chest. In still another category—composed of a considerable number of instances—certain physical signs are quite pronounced. Inspection reveals decided contraction, with immobility of the affected side and a compensatory distention of the healthy side. The heart is displaced, and the apex-beat may be missing (*e. g.* when the heart is drawn or pushed behind the sternum, or overlapped by the emphysematous lung). The spinal column is curved, the scapula dislocated, the shoulder ill-shapen and drooping, and the lower part of the thorax shrunken, while the ribs are obliquely placed and closely approximated, or even overlap one another. The tactile fremitus is decreased or absent over the lower portion of the chest, and there is impaired percussion-resonance or dulness over the same area. The breath-sounds on auscultation are exceedingly feeble, and in some instances an occasional dry, leathery, or creaking friction-sound is audible.

Rarely, and particularly if the case be tuberculous, vasomotor symptoms arise in chronic pleurisy, such as unilateral flushing or sweating of the face, or dilatation of the pupil.

Doubtless some of the instances belonging to this affection merge into the pleurogenous type of cirrhosis of the lung, and fatal complicating conditions may arise in connection with the general circulation. Thus I have observed in one instance enlargement followed by dilatation of the right ventricle, and in turn by general dropsy, with fatal result.

Treatment.—In the treatment of this affection two objects must receive especial attention: (1) the removal of any effusion that may be present; and (2) the improvement of the nutrition of the patient. The first indication is presented only by a limited number of the cases, and the rules for meeting it have been stated in the treatment of sero-fibrinous pleurisy and empyema; the second indication is presented by all cases. Careful regulation of the diet is of the utmost importance: it must be generous, with modifications to suit special diatheses (as the gouty or tuberculous), if they be present. Lung-gymnastics are most useful if methodically pursued. The method of Ralston James (previously described) richly deserves a trial in suitable cases. It is to be borne in mind, however, that in old cases efforts at overcoming the lung-pressure will be unsuccessful. Climato-therapy is advantageous for this class of sufferers, particularly if the slightest tendency toward tuberculosis exists; and in my own experience low, mountainous elevations combined with purity of atmosphere have given the best results. Of medicines little need be said. It is especially important to promote the digestive power of the patient to the greatest possible extent. In cases in which the digestive function has been feeble I have observed excellent results from a brief stay at any well-regulated seaside resort or in the country. We may also try, with a probability that the effect will be beneficial, small doses (ʒj—4.0) of cod-liver oil, three times daily after food, or the following formulæ:

℞. Acidi muriat. dil.,	ʒijss (10.0);
Pepsini pur.,	ʒij (8.0);
Tinct. nucis vom.,	ʒiss (6.0);
Glycerini,	ʒiss (48.0);
Aquæ,	q. s. ad ℥ij (64.0).—M.

Sig. ʒj (4.0), well diluted, ten minutes after each meal.

Intercurrent catarrh of the stomach may sooner or later become a troublesome feature, and in combating it lavage is frequently our most effective measure.

PNEUMOTHORAX.

(*Sero-pneumothorax; Pyo-pneumothorax.*)

Definition.—A collection of air in the pleural cavity. Since the latter, as a rule, contains at the same time serum or pus, the terms sero- and pyo-pneumothorax are frequently employed to describe the same condition. It is an uncommon condition.

Pathology.—When the pleural sac is punctured air usually escapes, accompanied sometimes by an audible hissing sound. The pleural sac in pure pneumothorax is greatly distended, and the lung is impacted against the spinal column. Other organs (spleen, heart) are also displaced, owing to positive intrathoracic pressure. The heart is not rotated, however, and the relation of its parts is maintained much as in the normal condition (Osler). The air may occupy but a portion of the pleural cavity, on account of previous firm adhesions (*circumscribed pneumothorax*). The point of perforation, as a rule, can be easily found, and frequently corresponds to the seat of rupture of the tuberculous cavity or superficial caseous mass. In other instances the cause of pneumothorax cannot be discovered. Inflation of the lung under water may reveal the aperture, which is usually small, by the escape of air-bubbles at the seat of puncture. Occasionally a fistulous connection between the pleural sac and the bronchi can be traced.

Simple pneumothorax is, however, of rare occurrence. The air that gains admission into the pleural sac is laden with micro-organisms (*vide* Bacteriology, p. 545), which set up various forms of inflammation, accompanied by equally various exudations. Hence the cavity is usually filled, in part, with an effusion that is purulent or sero-purulent, as a rule, and rarely serous or sero-fibrinous. The *gas* in cases of pneumothorax may be of bacterial origin; this contains substances not found in air, such as H, H_2S, or marsh-gas, and gas-forming organisms (b. coli).

Etiology.—The *predisposing* influences are—(*a*) *age*—the condition occurring in adults as a rule, though instances are also observed in young children; (*b*) *sex*—males suffer more often than females; (*c*) the left side is affected nearly twice as often as the right; (*d*) *emphysema*, in which the superficial air-sacs are dilated and atrophied, rendering the latter liable to rupture from excessive muscular exertion.

The exciting causes are—(1) *Perforation of the lung and pulmonary pleura* (the most frequent cause), arising in one or other of three ways —(*a*) From the rupture of a tuberculous cavity into the pleural cavity. This accident rarely occurs at the apex of the lung, but commonly near the upper border of the lower or middle lobe; and less frequently near the lower border of the upper lobe. A caseous focus immediately beneath the pleura may also, during the process of softening, puncture the pleural sac and invite the entrance of air during the early stages. It cannot occur, however, except in cases in which previous adhesions have failed to form at the point of perforation. At least 70 per cent. of the cases of pneumothorax are tubercular (Morse).[1] (*b*) As the result of necrotic processes, in connection with certain other lung-affections, as gangrene, broncho-pneumonia, suppurating bronchial glands, abscess, and echinococcus cysts. (*c*) From rupture of the normal air-sacs in consequence of severe muscular effort (S. West, DeH. Hall). This accident is sometimes ascribable to the violent paroxysms of cough in pertussis.

(2) Some cases of *empyema*, by perforating the visceral pleura, the lungs, and bronchi.

(3) Perforations of the pleura in *malignant disease* and *abscess of the esophagus.*

(4) A *peripheral bronchiectasis* may open the pleural space and thus establish a communication between it and a bronchus.

(5) Pyo-pneumothorax may be of *subdiaphragmatic origin*, consecutive to *perforation by malignant disease* or *ulcer of the stomach or colon.*

(6) Pneumothorax may be occasioned by *gases* resulting from the action of a gas-forming bacterium on the pleural exudate.

(7) *Wounds* causing direct or indirect perforative lesions of the lungs. Fractures of the ribs may produce laceration of the visceral pleura, and allow the air to enter the pleural sac.

Symptoms.—The earliest symptoms vary according to the cause or causes that produce the condition. When it develops, as it does so often, in the course of pulmonary tuberculosis, the *onset* is sudden, marked by agonizing *pain* in the side, by intense *dyspnea*, and frequently *cyanosis*. The dyspnea is often accompanied by a sense of impending suffocation. The severity of the pain and the degree of oppression depend largely, however, upon the amount of air that gains entrance into the pleural sac or is formed from the exudate, the rapidity with which it enters, and the condition of the pleural cavity as regards the presence or absence of previous pleuritic adhesions. If the orifice be large and valvular, the air cannot escape, but rapidly accumulates and forces all the air out of the lung by compression; the patient then sinks rapidly into collapse from shock, and sudden death ensues. Fortunately, the *open* form is commoner, especially in non-tuberculous pulmonary affections. The *respirations* are frequent; the *pulse* is also frequent and feeble, sometimes reduced to a thread; and cold sweats are not uncommon. The *temperature* at first is apt to fall one or two degrees below the normal, owing to sudden collapse; *fever*, however, follows almost invariably, and frequently is of the *hectic* type. Its cause is pleuritis, often of a purulent form, and if this be the case, the dyspnea may be due in part to the increasing effusion. The patient now also suffers from the grave symptoms of empyema above described. Edema of the hand of the affected side is sometimes present as an early manifestion; it rapidly disappears (Weil). When pneumothorax develops in the last stages of phthisis acute symptoms may be entirely absent.

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[1] *American Journal Medical Sciences,* May, 1900.

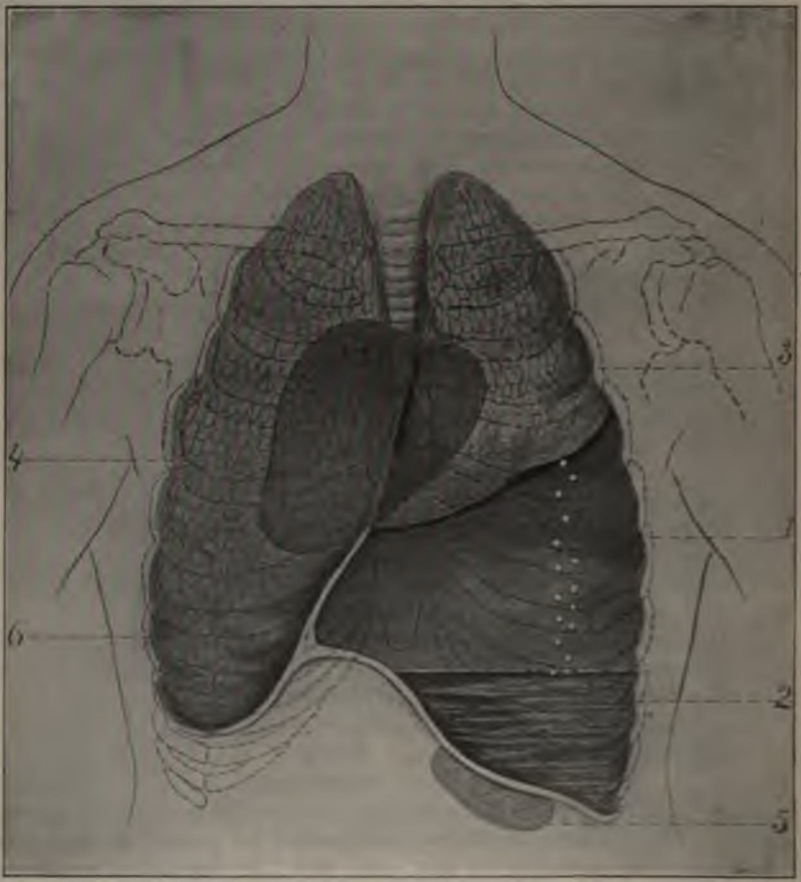

FIG. 46.—1. Air in the pleural sac; 2, fluid exudate at base of pleural sac; 3, compressed portion of lung; 4, displaced heart; 5, depressed spleen; 6, mediastinum pushed toward the right.

Physical Signs.—These are marked (see Fig. 46). *Inspection* shows marked distention and immobility of the affected side; also some degree of distention with unnatural mobility of the healthy side.

Palpation shows the tactile fremitus to be diminished above and greatly diminished or wholly absent over the effusion below. Edema of the chest-wall can frequently be made out. The impulse-beat of the heart is found to be feeble and displaced.

On *percussion* a deep and full, or modified tympanitic note (*bell-tympany*) can usually be elicited over the area corresponding to the contained air, and the excessive tension in the pleural sac, due to the enormous amount of air it contains, may cause an elevation in the pitch of the note even to dulness. The "cracked-pot" sound is audible when the air in the pleural cavity freely communicates with the external air. Wintrich's sign, or a change in the pitch of the percussion-sound when the mouth is open or closed (being lowered when the mouth is closed and raised when open), may also be observed. In pyo-pneumothorax a flat note is elicited from the base upward as far as the fluid extends, and there is a more marked temporary change in the upper level of flatness than in pleurisy with change of posture. Modifications in the pitch of the percussion-sound result from an alteration in the form as well as in the dimensions of the air-space. Owing to displacement of the heart, there is, as a rule, resonance over the normal cardiac region, and particularly when the patient assumes a recumbent posture. The liver and spleen, according to the side affected, are displaced downward to a greater degree than in simple pleuritic exudates.

Auscultation discloses a greatly weakened or altogether suppressed respiratory murmur when collapse of the lung is incomplete. Amphoric breathing is audible in cases of *open* pneumothorax, and bronchial râles possessing a metallic quality are sometimes heard, as well as metallic tinkling on deep inspiration or on coughing. The metallic tinkling is caused frequently by drops of fluid falling from above upon the surface of the effusion; less frequently by a re-echoing of vibrations of moist bronchial râles communicated to the air in the pleural chamber. The vocal resonance is enfeebled, as a rule, and evinces the same metallic quality. The so-called coin-test is a pathognomonic sign, and is elicited in the following manner: An assistant places one coin on the front of the chest and taps it with another while the ear of the examiner is placed on the thorax posteriorly, where will be heard the intensified echo of the coin-sound thus produced. Another most characteristic sign is the so-called Hippocratic succussion, which is elicited by placing one ear upon the patient's chest while the latter's body is shaken, and a distinct splashing sound is heard.

Diagnosis.—When the attack is of ordinary severity, pneumothorax is diagnosticated by the history of one or other of the causal factors, together with certain physical signs that do not belong to any other affection (*coin-sound, succussion-splash*). It is only when the air and fluid in the pleural sac are encapsulated that it may become difficult to eliminate (*a*) a large pulmonary cavity; (*b*) excessive gaseous distention of the stomach; (*c*) an abscess below the diaphragm into which air has entered (*pyo-pneumothorax subphrenicus*); (*d*) a diaphragmatic hernia; (*e*) emphysema; and (*f*) pleurisy with effusion.

(*a*) *A Large Pulmonary Cavity.*—The "cracked-pot sound" and Wintrich's sign are more frequent in cavity than in pneumothorax, and the former condition does not tend to dislocate the adjacent organs. There is no response to the coin test and an absence of the succussion-ment are more numerous than is generally indicated by writers upon the subject, and not infrequently is hydrothorax symptomatic of either chronic dysentery, chronic diarrhea, leukemia, pernicious anemia, carcinoma, malaria, syphilis, or scurvy. Strictly local causes may also induce it, as carcinoma of the pleura, or the compression of the superior vena cava or of the thoracic duct by a tumor.

Symptoms.—The *subjective symptoms* are attributed to the mechanical effects of the fluid, and the causal affection may have symptoms quite in common; these are *dyspnea* (often culminating in orthopnea), *cyanosis*, *asthmatic seizures*, *irritative cough*, and a *feeble circulation*. The *general symptoms* arise from the primary affection.

Physical Signs.—The physical signs are much the same as in pleurisy with effusion—with this difference, that they are more often bilaterally distributed. Hydrothorax is often unilateral, however, and an enlarged right auricle may be the cause of this condition in some instances. The right side is the one usually affected. I have also observed that quite frequently the two sides of the chest exhibit great variations as to the relative amount of fluid contained.[1]

Prognosis.—This depends upon the nature of the primary disorder that causes the dropsical transudation.

Treatment.—The treatment of hydrothorax has intimate relations with the indications presented by the underlying affection. If the measures directed toward the removal of the general dropsy (*anasarca*), of which hydrothorax is a part, are unsuccessful, and the amount of transudation in the pleural sac interferes with the functions of the heart and lungs, then aspiration must not be too long delayed, and must be repeated as often as occasion demands.

NEW GROWTHS OF THE PLEURA.

ALMOST all instances of new growths developing in the pleura are secondary to primary carcinoma of the lung, the pleura being invaded by the direct extension of the neoplasm. It may also arise by metastasis from carcinoma of the lung, mammary glands, etc. The pleura presents circumscribed areas of thickening, or the growth takes the form of papular projections from its surface, and as these enlarge they become pedunculated. Their size varies from that of a pea to that of an orange. The adjacent pleura is inflamed, often adherent, and much thickened, and an effusion into the pleural cavity is often observed.

Primary carcinoma of the pleura is very rare indeed, and E. Wagner, who first described it, called it endothelial carcinoma. Most pathologists of to-day, however, look upon endothelioma as a variety of *sarcoma*. It owes its orgin to a proliferation of the endothelial cells of the connective tissue and the lymph-apparatus of the pleura. This invariably assumes the diffuse form, and by metastasis we have involvement of the other organs (lungs, lymphatics, liver).

Spindle-cell sarcoma of the pleura, as well as the round-cell variety, is occasionally met with.

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[1] For the differential diagnosis between pleurisy and hydrothorax see Pleurisy, p. 558.

Prognosis.—This depends largely upon the cause. The cases attributed to advanced phthisis usually reach a fatal issue in the course of one, two, or more weeks, and rarely they run a very rapid and fatal course. On the other hand, the pulmonary condition is at times favorably influenced by its occurrence. Following empyema, or when due to trauma or abscess of lung, pneumothorax sometimes takes a favorable course. It is fraught with especial danger when it is the resultant condition of some acute lung-disease (gangrene, broncho-pneumonia).

Treatment.—The leading indication is the alleviation of the patient's sufferings by a prompt resort to morphin, and it often becomes necessary to administer it hypodermically. If the patient's previous strength has been moderately good, the question of operative intervention should be seriously considered, the nature of the surgical procedure then depending upon the character of the effusion. If this be sero-fibrinous, aspiration, as in simple pleurisy, must be performed to relieve the urgent dyspnea; if purulent, permanent drainage should be procured for the same indication. A costal resection may be advisable. When pneumothorax develops late in phthisis radical measures are not to be thought of, and the physician must rely upon aspiration (when necessary) to relieve urgent symptoms. We may also tap the air-chamber above the fluid with a fine needle, with a view to lessening the excessive tension. Unverricht has recently reported good results from a somewhat novel mode of treatment. When there is a pulmonary fistula present, he inserts a tube into the pleural sac. This allows free entrance of air, the lung collapses completely, and the fistula has a chance to heal. For the dyspnea, atropin administered hypodermically is valuable; for the feeble cardiac action, alcoholic stimulants, aromatic spirits of ammonia, strychnin, ether, and other cardiac stimulants should be employed. Locally, cutaneous irritants may be applied (turpentine stupes, mustard pastes).

HYDROTHORAX.

(*Dropsy of the Pleura; Thoracic Dropsy*).

Definition.—A collection of transuded serum in the pleural cavity.

Pathology.—Hydrothorax is generally a bilateral condition. The transudate is a clear, amber-colored liquid that is free from fibrin, but may contain cholesterin and a few endothelial cells. It has an alkaline reaction, a comparatively low specific gravity (1009 to 1012), and is non-inflammatory. The pleural surfaces are usually smooth, though sometimes decidedly pale and edematous. The mechanical effects of hydrothorax upon the lungs and other thoracic and abdominal viscera are similar to those of the exudates that accompany inflammation of the pleura, though they are rarely so marked as in sero-fibrinous pleurisy.

Etiology.—Hydrothorax is a secondary affection, and is usually connected with one or other of the various forms of general dropsy (hemic, renal, cardiac). The cases that are due to blood-impoverish-

Abscess of the Mediastinum.—This is of rare occurrence, its most frequent seat being the anterior mediastinum. Of the commoner causes may be mentioned traumatism and the infectious diseases—erysipelas, rheumatism, measles, and small-pox in particular. It may also be the result of an extension of a suppurative process from neighboring structures. Pulmonary tuberculosis is the most potent factor in producing chronic abscess in this situation.

Symptoms.—*Acute Abscess.*—Pain and tenderness in the sternum are the most prominent features, the pain being acute and often of a throbbing character. Cough and dyspnea are usually present. The general features are fever, frequently accompanied by rigors and profuse sweats and considerable physical prostration. The chief physical sign is dulness upon *percussion*, usually found anteriorly and increasing gradually with the development of the abscess. Later, the tumor may reach the surface of the body, and rarely the sternum is eroded. *Palpation* now detects pulsation and fluctuation. The abscess may either find its way downward into the abdomen, or it may perforate the trachea or the esophagus.

In *chronic abscess* the symptoms bear a closer similarity to those of solid tumors than those in the acute form. Fortunately, chronic abscess quite often results in spontaneous cure, in which case it is in part absorbed, and the remainder of its contents become inspissated. In obscure cases an exploratory puncture with a small needle may be safely practised, and with definite results, as a rule.

Diagnosis.—*Acute* abscess must be differentiated from solid mediastinal tumors and aneurysm. The more acute onset and general symptoms of the suppurative process (hectic type of fever, chills, sweats) and the more rapid course will serve to distinguish abscess from *aneurysm* on the one hand, and *solid tumors* on the other. Further, the absence of strong expansile pulsation, diastolic shock, and the aneurysmal bruit aid materially in eliminating *aneurysm of the arch.*

The **treatment** is mainly surgical.

(*b*) **Tumors of the Mediastinum.**—Two forms only demand practical consideration—carcinoma and sarcoma. Hare's analysis of 520 cases gave the following ratio: of carcinoma, 134; sarcoma, 98; lymphoma, 21; fibroma, 7; dermoid cyst, 11; hydatid cyst, 8; and fewer cases of ecchondroma, lipoma, and gumma. In 48 of the cases of carcinoma and in 33 of sarcoma the tumor occupied only the anterior mediastinum. It is quite probable, however, that sarcoma, and not carcinoma, is the commoner neoplasm of this region. The clinical term "cancer" was formerly used promiscuously by many authors, and the pathologic diagnosis was then difficult, so that statistics are scarcely trustworthy. Upon investigating 25 of the older reports of "cancer," Pepper and Stengel found in 13 unquestionable evidence that the growth was sarcoma, while in the remaining 12 they could not, for the greater part, decide to which form the disease belonged. Primary sarcoma may spring from the remnant of the thymus gland, from the lymphatic glands, the pleura, or lungs, or from the fibrous tissues of the mediastinum. Primary carcinoma may originate in the esophagus, bronchi, lungs, or rarely in the thymus gland. Secondary mediastinal tumors are most apt to have their seat in the lymphatic glands. Carcinoma is less frequently primary than sarcoma. Among *predisposing causes* are sex and age—males being more prone to the affection than females, and the period of chief liability is between the thirtieth and fortieth years.

Symptoms.—The earlier symptoms are vague (slight substernal pains, dyspnea, general languor). Later, pressure-symptoms gradually supervene.

The *pain* may or may not be severe, but is invariably accompanied by a feeling of oppression. Its chief seat is in the upper sternal region, but it may radiate to the sides of the chest and even down the arms (in which case it is due to pressure on the brachial plexus). *Dyspnea* appears early, is constant, and may become intense. It is caused by pressure either upon the trachea, upon a primary bronchus, or upon a recurrent laryngeal nerve. *Asthmatic seizures* may occur before there is constant dyspnea and before the tumor has reached notable size within the chest. There is *cough*, which may be paroxysmal and of a brazen character. Aphonia may be present. There may be *dysphagia* from pressure upon the esophagus, though this is rare. If there is an inflammation of the vagus or sympathetic nerve, the rate of the pulse may be either slowed or markedly quickened. Owing to implication of the sympathetic there may be local hyperemias and pupillary inequalities.

Compression of the superior vena cava or of the subclavian vein may be followed by cyanosis and edema of the parts drained by these vessels, and the early occurrence of venous occlusion and marked dilatation of the superficial veins is quite characteristic. Collateral circulation may be rarely established. Less frequently the inferior cava may also be pressed upon.

Physical Signs.—*Inspection.*—In advanced cases a swelling, usually somewhat irregular and often diffuse, appears in the sternal region. The tumor may cause erosion of the sternum, and a little later occupy a position immediately beneath the skin, Osler[1] being of the opinion that the rapidly-growing lymphoid tumors, more commonly than others, perforate the chest-wall. I saw a case in which the perforation occurred at the right edge of the sternum, precisely at the point at which aneurysms of the ascending arch most frequently appear. In the early stages, however, this prominence is not present. *Palpation.*—When a tumor is present it may pulsate distinctly, and the heart's apical impulse may be detected in various abnormal positions. Tactile fremitus is absent over the seat of the growth if the latter be in contact with the chest-wall.

On *percussion* dulness is noted, and this is true even in many instances that do not present a visible swelling. The dull area varies in outline with the size and position of the tumor. *Auscultation* usually reveals no sounds over the dull area, except a bruit in rare instances. The heart-sounds are inaudible over the tumor-site as a rule, and the breath-sounds and vocal resonance are feeble or absent. To the above physical signs are frequently added those of pleural effusion.

The **diagnosis** of mediastinal growths is made, if at all, principally by exclusion.

Aneurysm is differentiated from solid mediastinal tumors with only slight success in many instances. It is most valuable to note carefully the length of time the condition has lasted, since aneurysm runs a longer course, on the average, than mediastinal tumor. The tumor when due to aneurysm communicates a strong, heaving, expansile pulsation—a characteristic that is absent or only feebly manifested in the case of solid mediastinal growths. The severe diastolic shock, as noted on both palpation and auscultation in cases of aneurysm, is also absent in solid tumor. Kassabian has shown that new growths can be early recognized by an x-ray examination. On the other hand, shadows situated in the anterior portion of the chest and to the right of the median line are generally produced by aneurysms.

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[1] *Practice of Medicine*, p. 579.

The *duration* of the disease varies from six to eighteen months.

The **prognosis** is absolutely hopeless, except in the case of benign tumors, which may be removed in some instances.

The **treatment** is directed toward the relief of the most urgent symptoms. Anodynes are required sooner or later, and should not be withheld if indicated. As a routine the preparations of iodin and mercury are employed; but, as these are useless, they are unwarranted. Arsenic has sometimes seemed to influence sarcomatous and lymphadenomatous growths favorably, though only temporarily.

(*c*) **Diseases of the Thymus Gland.**—Nothing is known definitely concerning the functions of the thymus gland. Tumors may have their origin in the thymus gland, and the organ may become enlarged (hypertrophy, abscess); these conditions are indistinguishable from and associated with mediastinal tumor or abscess as above described.

Jacobi believes that fatal cases of laryngismus stridulus may be rarely ascribable to an enlargement of the thymus gland. C. H. Hunter[1] reports two cases of primary fatal laryngeal stenosis occurring in the same family in children aged 19 and 7 months respectively. The question as to the existence of the so-called asthma thymicum, however, is still *sub judice*, although the number of sudden deaths in young children reported in connection with various forms of enlargement of the thymus gland is steadily increasing. The dyspnea, generally fatal, that has been found to be caused by an enlarged thymus is scarcely the result of direct pressure on the trachea. Arnold Paltauf attributes sudden death in cases in which the thymus was found enlarged to hyperplasia of the entire lymphatic system (*constitutio lymphatica*). Ohlmacher[2] noted in 18 cases of epilepsy a large and apparently functionally-active thymus gland. There was hyperplasia of the lymph-glands throughout the body and of the lymph-follicles of the mucous surfaces.

Persons who manifest the hemorrhagic diathesis, or those who suffer from hemorrhagic affections, may also show hemorrhage into the thymus gland—a condition that is identical with that produced by hemorrhage into the mediastinum. There is no treatment for enlargement of the thymus, although Siegel in one case, a boy of 2½ years, elevated and stitched the thymus to the fascia over the sternum, with the result that threatening dyspnea disappeared and the child eventually made a good recovery.

(*d*) **Mediastinal Hemorrhage.**—This term signifies hemorrhage into the mediastinal connective tissue. It oftenest results from the rupture of aneurysms of the arch or of the large vessels within the thorax, or it may be of traumatic origin (wounds, fractures).

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[1] British *Med. Jour.*, April 2, 1898.

[2] Phila. *Med. Jour.*, Jan. 1, 1898; Saunders' *Year-Book* for 1899.

coma. Among *predisposing causes* are sex and age—males being more prone to the affection than females, and the period of chief liability is between the thirtieth and fortieth years.

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The **diagnosis** of mediastinal growths is made, if at all, principally by exclusion.

Aneurysm is differentiated from solid mediastinal tumors with only slight success in many instances. It is most valuable to note carefully the length of time the condition has lasted, since aneurysm runs a invariably present, the amount of serous effusion, as the term would indicate, is never large in dry or plastic pericarditis. Myocarditis may frequently be found as an associated condition.

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[1] *Practice of Medicine*, p. 579.

Etiology.—In each variety of pericarditis there are special contributing factors, so that it is desirable to give its etiology separately, except in the sero-fibrinous and acute plastic types, which have practically the same etiology. The two latter are the more common forms of the disease. Acute plastic pericarditis most frequently occurs in young and middle-aged males. It may be *primary* or *secondary*. It often occurs in acute articular rheumatism (in more than one-half the cases), chronic nephritis, and, rarely, in other acute infectious diseases. In this form the infective agents are transmitted to the pericardium by means of the circulation. It may be caused also by direct extension of inflammation from adjacent structures (secondary pericarditis), as in simple pleurisy; more commonly the extension occurs from a pneumonia or tuberculous pleurisy, or the condition may complicate new growths and inflammatory conditions affecting the esophagus and bronchial glands. It may also be secondary to chronic disease of the aortic valve, the pericardium becoming involved by extension through the walls of the aorta. Finally, it may be the result of traumatism, and this may cause any of the other forms of pericarditis.

Clinical History.—Owing to the fact that acute plastic pericarditis is usually a secondary affection, the symptoms that enable one to recognize it are obscured by the disease of which it is a sequel. This is particularly true of that large class of cases that develop in acute articular rheumatism, in which subjective symptoms are often entirely wanting. It is only in the severest types of this sort that the symptoms referable to the heart are well enough marked to arrest the attention. There may be a feeling of *distress* or *constriction* with or without slight *pain* in the precordium. During the first stage or prior to the pouring out of the effusion the pain is most marked, extending sometimes into the left arm or the back, and at others to the ensiform cartilage or even to the abdomen. This pain is, rarely, increased by pressure over the pericardium. *Palpitation* and *dyspnea* may be present, and the *pulse* is increased in frequency and strength, as a rule, except in the later period, when it may be weak and slightly irregular, particularly if the muscular tissue of the heart be involved. There is some *fever*, but the degree of elevation of temperature perhaps never exceeds 102° F. (38.8° C.). In this class of cases the *urinary features* depend largely upon the character of the leading etiologic factors; though in many instances the urine is scanty, high-colored, and acid in reaction.

Physical Signs.—*Inspection* discloses increased vigor of the apex-beat. Friction-fremitus (due to rubbing of the altered pericardial layers upon one another) may sometimes be felt during the earlier and later courses of the disease or when the membrane is comparatively dry, and is usually most intense near the base, just to the left of the sternum. *Percussion* gives negative results. *Auscultation* usually reveals a double friction-sound, sometimes quadruple (locomotive murmur)—a characteristic sign, though one on which sole reliance must not be placed in this disease. The friction-rub is caused partly by the exudate and partly by the dry state of the membrane. Its usual seat of maximum pronunciation

PART V.

DISEASES OF THE CIRCULATORY SYSTEM.

I. DISEASES OF THE PERICARDIUM.

PERICARDITIS.

Definition.—An inflammation of the serous covering of the heart.

Varieties.—(*a*) Plastic, or fibrinous; (*b*) sero-fibrinous, or subacute; (*c*) purulent; (*d*) hemorrhagic; (*e*) adhesive. There is also a tuberculous pericarditis which has been described (*vide* Tuberculosis, page 311).

Bacteriology.—Rudini's experiments have shown that the staphylococcus aureus may be a cause of pericarditis; but they have not conclusively demonstrated that it is the specific cause, as is evidenced by the fact that the disease is sometimes caused by other organisms. Moreover, staphylococci have not been encountered without demonstrable cause. Among other organisms, the pneumococcus, streptococcus, the bacillus coli, the tubercle bacillus, and probably also a variety of the bacillus pyocyaneus and the gonococcus may be named. Micro-organisms are not always found in pericarditic exudates.

ACUTE PLASTIC OR FIBRINOUS PERICARDITIS.

Pathology.—The morbid changes are frequently localized, and less frequently are general. At the onset the membrane is smooth, swollen, and injected, and punctured ecchymotic spots may be visible; soon it presents a grayish, roughened appearance in consequence of a deposition of a thin layer of fibrin. In the severer types the fibrinous deposit increases in thickness for a time, and the natural movements of the pericardial surfaces upon one another sometimes cause the exudate to assume a honeycombed appearance. Most examples that I have seen, however, have resembled the roughened surfaces produced by separating two slices of bread that had been thickly buttered; the surfaces are grayish-yellow in color. In the later stages the exudation becomes partly organized, and as the result of friction produced between the opposed surfaces by the incessant action of the heart, the pericardial surface may present a villous appearance; hence the term "hairy heart" which was employed by ancient authors. For like reasons we may see the exudate arranged in the form of little ridges, forming a "tripe-like membrane." Though the limits of the precordia. The sitting posture, leaning forward, or moderate pressure with the stethoscope, all fail to produce or to increase *endocardial murmurs*, whether acute or chronic. A double aortic murmur is associated with cardiac hypertrophy, the Corrigan pulse, and systolic flushing of the capillaries.

Prognosis.—The termination is always favorable as to life. Complete resolution does not often occur, but the exudate becomes connective tissue, and agglutinates the two layers of the pericardial sac. The acute may merge into the chronic form, and dry, plastic pericarditis often constitutes the first stage of severer grades of the disease.

Treatment.—Absolute quiet in the recumbent position should be enjoined. The diet should be composed chiefly of light, easily digested solids, allowing as little drink as is possible, and thus endeavoring to avoid an overfilling of the vessels. With the same object in view, if the patient's strength be good, a half-dozen leeches should be applied over the heart, followed by the use of the ice-bag; the bowels are to be kept soluble by using stewed fruits or saline laxatives if needful. Calomel in doses ranging from gr. $\frac{1}{4}$ to $\frac{1}{2}$ (0.016–0.032) every hour or two, combined with a little opium to prevent purgation, is serviceable. At the beginning veratrum viride may also be cautiously administered, with a view to dilating the arterioles throughout the rest of the body, and thus virtually " bleeding the patient into his own vessels." Later, digitalis in combination with the iodids of potassium and iron should be substituted for the purpose of absorbing the effused material. Tonics and a change of air may be required during convalescence.

SERO-FIBRINOUS PERICARDITIS.

Pathology.—The anatomic changes may be grouped into three stages—the *first* being characterized by a plastic exudation (corresponding with the lesions in dry, plastic pericarditis, though more pronounced); the *second stage*, by a variable amount of effusion composed largely of serum. The exudation usually begins about the origin of the great vessels springing from the base of the heart, and ultimately forms a thick covering of fibrin, especially on the visceral layer. The quantity of serous effusion may be from 2 to 10 ounces (64.0–320.0), but occasionally it is as much as 3 pints ($1\frac{1}{2}$ liters). The admixture of a slight amount of blood- or pus-corpuscles sometimes occurs in this form of the complaint. The *third* is the stage of absorption in the most favorable cases. Perfect resolution rarely takes place, but, instead, the liquid effusion is alone absorbed, and the lymph causes firm adhesions of the visceral and parietal membranes. If, as sometimes happens, the serum remains, the acute passes into a chronic condition. The muscular tissue of the heart may become involved by an extension of inflammation from the visceral layer which lies in contact with it; it is always the seat of more or less collateral edema. The grade of the myocardial inflammation will depend much upon the extent and duration of the pericarditis, though usually it is moderate in the fibrino-serous variety.

Etiology.—The disease is most frequently observed to be associated with acute rheumatism, Bright's disease, and pulmonary tuberculosis. Sears collected 100 cases of pericarditis, of which 51 were due to acute rheumatism; and, according to Baumgarten, the former disease arises as a complication of the latter in about one-third of the cases. I believe that exceptionally both serofibrinous and plastic pericarditis may occur in the course of rheumatic dyscrasia without the slightest evidence of arthritis. The disease also occurs in the course of the eruptive fevers and lobar pneumonia, and from extension of inflammation from neighboring parts. Of 66 instances of pericarditis in children, 24 were caused by rheumatism. Next in frequency were tuberculosis and pleuro-pneumonia (Baginsky). (See also Bacteriology, p. 579.)

Clinical History.—When, as rarely occurs, a *primary* pericarditis develops, the initial symptoms common to inflammation of other serous membranes manifest themselves, as *anorexia*, sometimes *nausea and vomiting*, *chills*, *fever*, *increased respiration* and *pulse-rate*, together with local *pain*. The pain is usually of a dull, aching character, and less frequently merely a slight soreness, or it may be absent altogether. Acute pain is experienced only when the pleura is implicated.

When pericarditis is *secondary* there are, in many cases, no subjective symptoms to indicate its presence. In other instances there may be *precordial oppression* with or without slight pain or a feeling of soreness. Hence the rule should be absolute that in all affections in which pericarditis is likely to arise physical examinations of the heart should be frequently made, and particularly during the height of the disease.

Dyspnea comes on simultaneously with the appearance of the effusion and may lead to actual orthopnea. Pressure is exerted upon the left lung if the effusion be large—a fact that explains in part the presence of dyspnea. The cardiac muscle, especially the right ventricle, is also pressed upon by the effusion, thus impeding to a greater or lesser extent the cardio-pulmonary circulation as well as the cardiac diastole. We have here an additional reason why dyspnea occurs, and also why deficient aëration of the blood and a feeble peripheral circulation are found in this complaint. Prior to the occurrence of the effusion the circulation is too actively carried on, the *pulse* being full and strong. It is clear from the above explanation relative to the mechanical effects of large effusions that during the second stage the pulse is small, feeble, and irregular. When the liquid effusion is not large the heart-action may be apparently feeble, while the pulse remains strong—a valuable rational sign. On the other hand, an excessive amount of fluid may cause the radial pulse to become quite small or even to disappear during inspiration (the *pulsus paradoxus*). *Fever* is present, as a rule; the temperature is irregularly elevated, ranging from 101° to 103° F. (38.3°–39.4° C.). In favorable cases defervescence takes place by lysis. *Nervous symptoms*, as headache and mild delirium, often appear, and sometimes give place to stupor or even coma.

Physical Signs.—*Inspection.*—The skin-surface and mucous membranes are observed to be pale and more or less cyanotic. The neck-veins are prominent, and sometimes exhibit undulatory movements or pulsations. The face wears an anxious expression; the respirations are increased, labored, and at times irregular. The decubitus is dorsal; the head and shoulders are elevated, and the patient may be forced to assume the sitting posture. In young subjects precordial prominence, with effacement or even bulging of the intercostal spaces, may result from the presence of a moderate effusion. In adults, however, a large collection is indispensable for the production of this effect. If the lung be shrunken or if there are pleuritic adhesions, expansion of the pericardium, and hence also bulging, will be prevented. The distended pericardium may depress the diaphragm. Elevation of the left nipple in consequence of marked anterior expansive bulging has been observed. In the first stage the apical beat is exaggerated, but as the effusion increases (forcing the heart backward and upward) it is displaced in an upward and outward direction, at the same time becoming weaker as well as more diffused, since with expansion of the sac comes greater mobility of the organ. When the pericardial sac becomes filled the impulse-beat disappears, for the reason that the fluid now completely surrounds the heart and pushes it away from the chest-wall.

Palpation confirms the results of inspection. The apical beat is diffused and feeble or lost. When detectable it is found to be displaced upward and to the left. Altering the patient's posture changes the seat of the apex-beat (Oppolzer), and if the shock has been lost, turning the patient on his left side or bending his body forward may cause its return. The cardiac impulse disappears earlier when, on account of myocarditis, the systole is greatly enfeebled. On the other hand, old adhesions and marked hypertrophy of the heart, may retain the apex-beat in contact with the chest-wall, despite the presence of a large accumulation. A friction-rub can be felt occasionally over the base of the heart even in the stage of effusion, and, if absorption takes place, the friction fremitus becomes more marked. Fluctuation is rarely detected. In large effusions the liver is *depressed and easily palpable.*

Percussion.—The area of cardiac dulness is greatly increased, and assumes a characteristic triangular outline with the base downward and the apex extending up to the third or even second interspace to the left of, though near, the sternum. The lateral border-lines of dulness obviously diverge from above downward, the right passing to a point corresponding with the right edge of the sternum, along which it runs to the seventh rib; the other to the left, finally intersecting the base-line at the left anterior axillary line. Flatness may be met in the axillary region, even obliterating Traube's semilunar space. The lower level of the fluid, being continuous with the liver dulness, is not definable. Rotch points out that even in moderate effusions there is flatness in the fifth interspace to the right of the sternum (*cardio-hepatic triangle*—Ebstein) in large pericardial effusions. The margins of the lungs surrounding the heart may be retracted and the heart carried forward or dilated, owing to the presence of adhesions; the dull space will then appear larger than is justified by the amount of fluid. Retraction or moderate compression of the lung may, however, give rise to a modified tympanitic resonance to the left of the flat area. Occasionally the lung is attached anteriorly, and the heart is crowded backward by the effusion, while the area of flatness on percussion is relatively diminished. The triangular shape of the flat space, noted when the patient is in the sitting posture, is to a considerable extent lost and its area diminished when he changes to the supine position or lies on either side, the effusion obeying the laws of gravitation. The feeble impulse can be at times felt within the dull area and not at its boundary.

Auscultation.—The characteristic friction-rub of the first stage has already been described. It may, however, also be audible over the base during the stage of effusion, and always returns, after absorption of the fluid, for a brief period. The heart-sounds grow more and more distant, faint, and muffled, though the second sound, as heard over the extreme base of the organ, may remain clear. Over the area of dull tympany corresponding to the lower antero-lateral portion of the left lung (which, as before pointed out, is more or less compressed) may be heard broncho-vesicular breathing.

Course and Duration.—It will appear obvious that the course must vary in individual cases with the cause and severity of the special type of infection. Observation has shown that in one class of cases the three stages (dry, effusion, and absorption) are passed through in rapid succession, while in another class each stage is proportionately lengthened. The latter form has been termed "chronic" by some and "subacute" by others. The *acute* may also be followed by the *chronic* variety. Usually sero-fibrinous effusions complicating rheumatism are absorbed with rapidity once the process has begun, seldom requiring more than two weeks. When recovery is about to occur the temperature falls by *lysis;* the dyspnea gradually disappears, and with it the effusion is gradually absorbed. Convalescence is further indicated by a return of the appetite, normal heat of the skin, and a less frequent, full, and regular pulse. In cases that tend to a fatal termination either the fever continues or there is suddenly developed *hyperpyrexia*, as may happen when pericarditis occurs in the course of acute rheumatism; in such cases the dyspnea is urgent and cyanosis is often marked, with signs of failing circulation. *Nervous symptoms*, as extreme restlessness, insomnia, and active delirium, may be present. Under these circumstances death usually ensues at the end of a week or ten days. In a fatal case of *acute articular rheumatism* that I saw, complicated by pericarditis, with hyperpyrexia, death occurred on the sixth day.

Complications.—Copious effusion may, by causing pressure upon the recurrent laryngeal nerve, produce paralysis of the vocal apparatus, or, it may press upon the esophagus, and cause difficult deglutition. Rarely *acute pleuritis* with effusion is a complication, and its occurrence usually lengthens the course of the pericarditis and renders the outcome uncertain. When there coexists *extensive myocarditis* syncopal attacks often endanger the life of the patient. Associated endocarditis and a complicating pneumonia may be observed.

Prognosis.—In sero-fibrinous pericarditis recovery is the rule under favorable conditions. The outlook, however, becomes gloomy when the above-mentioned complications arise, and particularly when there is hyperpyrexia in connection with acute rheumatism. Occurring as a secondary event in serious acute diseases, as pneumonia, or in chronic diseases, as Bright's, or organic affections of the heart, the pericarditis often precipitates a fatal termination. The strong possibility that these cases may only partially recover or assume a chronic form must be recollected in making a prognosis.

Diagnosis.—The disease is often overlooked, because unsuspected. Ordinarily the recognition of pericarditis by the characteristic triangular area of percussion-dulness and by the friction-sound is not difficult. Atypical cases or those first seen during the stage of effusion can only be correctly diagnosticated by exclusion.

Differential Diagnosis.—*Acute pleurisy* of the left side may simulate pericarditis with copious effusion, and, as before stated, these diseases may coexist. Acute pain, however, belongs to pleurisy alone. In pericarditis the characteristic physical signs are elicited over the precordia; in pleurisy they are apt to occupy not only the anterior but also the axillary and posterior aspects of the chest; hence the percussion-flatness in pleurisy extends to the left, far beyond the boundary-line of the percussion-flatness in pericarditis. The pericardial friction-sound has a different situation usually from the pleuritic, and the latter is heard synchronously with the respiratory movements, while the former is intimately related to the time of the cardiac movements. The friction-murmur of pleurisy ceases if the breathing be momentarily suspended. *Encapsulated pleural effusions* that are limited in area to the antero-lateral portion of the chest are exceedingly difficult of elimination, and especially in the absence of pleuritic friction. In the latter complaint, however, the heart-sounds are clear and the apex-beat often pushed some little distance to the right; on the other hand, in pericarditis the general disturbance is usually greater, while a friction-rub may be detectable over the base. The heart-sounds are distant and muffled. The diagnosis is often aided by a consideration of the previous history and the bearing of any facts thus obtained upon the known etiology of these affections. We encounter formidable difficulties when we attempt to exclude *cardiac dilatation*, though the following brief table will be of assistance in the diagnosis:

Pericarditis with Effusion.	Cardiac Dilatation.
(Previous History.)	
Recent history of gout, acute rheumatism, acute infectious or septic disease, scurvy, chronic nephritis, or tuberculosis.	Usual history of chronic valvular disease of the heart.
(Clinical History.)	
Fever and slight pain are usually associated.	No fever or pain, as a rule.
Nervous symptoms are often present.	Absent.
(Physical Signs.)	
Inspection often reveals bulging (more marked in the young). Apex-beat pushed up, is feeble, and later absent.	Apex-beat usually visible, wavy, and diffused.
Heart's impulse usually absent or occupies center of dull area. Friction-fremitus may be present over the base.	Though feeble, the impulse is palpable.
Percussion shows a triangular flat area, and the boundary-line above changes on altering the position. There is dull tympany (flatness in massive exudations) in the axillary region.	Dull area varies with chambers dilated; it is coextensive with a wavy impulse, does not extend so high (except in mitral stenosis), and does not vary with change of position. No dull tympany.
Auscultation shows the first sound distant and muffled; a double friction-rub is often present over the base.	First sound clear, short, and sharp. No friction-murmur present, but an endocardial murmur may appear.
X-ray shows triangular, movable shadow.	Upper level of shadow (quadrangular) fixed.

Treatment.—The management of the first (or dry) stage is identical with that detailed in discussing the plastic variety. During the stage of effusion the patient should be kept at absolute rest in the recumbent posture, and mental excitants should be rigidly prohibited with a view to minimizing the labor of the heart. The *diet* is to consist mainly of easily digested albuminous articles; fluids are not to be given in large amounts, since this tends to overfilling of the vessels, increases the arterial tension, and delays absorption.

Local Measures.—Flannel should be kept over the precordia, so as to avoid exposure and undue chilling. The ice-bag or Leiter's coils (to be used in the first stage) should be cautiously employed during the second stage, until the temperature has defervesced considerably, thus indicating a subsidence of inflammation in the pericardium.[1] Subsequently, if absorption does not proceed satisfactorily, blisters may be applied over the precordia; but should the patient's general condition be decidedly bad, an absorbifacient containing iodin, lanolin, and ichthyol may be substituted with advantage.

The *therapeutic measures* must be chosen with sole reference to the primary disease, which the physician must continue to treat while he attempts by other means to relieve certain symptoms and promote absorption. For example, if the pericarditis be due to rheumatism, the use of the salicylates must be persevered in, and opium may be added to quiet restlessness and procure relief from pain. In my own experience absorption has been best promoted by the use of the double iodid of potassium and iron, or of iron and manganese. These agents are seldom contraindicated unless they are badly borne by the stomach. Diuretics and saline purgatives are not without value, but do good only in the later stages. Depressing measures of whatever sort are not to be resorted to unless the circulation be good. If the pulse be small, weak, and rapid, with marked cyanosis, stimulants are indicated and are to be given in moderate quantity; the pulse will then be found to grow stronger and the dyspnea and cyanosis less marked. Strychnin and the salts of ammonium will be found to be useful. Digitalis and strophanthus are not to be thought of when myocarditis is associated; at other times they often improve the peripheral circulation and increase the urinary secretion. When the breathing becomes greatly embarrassed and the circulation fails, as shown by the feeble, broken, rapid pulse and the cyanotic hue of the lips, eyelids, and finger-tips, cardiocentesis is indicated, and in sero-fibrinous effusion aspiration has, in recent years, given good results if not too long delayed. If the slightest doubt arises as to the character of the fluid, a preliminary puncture with a hypodermic needle should be made. The point for puncturing is the fourth interspace, 1 inch (2.5 cm.) from the parasternal line, or the fifth interspace, 1½ inches (3.7 cm.) from the left edge of the sternum. The operation must be performed with the strictest asepsis, and the amount of liquid withdrawn at any one time should not exceed two or three ounces. It is better to repeat the puncture several times than to remove the pressure too suddenly from the damaged heart. Of 60 cases of paracentesis for pericarditis of different varieties, collected by Roberts, 24 terminated in recovery.

PURULENT PERICARDITIS.

(*Empyema of the Pericardium.*)

Pathology and Etiology.—The condition often follows the sero-fibrinous form. Septic and tuberculous processes involving the pericardium are also apt to cause purulent effusion, and many of the cases that arise in the course of the acute infectious diseases belong to this category. The pneumococcus has been found in the pus (Shattuck and Porter). The membrane is much thickened and presents a gray, granular surface, and the myocardium underlying the visceral layer is softened, fragile, and pale-looking (fatty).

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[1] If the pericarditis be secondary to an acute febrile disease, this fact must modify the method here recommended accordingly.

Clinical History.—The local subjective symptoms and physical signs are the same in kind as in the former variety, but the amount of exudation is frequently less. At the onset *rigors* often occur, and may be repeated at varying intervals. The *temperature-curve* is of the suppurative type; the *pulse* is small, rapid, and irregular; and *physical prostration* is pronounced. Purulent pericarditis runs a comparatively rapid and an almost uniformly unfavorable course.

Diagnosis.—The chief clinical features are often referable to the primary or causal disease; hence in every instance in which purulent pericarditis is apt to arise a physical exploration of the chest is imperative. The purulent character of the effusion cannot readily be ascertained, as a rule; but the history of an affection having etiologic importance, the observance of rigors, and the presence of the fever-curve peculiar to suppuration would all point strongly to purulent effusion, and should lead to aspiration with the hypodermic needle—a harmless procedure if carefully performed, and one that almost constantly gives reliable results.

Treatment.—It is within the physician's province to treat the primary disease assiduously, but not pericardial empyema. Incision (after preliminary resection of a rib—Brentano), irrigation of the sac, and drainage by a strand of iodoform gauze are advisable and feasible measures.

HEMORRHAGIC PERICARDITIS.

In purulent pericarditis the effusion may be hemorrhagic, and particularly when it is of tuberculous origin. In non-purulent tuberculous pericarditis also the exudation is apt to be hemorrhagic. In the non-purulent instances that are due to chronic Bright's disease or that occur in the aged the effusion is sometimes blood-stained; and future observation may show that the hemorrhagic variety is of more frequent occurrence than has hitherto been supposed. In ordinary serous pericarditis there is apt to be present more blood than in serous pleuritis. M. T. Ferrier has found 5 examples in 9 collections. Sears found a pure growth of pneumococci in the exudate from a case of hemorrhagic pericarditis. This etiologic variety scarcely calls for separate clinical consideration.

ADHESIVE PERICARDITIS.

(Chronic Pericarditis.)

Pathology and Etiology.—Chronic pericarditis follows the acute forms, and, as in the case of the latter, it may be partial or general. The effusion may rarely remain as a permanent condition, though not infrequently a clear history of the preceding acute attack is wanting. In most instances the opposed surfaces of the membrane are either universally or over a limited area firmly adherent. The amount of new connective tissue present or the degree of thickening of the layers varies greatly, and is dependent upon the type of the primary acute attack. If the latter is of mild grade—as, for example, in the case of the sero-fibrinous variety complicating rheumatism—then not much thickening is encountered in the resulting chronic form.

Chronic tuberculous pericarditis is not uncommon, and may be primary, though more commonly it is secondary, in its origin. The disease may be chronic from the time of onset. I have noticed that often more or less effusion prevails. The layers become enormously thickened, and total obliteration of the sac by agglutination of the surfaces is not infrequent.

In the dense exudate that remains after complete absorption of a pericardial effusion calcareous depositions occur, forming a bony casing, which either partially or totally encircles the organ. The external surface of the pericardium may become united with adjacent tissues. The myocardium is the seat of atrophic and degenerative changes.

Symptoms.—Autopsies frequently discover an unsuspected adhesive pericarditis. Hypertrophic dilatation of the chambers usually develops sooner or later, and is due to adhesions that interfere with the free action of the organ as well as with its systole. When present the subjective symptoms point to a giving way of the right ventricle, as shown by the presence of *venous stasis* and *dropsy*. The *pulse* is rapid, of low tension, and irregular, and, though not diagnostic, the *pulsus paradoxus* is noted.

Pericarditis Callosa (Galvagni[1]).—A form of chronic fibrous pericarditis which comes on insidiously during childhood and is exceedingly difficult of diagnosis (*vide infra*). Pericarditis callosa is characterized principally by facial cyanosis, slight edema, full and tortuous jugular veins without pulsation. The typical physical signs of pericarditis are wanting also. On the other hand, a congestive cirrhosis of the liver may supervene and lead to ascites.

Physical Signs.—*Inspection.*—Depression or pitting of the intercostal spaces over the position of the heart may be noticed. Synchronous with the systole there is also a retraction of the chest-wall in the apical area, and less frequently over the whole precordia, the latter being an unerring sign of universal adhesions. The degree of systolic recession is slightly influenced by the respiration, inspiration increasing it, except adhesions exist between the pericardium and the adjoining pleura, and it is best appreciated on palpation. When the apex-beat is not palpable, the systolic pitting over its site may be due to atmospheric pressure. During the diastole the heart forcibly rebounds, causing the so-called diastolic shock, which is of great diagnostic importance when associated with marked systolic retraction. Though not always visible, it can be readily felt on *palpation*. Friedreich's sign (the sudden collapse of the jugulars during diastole) may frequently be observed, but I have also noticed this in cardiac dilatation without adhesions. Prior to the onset of dilatation the apex-beat may be forcible and visible over an increased area, indicating hypertrophy; but after the myocardium is weakened (from interference with its nutrition) and dilatation comes on, the impulse-beat is faint or wanting, and in marked systolic retraction may be seen to be vibratory. The fixed position of the apex-beat when the patient is turned over upon his left side is a strong confirmatory sign.

Percussion.—The area of cardiac dulness is increased, especially upward and to the left, owing to the associated hypertrophy and pleuro-pericardial adhesions, and, since the adhesions between the pleura and the pericardium do not allow the lungs to overlap the heart during inspiration, the upper and left lines of dulness remain fixed (C. J. B. Williams).

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[1] *University Med. Mag.*, March, 1899; *Clinique moderne*, ann. iv., No. 341.

Auscultation.—When dilatation reaches a high degree the auscultatory signs peculiar to that condition appear. In many cases no murmurs are detectable, but in a third group loud murmurs, quite independent of any value as regards cardiac lesions, are audible; these murmurs may be due to the vortiginous movements in the endocardial blood-current occasioned by the jogging cardiac action. The murmur of tricuspid regurgitation, from a breakdown of the right ventricle without apparent exciting cause, is most significant.

Differential Diagnosis.—The condition is apt to be confounded with *chronic myocarditis* and *simple hypertrophic dilatation.*

As before stated, chronic pericarditis may be associated with effusion, and it is important to distinguish such instances from the adhesive form, if we would institute a proper treatment. In chronic pericarditis with moderate effusion the seat of the apex-beat is higher and less undulatory, and when the amount of effusion is large the impulse is absent and there is bulging. Adhesive pericarditis with hypertrophy causes bulging in young subjects, but the apical beat is retained. In pericarditis with effusion the upper and left limits of dulness are not stationary, and there is an absence of systolic retraction and diastolic concussion.

Course and Prognosis.—The hypertrophy that comes on early in consequence of the obstruction offered to cardiac action is compensatory, and this harmonious balance may be maintained for a long period of time with apparent comfort. After myocardial degeneration, followed by atrophy or dilatation, has occurred, the condition becomes quite serious, and death usually ensues amid signs of extreme cardiac dilatation.

The **treatment** must be ordered chiefly with reference to the nutrition of the heart-muscle, following the principles noted in dealing with the management of valvular affections of the heart. If chronic effusion be present early, operative measures are to be warmly advocated.

HYDROPERICARDIUM.

(*Dropsy of the Pericardium.*)

Definition.—A condition in which the pericardium contains a serous transudation, while the membrane itself shows no signs of inflammation.

Etiology.—(*a*) Hydropericardium is usually associated with general cardiac or renal dropsy, of which it forms a component part. Under these circumstances it develops late, and frequently follows hydrothorax, on account of which condition it is liable to be overlooked. It may also occur suddenly in chronic nephritis, and particularly in the scarlatinal variety. (*b*) It arises not infrequently from local mechanical causes, as the pressure of mediastinal tumors, aneurysm, or thrombosis of the cardiac veins.

Symptoms.—No subjective symptoms are present, save perhaps dyspnea, and the diagnosis rests upon the history and the physical signs. None of the latter, however, are particularly significant. They point to the latter is of mild grade—as, for example, in the case of the sero-fibrinous variety complicating rheumatism—then not much thickening is encountered in the resulting chronic form.

Chronic tuberculous pericarditis is not uncommon, and may be primary, though more commonly it is secondary, in its origin. The disease may be chronic from the time of onset. I have noticed that often more or less effusion prevails. The layers become enormously thickened, and total obliteration of the sac by agglutination of the surfaces is not infrequent.

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Symptoms.—Autopsies frequently discover an unsuspected adhesive pericarditis. Hypertrophic dilatation of the chambers usually develops sooner or later, and is due to adhesions that interfere with the free action of the organ as well as with its systole. When present the subjective symptoms point to a giving way of the right ventricle, as shown by the presence of *venous stasis* and *dropsy*. The *pulse* is rapid, of low tension, and irregular, and, though not diagnostic, the *pulsus paradoxus* is noted.

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Physical Signs.—*Inspection.*—Depression or pitting of the intercostal spaces over the position of the heart may be noticed. Synchronous with the systole there is also a retraction of the chest-wall in the apical area, and less frequently over the whole precordia, the latter being an unerring sign of universal adhesions. The degree of systolic recession is slightly influenced by the respiration, inspiration increasing it, except adhesions exist between the pericardium and the adjoining pleura, and it is best appreciated on palpation. When the apex-beat is not palpable, the systolic pitting over its site may be due to atmospheric pressure. During the diastole the heart forcibly rebounds, causing the so-called diastolic shock, which is of great diagnostic importance when associated with marked systolic retraction. Though not always visible, it can be readily felt on *palpation*. Friedreich's sign (the sudden collapse of the jugulars during diastole) may frequently be observed, but I have also noticed this in cardiac dilatation without adhesions. Prior to the onset of dilatation the apex-beat may be forcible and visible over an increased area, indicating hypertrophy; but after the myocardium is weakened (from interference with its nutrition) and dilatation comes on, the impulse-beat is faint or wanting, and in marked systolic retraction may be seen to be vibratory. The fixed position of the apex-beat when the patient is turned over upon his left side is a strong confirmatory sign.

Percussion.—The area of cardiac dulness is increased, especially upward and to the left, owing to the associated hypertrophy and pleurothe distinction from pericarditis can rarely be made. In pneumopericardium there is tympanitic percussion-resonance over the precordia, though the fluid, when present, gives rise to a boundary-line of dulness. The change of the patient's posture decidedly alters the area of the tympanitic note. On auscultation may be heard loud, rasping, friction-sounds having a metallic quality, intermingled with churning, splashing noises, or the so-called "water-wheel sounds." I have, however, met with two cases in which the heart-sounds were exceedingly feeble. *Pneumothorax* when encysted in close proximity to the heart, displacing the latter organ, must be eliminated. The latter complaint gives cardiac dulness in an abnormal position and a metallic sound synchronously with the respiratory movements—two signs diagnostic of pneumothorax that are absent in pneumopericardium. *The prognosis* is grave, death coming on most commonly in a day or two. The admission of air might alone result in a spontaneous cure, as occurs rarely in pneumothorax. *The treatment* is the same as has been recommended for purulent pericarditis.

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[1] *University Med. Mag.*, March, 1899; *Clinique moderne*, ann. iv., No. 341.

II. DISEASES OF THE HEART.

ENDOCARDITIS.

Definition.—Inflammation of the lining membrane of the heart. The process is usually confined to the valves, though the cardiac layer may also be affected.

Varieties.—(*a*) Simple acute endocarditis; (*b*) ulcerative endocarditis; (*c*) chronic endocarditis. The pathologic processes involved in the first two, the acute forms, are identical in nature, though they differ in severity. I have met with two instances that could be referred to neither sub-variety, apparently occupying a middle ground.

SIMPLE ACUTE ENDOCARDITIS.

(*Endocarditis Verrucosa.*)

Pathology.—The disease is characterized by the formation of small vegetations on the segments, varying in size from excrescences that are scarcely visible to those the size of a pea. They are found chiefly on surfaces that are opposed to the blood-current, near the margin of the valve, and "forming a row of bead-like outgrowths." Their seat corresponds to the point of maximum contact (Sibson), but the mitral valve is much more commonly affected than the aortic. With the segments the chordæ tendineæ are sometimes affected, and very rarely the latter are alone involved. The left side of the heart is much more frequently the seat of acute endocarditis than the right, except during fetal life, when the right side is almost exclusively involved. To account for the greater frequency of occurrence on the left side after birth, it has been suggested that freshly oxygenated blood affords the most favorable condition for the multiplication of the micro-organisms that are concerned in the inflammatory process. As corroborating this view, the fact is adduced that during the presence of fluid, and the area of percussion-dulness assumes the same form and exhibits even greater change, with alteration of the patient's posture, than in pericarditis. No friction-murmurs are heard and no bulging of the pericardium is observed. It is rare indeed, I have found, to see an excessive amount of serum in the pericardium at the *postmortem*. The symptoms and signs of hydrothorax generally precede and accompany hydropericardium, and the latter condition tends to intensify the effect of the former. The condition, *per se*, is rarely of serious import. Osler remarks: "Naturally there are in the pericardial sac a few cubic centimeters of clear, citron-colored fluid, which probably represents a postmortem transudate." In rare instances the transudate has a milky appearance (*chylo-pericardium*).

The **treatment** suitable for cases of general dropsy, as a rule, affords relief. In large serous accumulations aspiration should be practised.

HEMOPERICARDIUM.

By the term "hemopericardium" is meant hemorrhage into the pericardial pouch—a rare event. Among the causes are—(*a*) perforation by aneurysms of the aorta and the coronary arteries into the sac; (*b*) rupture of the heart, due to injuries or cardiac aneurysms and fibrous formations from myocarditis; (*c*) direct injuries, especially stab- and bullet-wounds. *The symptoms and course* depend greatly upon the nature of the exciting cause. The most frequent factor, rupture of an aneurysm, proves quickly fatal from overcrowding of the heart. In rupture of the heart-muscle there is sometimes a slow outpouring of blood, with a correspondingly slow course, varying from a few hours to a couple of days in duration. The physical signs of effusion come on with dyspnea and failing circulation, which lead to cardiac exhaustion and death. The blood-stained effusions, before considered, that are met with in certain forms of pericarditis, are not to be regarded as instances of hemopericardium.

PNEUMOPERICARDIUM.

(*Air in the Pericardium.*)

In this complaint, besides air or gas, there is usually present pus, and less frequently blood; hence an appropriate term in most instances would be *pyo-pneumopericardium*. When the pericardium is perforated purulent pericarditis results. *The causes* are the following: (*a*) wounds; (*b*) a fistulous connection between the adjacent air-containing organs and the pericardium as the result of diseased processes, such as pulmonary tuberculosis or empyema; (*c*) rarely decomposition of liquid pericardial effusions. *The symptoms* are equivocal. In the main they do not differ from those of pericarditis with effusion, excepting that dyspnea is more intense than in the latter affection. By attention to the physical signs microörganisms or their toxins, whose action is assisted by the friction between the blood current and the surfaces of the valves. Fränkel and Sänger affirm that the staphylococcus pyogenes aureus is the chief specific, causative, agent. The diplococcus pneumoniæ, the streptrococcus pyogenes, and, less commonly, the Bacillus coli communis, the gonococcus, the Bacillus diphtheriæ, the bacillus of Eberth, and the micrococcus endocarditis capsulatus and rugatus, have been found.

Clinical History.—It is only occasionally that definite subjective symptoms, as precordial *pain* (sometimes extending down the left arm), *dyspnea*, and *cardiac palpitation*, are complained of by the patient. If fever have been present, as is common, the temperature usually rises rather abruptly. In the vast majority of instances the condition is discovered accidentally. This being true, its frequent occurrence in acute articular rheumatism, and its occurrence in the other diseases mentioned under "Etiology," should be kept in remembrance. The symptoms of *embolism* are rarely observed. F. Billings reports a case with multiple emboli.

The **physical signs** by which acute endocarditis is recognizable are dependent upon the valvular insufficiencies caused by the morbid lesions previously described. Hence there must be not a small proportion of mild cases, including those in which the valves are not affected, that give rise to no distinct physical signs.

On *inspection* the area of visible impulse may be seen to be increased, though, as a rule, it is normal. The impulse is sometimes forcible and often irregular during the initial period, but later it becomes less distinct and more feeble. *Palpation* confirms the result of inspection. I have found the impulse to vary at each visit, with a general tendency to lessen in intensity in the later period of the disease. A very weak impulse is indicative of associated myocarditis or of the poisonous effect of a severe type of primary infection. In recurrent endocarditis the apical impulse is often heaving, on account of pre-existing compensatory hypertrophy, and its area is exceedingly variable. A systolic thrill is sometimes felt.

On *percussion* the area of the heart's dulness is found to be normal, except in cases of intense myocardial involvement, with dilatation of the chambers when the area of percussion-dulness is increased in the transverse diameter. In recurrent acute endocarditis the area of dulness corresponds to the increased area of the apical beat.

Auscultation.—Acute endocarditis is usually attended with a soft blowing, systolic murmur, which, owing to the fact that the mitral segments are the favored seat of the disease, is heard much more frequently at the apex than at the base. The point of maximum intensity of this murmur is often movable, but its area of transmission is limited. In rheumatic endocarditis this murmur is preceded by a prolongation of the first sound, and it may be rough in character, and associated with accentuation of the aortic second sound, later with a valvular pulmonary second. The murmur is sometimes heralded by a feeble or muffled first sound, with apparent intensification of the second, suggesting ventricular dilatation as the cause of the murmur. In acute endocarditis affecting the mitral valves aortic murmurs may coexist, but their true nature is more than doubtful. There is also a short, low-toned, and double systolic murmur over the tricuspid orifice in a small proportion of the cases; this is due most probably to a relative incompetency. When acute endocarditis arises in connection with chronic valvular disease, the auscultatory signs of the latter are but little changed, and hence an assured diagnosis is not possible.

Complications.—There may be developed by direct extension secondary myocarditis, a disease that will receive separate consideration.

The **diagnosis** is based principally on the physical signs, though these are by no means trustworthy. The points gained by careful, inspection and palpation are of especial diagnostic importance, as is also the previous history of the patient. Leube[1] points out that if the dulness is slightly increased to the left and there is fever—in fact, if there is infectious disease present—a diagnosis must be made of acute insufficiency of the ostium mitralis occurring in the course of acute endocarditis.

Differential Diagnosis.—The soft bellows murmur is often present in *acute febrile diseases* in which the autopsy fails to reveal the lesions of acute endocarditis. The functional murmurs that arise in the specific fevers, however, are principally heard over the aortic area, while those occurring in endocarditis are commonly heard over the mitral area. The murmurs present must be called *accidental* if the area of cardiac dulness is normal, the second pulmonary sound not accentuated, and if the murmur be heard only at the pulmonary cartilage, or at this point and at the apex, and, at any rate, more distinctly at the pulmonary cartilage (Leube[2]). The distinction between simple acute endocarditis and *pericarditis* should be categorical, in view of the manifold differences between their signs. But the fact that these two affections may be associated, more especially when they are of rheumatic origin, must be steadily borne in mind, and also that when combined the signs belonging to the endocarditis are not open to observation, owing to the pericardial friction-sound, and later the presence of the effusion. I have found, however, that, fortunately, endocarditis precedes pericarditis in the majority of the cases. The elimination of *old endocarditis* or *chronic valvular disease*—a matter of importance—may be accomplished by attention to the character of the murmur in acute endocarditis, as well as to its limited area of diffusion, and by the absence of the signs of hypertrophy and of marked accentuation of the second pulmonary sound.

A *relative insufficiency* distinguishes itself by a pure systolic murmur, loud and not invariably uniform, by a weak cardiac impulse, a slight accentuation of the second pulmonary sound, and a comparatively small and often irregular pulse. It is met with in excessive dilatation of the left ventricle, in anemia, "and particularly in certain changes of the valvular muscles, due to myocarditis" (Leube).

Prognosis.—The immediate dangers are few, and depend largely upon the primary disease. In many instances, however, acute endocarditis initiates permanent lesions of the valves.

Treatment.—**Prophylaxis.**—The prevention of acute endocarditis in rheumatism has been dealt with in discussing the latter disease. No known direct measures can prevent the development of this condition in the course of the specific fevers, though absolute rest in bed and protection of the body against "cold" may diminish somewhat the tendency to it.

The Attack.—The sick-room should be free from draughts, though well ventilated, and flannel is to be applied to the chest. The diet may be liberal, but should be composed chiefly of milk and other light nutritious substances. Stimulants are required in most instances, and microörganisms or their toxins, whose action is assisted by the friction between the blood current and the surfaces of the valves. Fränkel and Sänger affirm that the staphylococcus pyogenes aureus is the chief specific, causative, agent. The diplococcus pneumoniæ, the streptrococcus pyogenes, and, less commonly, the Bacillus coli communis, the gonococcus, the Bacillus diphtheriæ, the bacillus of Eberth, and the micrococcus endocarditis capsulatus and rugatus, have been found.

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[1] *Deutsch. Archiv f. klin. Med.*, Nov. 5, 1896.

[2] *Loc. cit.*

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On *inspection* the area of visible impulse may be seen to be increased, though, as a rule, it is normal. The impulse is sometimes forcible and often irregular during the initial period, but later it becomes less distinct and more feeble. *Palpation* confirms the result of inspection. I have found the impulse to vary at each visit, with a general tendency to lessen in intensity in the later period of the disease. A very weak impulse is indicative of associated myocarditis or of the poisonous effect of a severe type of primary infection. In recurrent endocarditis the apical impulse is often heaving, on account of pre-existing compensatory hypertrophy, and its area is exceedingly variable. A systolic thrill is sometimes felt.

On *percussion* the area of the heart's dulness is found to be normal, except in cases of intense myocardial involvement, with dilatation of the chambers when the area of percussion-dulness is increased in the transverse diameter. In recurrent acute endocarditis the area of dulness corresponds to the increased area of the apical beat.

Auscultation.—Acute endocarditis is usually attended with a soft blowing, systolic murmur, which, owing to the fact that the mitral segments are the favored seat of the disease, is heard much more frequently at the apex than at the base. The point of maximum intensity of this murmur is often movable, but its area of transmission is limited. In rheumatic endocarditis this murmur is preceded by a prolongation of the first sound, and it may be rough in character, and associated with accentuation of the aortic second sound, later with a valvular pulmonary second. The murmur is sometimes heralded by a feeble or muffled first sound, with apparent intensification of the second, suggesting ventricular dilatation as the cause of the murmur. In acute endocarditis affecting the mitral valves aortic murmurs may coexist, but their true nature is more than doubtful. There is also a short, low-toned, and double systolic murmur over the tricuspid orifice in a small proportion of the cases; this is due most probably to a relative incompetency. When acute endocarditis arises in connection with chronic valvular disease, the auscultatory signs of the latter are but little changed, and hence an assured diagnosis is not possible.

Complications.—There may be developed by direct extension secondary myocarditis, a disease that will receive separate consideration.

The **diagnosis** is based principally on the physical signs, though these are by no means trustworthy. The points gained by careful, inspection and palpation are of especial diagnostic importance, as is also the previous history of the patient. Leube[1] points out that if the dulness is slightly increased to the left and there is fever—in fact, if there is infectious disease present—a diagnosis must be made of acute insufficiency of the ostium mitralis occurring in the course of acute endocarditis.

Differential Diagnosis.—The soft bellows murmur is often present in *acute febrile diseases* in which the autopsy fails to reveal the lesions of acute endocarditis. The functional murmurs that arise in the specific fevers, however, are principally heard over the aortic area, while those occurring in endocarditis are commonly heard over the mitral area. The murmurs present must be called *accidental* if the area of cardiac dulness is normal, the second pulmonary sound not accentuated, and if the murmur be heard only at the pulmonary cartilage, or at this point and at the apex, and, at any rate, more distinctly at the pulmonary cartilage (Leube[2]). The distinction between simple acute endocarditis and *pericarditis* should be categorical, in view of the manifold differences between their signs. But the fact that these two affections may be associated, more especially when they are of rheumatic origin, must be steadily borne in mind, and also that when combined the signs belonging to the endocarditis are not open to observation, owing to the pericardial friction-sound, and later the presence of the effusion. I have found, however, that, fortunately, endocarditis precedes pericarditis in the majority of the cases. The elimination of *old endocarditis* or *chronic valvular disease*—a matter of importance—may be accomplished by attention to the character of the murmur in acute endocarditis, as well as to its limited area of diffusion, and by the absence of the signs of hypertrophy and of marked accentuation of the second pulmonary sound.

A *relative insufficiency* distinguishes itself by a pure systolic murmur, loud and not invariably uniform, by a weak cardiac impulse, a slight accentuation of the second pulmonary sound, and a comparatively small and often irregular pulse. It is met with in excessive dilatation of the left ventricle, in anemia, "and particularly in certain changes of the valvular muscles, due to myocarditis" (Leube).

Prognosis.—The immediate dangers are few, and depend largely upon the primary disease. In many instances, however, acute endocarditis initiates permanent lesions of the valves.

Treatment.—**Prophylaxis.**—The prevention of acute endocarditis in rheumatism has been dealt with in discussing the latter disease. No known direct measures can prevent the development of this condition in the course of the specific fevers, though absolute rest in bed and protection of the body against "cold" may diminish somewhat the tendency to it.

The Attack.—The sick-room should be free from draughts, though well ventilated, and flannel is to be applied to the chest. The diet may be liberal, but should be composed chiefly of milk and other light nutritious substances. Stimulants are required in most instances, and in abundance should the heart be failing. Digitalis is to be employed cautiously if at all. When the myocardium is involved, its use is not without danger; under these circumstances the drug increases the sufferings of the patient by throwing the inflamed and weakened cardiac muscle into firm contractions. The salts of ammonium, particularly the carbonate, should be given continuously with a view to obviating intracardial coagulation of blood; and should the latter accident occur despite all efforts to prevent it, the carbonate, together with strychnin and alcoholic stimulants, should be freely administered. I am convinced that in endocarditis due to acute articular rheumatism it is wise to continue the exhibition of the salicylates, though in moderate doses, provided that the heart is guarded by the use of stimulants. During convalescence from an acute endocarditis the patient should be kept at rest, so as to minimize the strain upon the affected valves; even after he has apparently recovered, and particularly should the murmur still be present, perfect quiet is to be enjoined for a period of several weeks.

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[1] *Deutsch. Archiv f. klin. Med.*, Nov. 5, 1896.

[2] *Loc. cit.*

ULCERATIVE ENDOCARDITIS.

(*Malignant or Infectious Endocarditis.*)

Malignant endocarditis is variously characterized, though usually either by perforative ulceration, by suppuration of the valves, or by both, giving rise to the physical signs of acute endocarditis. These develop amid the symptoms of some severe primary infectious or septic disease. There is at hand sufficient clinical evidence to warrant the assumption that ulcerative endocarditis also occurs, though very rarely, as a primary affection.

Pathology.—(*a*) *Valvular Endocarditis.*—In its early development the valves are the seat of vegetations (such as are met with in simple acute endocarditis) which later undergo necrosis. The latter process manifests a tendency to spread, destroying more or less of the endocardium. In the interior of the vegetations the process of suppuration not infrequently takes place, and the abscesses thus formed rupture and produce various lesions according to their size and situation. The vegetations take on a grayish- or yellowish-green appearance. Histologically, they are composed of granulation tissue, veiled by granular and fibrillated fibrin that contains numerous micro-organisms. At the base there is usually developed more or less reactionary inflammation. After rupture the blood-current may enter the abscess-cavity, and, if there be no complete perforation, the endocardium will be pouched out, and an aneurysmal dilatation of the valve will result. Ulcerative lesions are most frequently observed. They may be mere erosions of the endocardium, but, as a rule, are penetrating in character and often result in complete perforation. I have seen repeated instances in which the three classes of lesions above depicted were all present. Osler, in an analysis of 209 cases examined by him with a view to ascertaining approximately the relative frequency with which the different parts of the heart were affected, obtained this result: Aortic and mitral valves together, 41; aortic valves alone, 53; mitral valves alone, 77; tricuspid in 19, pulmonary valves in 15, and the heart-wall in 33 instances. In 9 instances the right heart alone was involved.[1]

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[1] *Text-Book of Medicine*, p. 631.

(*b*) *Malignant mural endocarditis* gives the same set of changes as the valvular form; indeed, the latter may be combined with the former throughout. It is a comparatively rare condition, as is shown by the foregoing figures of Osler. The ulcerative process may invade the chordæ tendineæ and the valves, and may perforate the septum or even the ventricular wall itself. The vegetations are detached in small or large masses, and are conveyed by the circulating medium to various distant organs, especially to the spleen and kidneys, less frequently the intestines, meninges of the brain, and the skin. Their site is determined largely by their size, and they may be so large as to plug vessels of the caliber of the external iliac. When found in the lungs they may originate in endocarditis affecting the right heart. These emboli, containing, as they do, the agents of inflammation, form suppurative infarcts that may be either white or red in color. The detached vegetations are sometimes so laden with irritants as to cause rapid softening of the coats of the vessel at the point where they become arrested, with consequent aneurysmal dilatation directly opposite their seat. As to number, the infarcts vary greatly in different cases; thus there may be only one or two, as in a case in my own knowledge in which the spleen alone contained two small infarcts, or there may be more than a thousand minute abscesses widely scattered throughout the body.

Etiology.—It is to be kept in remembrance that the condition is, with few exceptions, most probably a secondary one. This explains why the lesions peculiar to simple acute endocarditis usually precede and accompany those of the ulcerative form.

Bacteriology.—The specific irritant is probably the *streptococcus pyogenes* (Fränkel and Sänger); hence the diseases in which ulcerative endocarditis occurs as a complication merely furnish the opportunity for the invasion of the streptococcus. The bacillus diphtheriæ, however, as well as the staphylococcus, the bacillus coli, the bacillus anthracis, the pneumococcus, the gonococcus, and other organisms, have been found in some cases in the absence of the streptococcus.

In purely septic diseases ulcerative endocarditis forms but a part of the serious general condition. Here the cardiac element serves to facilitate the generation and rapid diffusion of the poison; and, since the latter is prone to attack the valve-segments, the morbid lesions within the heart not rarely constitute the chief pathologic factor in septicopyemia. Instances, however, are met with in which the segments present slight changes.

Predisposing Affections.—The malignant form occurs, in connection with acute articular rheumatism, in about 10 per cent. of the cases in which acute endocarditis appears. In lobar pneumonia the ulcerative type is common, occurring almost as freqnently as the simple variety, and was found by Osler in 11 out of 23 cases. The septic processes that arise from the puerperal state or from gonorrheal infection may also be complicated with ulcerative endocarditis. Among many other diseases that furnish occasional instances of this serious complication are measles, scarlet fever, typhoid fever, erysipelas, small-pox, chorea, tuberculosis, and chronic Bright's.

Clinical History.—That form of ulcerative endocarditis which is a more or less prominent factor in septic diseases has been considered in connection with septicemia. Malignant endocarditis being usually a secondary event, its clinical features must not be confounded with those of the primary affection. It is, however, often impossible clearly to separate the symptoms of the former from those of the intercurrent affection. I shall describe first the common *typhoid form.*

Local symptoms are often entirely wanting, or, when present, consist merely in slight precordial pain and oppression, and are not sufficiently well pronounced to arrest attention. Subjective symptoms are, however, connected with other organs than the heart, and are due to the irritating effects of emboli that occupy the various organs of the body. *Gastro-intestinal disturbance*, as shown by the occurrence of vomiting and diarrhea, is common. *Pain* that is ascribable to local peritonitis over the spleen, and sometimes also over the liver, is observed. *Hematuria* and *dimness of vision* are also frequent concomitants, and are due to renal and retinal hemorrhages. The *urine* may be scanty and albuminous. The more *general features* that are the result of the local embolic processes and, in part, of the valvular lesions, are serious and for the most part typhoid in character. The *onset* is usually signalized by a severe rigor that may be repeated at intervals varying from one to several days, and the disease often presents an irregularly continued fever-curve, often touching a high mark (105° or 106° F—40.5° or 41.1° C.). I saw a case recently in which the febrile movement pursued the continued type for seven weeks. The *pulse* is rapid and irregular, though frequently becoming slow within a brief period. The patient rapidly *emaciates*, and from the earliest development is profoundly prostrated, and *nervous symptoms*, as headache, mild delirium, followed by somnolence, and sometimes even coma, appear. Profuse sweating sets in and persists, and as a result the *skin* may be covered by sudamina. An ecchymotic eruption due to cutaneous *emboli* is also common, this being often found associated with a papular or a diffused roseolar rash.

Physical Signs.—These may be negative as regards the heart. In the majority of instances, however, a *systolic murmur* is present, which, when associated with other clinical indications that point to this affection, is valuable for diagnosis, and especially so if developed while the patient is under treatment for the primary attack. The second sound is sometimes accentuated even when no organic lesions have previously existed. The physical signs of pneumonia and pleuritis (particularly the latter) may not infrequently be noted. Cases occur in which gangrenous infarcts of the right lung give rise to signs of localized consolidation ; the spleen becomes considerably swollen, as can be easily demonstrated by palpation, and is quite tender as a rule ; and the liver is likewise moderately enlarged and slightly sensitive.

Cerebral Variety.—In a small though decisive percentage of the cases all the clinical features of acute suppurative meningitis are presented, and sometimes to the almost total exclusion of symptoms pointing to the primary disease or to the more typical typhoid form of ulcerative endocarditis. For a description of the symptoms that characterize the cerebral form the reader is referred to the discussion of Purulent Meningitis.

Recurrent Malignant Endocarditis.—By this term is meant an acute ulcerative endocarditis coming on in the course of chronic valvular disease. As has been pointed out, simple acute recurrent endocarditis is common, though difficult of recognition. The latter condition, as well as the lesions in chronic valvular disease, predisposes to secondary infection by the streptococcus and other organisms. The onset is usually abrupt and marked by a chill. The patient has fever, which may be quite high (104° F.—40° C., or over), and may present either an irregularly intermittent or a truly intermittent curve. The latter is often associated with recurring chills. In either of the above *groups* the course is likely to be acute. In some cases the character of the pre-existing murmur is changed, becoming louder and more decidedly blowing; in many other instances, however, there is no appreciable alteration in the murmur. The condition may arise suddenly, amid the signs of failing compensation, as in a fatal case reported by Dr. H. P. Loomis,[1] in which the patient was semi-conscious, cyanotic, and suffering from intense dyspnea and general dropsy. It was impossible to diagnosticate the cardiac lesions by the murmur present. Occasionally these severe intercurrent febrile attacks end in recovery, and such cases probably belong to the benign form, though closely simulating the malignant in their clinical characters.

There is a third group of cases that run a subacute or even chronic course, with more moderate elevations of temperature, or, as rarely happens, none at all. Mullin of Hamilton has reported a case that lasted more than a year. Here the other clinical phenomena, especially those referable to the heart, are often scanty and indefinite.

Diagnosis.—It is of paramount importance to consider the previous history and all the circumstances under which individual cases occur. These points, together with the symptoms attending the onset and the first three or four days of illness, more particularly the severe rigor, early high temperature, and profound prostration, the sweatings, the various embolic phenomena, and the presence of cardiac symptoms, are often adequate for a certain diagnosis. With a clear history and the presence of the more characteristic general symptoms (in particular, the *signs of embolism*), a correct diagnosis is possible, even though cardiac murmurs be absent. Instances in which no data can be found to explain the occurrence of the disease are especially puzzling, and these will remain unrecognized if at the same time the heart manifests no special symptoms, and embolic phenomena are absent. The existence of a chronic valvular affection would under the latter circumstances afford strong probability of the presence of recurrent malignant endocarditis if the other significant clinical symptoms mentioned above were present.

Differential Diagnosis.—There is a group of cases in which either the history fails to furnish the essential causal factors on the one hand, or there is an absence of definite heart-symptoms on the other; this group cannot sometimes be separated from cases of *typhoid fever*. The subjoined table will, I feel, be found valuable as an aid in eliminating the latter disease from the typhoid form of malignant endocarditis:

ULCERATIVE ENDOCARDITIS.	TYPHOID FEVER.
Previous or associated disease, as acute rheumatism or pneumonia.	Previous health good. History of an epidemic.
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[1] *Transactions of the New York Pathological Society*, 1890.

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[1] *Transactions of the New York Pathological Society*, 1890.

ULCERATIVE ENDOCARDITIS.	TYPHOID FEVER.
Ushered in suddenly by a severe rigor, which may recur.	Invasion marked by slight recurring chilly sensations. (Severe chill very rarely.)
The fever rises rapidly.	More gradually, in step-like fashion.
Profound prostration as early as third day.	Profound prostration not earlier than seventh day.
The fever is markedly irregular from time of onset, as a rule.	Less so, especially in the first week.
Embolic symptoms (hemiplegia, etc.) may appear.	Extremely rare.
Cardiac symptoms, especially loud systolic murmur, often present.	Sometimes a soft systolic murmur.
The blood usually shows signs of septic leukocytosis.	The blood shows a decrease in the number of leukocytes.
Widal reaction and characteristic eruption absent.	Both symptoms usually present and diagnostic.[1]

Prognosis.—Most cases that run an acute course terminate in death, and when supposed instances of malignant endocarditis recover they are usually to be regarded as being of benign character. Subacute or chronic varieties, however, such as are most frequently met with in connection with organic heart-disease, sometimes end in recovery.

Treatment.—This is largely supportive. The feeding is to be pushed vigorously, and concentrated forms of liquid food should be given at regular, brief intervals. Arterial stimulants in liberal quantities are also demanded, and in addition quinin, sodium salicylate, and antiseptics may be tried. For the embolic symptoms the salts of ammonium give slight promise of beneficial results, and I prefer the carbonate for this purpose. Antistreptococcic serum has recently been used, and has proved efficacious in certain cases. It is obviously of no avail in instances in which the streptococcus is not the causative agent. Moritz treated a case with antistaphylococcic serum, with a favorable issue.

CHRONIC ENDOCARDITIS.

(*Chronic Interstitial Endocarditis.*)

Two clinical varieties are met with—one following the acute form, the other beginning as a chronic inflammation.

Pathology.—The lesions may be limited to the valvular endocardium (their most common seat), or the mural endocardium may also be involved. In not a few instances the lesions are confined to the edges or bases of the segments, and when seen in the early stages there may frequently be observed merely a slight thickening of the free border of the leaflets; in most cases small prominences appear near their free margins. The endocardium looks opaque and its normal elasticity is lost quite early. When the auriculo-ventricular valves are affected the primary seat of inflammation is the auricular face, but when the semilunar valves are diseased the morbid changes begin on the ventricular side and implicate the Aurantian body. Extension of the morbid process to other and all parts of the valvular curtain is common, and it is in cases of this sort that the greatest degree of shrinking and crumpling occurs. The most characteristic lesions consist of inflammation and exudation, which produce cohe-

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[1] The septic form may simulate malaria in its general course. The points of dissimilarity may be found in the discussion of Septicemia.

sion of the segments, roughen the surfaces, and lead to the deposit of fibrin upon them. The *histologic alterations* consist in a proliferation of the endothelial and a round-cell infiltration of the subendothelial connective tissue. Organization of these products of inflammation into connective tissue, with resulting induration and contraction, is the necessary subsequent pathologic event. In old cases calcification of the diseased structure is frequent. The fibrinous deposits in acute endocarditis may become calcareous "at the same time that the sclerotic processes are taking place in the valve" (Stengel). The shrinking shortens the curtains or curls their free edges, and produces insufficiency in either case, since on dropping into the plane of the valvular orifice they fail to close it perfectly. Valves thus deformed may also obstruct the blood-stream. As before mentioned, cohesion of the invaded segments take place, particularly at their bases, and may extend upward for a considerable distance, leading to constriction or stenosis.

Involvement of the semilunar (aortic) segments in the ways previously described opposes an obstruction to the outflowing blood-current on the one hand, and, owing to the inability of the segments to effect perfect closure of the aortic orifice, allows on the other hand a diastolic reflux of blood into the left ventricle. The aortic ring to which the semilunar segments are normally attached becomes sclerosed, and finally the seat of atheromatous changes, either fatty or calcareous. Again, chronic inflammation of the intima of the aorta produces a similarly thickened condition of this layer in spots, followed by atheroma. These changes are most prone to take place in the course of the ascending arch of the aorta or just above the aortic segments. From the aorta and subvalvular ring the diseased processes before described may extend to the coronary arteries. Hence sclerotic and atheromatous alterations in the blood-vessels are found frequently in association with organic valvular defects.

Much less commonly similar lesions are noted at the orifice of the pulmonary artery. A similar involvement of the auriculo-ventricular valves also causes regurgitant and obstructive deformities at the mitral orifice, and in advanced cases the chordæ tendineæ, and even the papillary muscles, are almost invariably invaded by direct extension from the valves. As these structures undergo marked thickening with subsequent contraction, they become shortened and rigid, causing an actual narrowing of the cardiac orifice. In mitral stenosis during the early stages or in the mildest types a more or less complete ring of vegetations encircles the mitral orifice on its auricular aspect. The margins of the orifice also become hardened and roughened, these changes frequently extending to the valvular curtains and the chordæ tendineæ. Under such circumstances the thickened valve could not, during the ventricular diastole, be forced back against the ventricular wall, but would occupy a nearly central position. Owing to cohesion of the free edges of the valvular structures and to contraction of the chordæ tendineæ drawing the leaflets toward the apex of the heart, the transition from this condition to the formation of a hollow cone (*funnel mitral*) is accomplished by natural, easy stages. Extensive union of the segments along their free margins may reduce the aperture to a mere button-hole slip (*button-hole mitral*) as viewed from the auricular aspect. The last two forms of lesions are far less commonly met with at the aortic orifice, though they occur rarely in moderate degree; on the other hand, curling of the valvular edges is far more commonly seen at the aortic than at the mitral orifice, if we except the cases that occur in children. The curtains of the thick, rigid valves may also permanently occupy the plane of the orifice, presenting a small ring-like opening (*annular mitral*).

Fatty degeneration leading to the formation of necrotic (atheromatous) ulcers is common; and calcareous deposits are frequently seen in old cases, either in localized areas or coextensive with the diseased tissue, converting the entire valve into a calcified mass, with loss of the valvular outlines.

In *chronic mural endocarditis* the lesions exhibited are grayish-white, slightly elevated patches that are usually found to invade the underlying muscular structure to a greater or a less extent. Under such conditions of the valves the deposit of fibrin would be greatly favored, and the presence of an ulcerative surface or of a fibrous deposit on the valves affords a ready and satisfactory explanation of the occurrence of embolism in these cases. Emboli may also become detached from cardiac thrombi or from thrombi formed in the peripheral veins. For anatomic reasons the favored seats of embolic processes are, as in acute endocarditis, the spleen, brain, and kidneys, and irritants that cause acute endocarditis find here a tissue-soil whose capacity for resistance to invasion is greatly lowered. Chronic mural endocarditis and chronic myocarditis are, as a rule, due to the extension of the inflammation from the valves, though the ventricular endocardium may be invaded independently of the valvular affection. In one instance of mitral stenosis I observed an enormous calcareous mass partly in the subvalvular tissue and partly in the wall of the ventricle, the segments remaining altogether intact. In advanced stages of most cases of chronic endocarditis myocardial degeneration occurs. It takes the form of fibroid change or fatty degeneration, or both. Aortic-valve involvement, especially when complicated with atheromatous change in the coronary arteries, is most prone to these forms of myocardial disease. Chronic endocarditis may be said, with the rarest exceptions, to persist until death, although Musser has reported two cases in which the murmur of chronic endocarditis disappeared during life. The effect of valvular deficiencies upon the several cardiac chambers and the muscular structure of the heart will be most advantageously studied when the individual lesions of the segments are considered.

Etiology.—There can be no doubt that most cases of organic heart-disease occurring in children and young adults are caused by primary *acute rheumatic endocarditis*; and, although the latter affection cannot in truth be said to terminate invariably in chronic endocarditis, it probably does in most instances. This result, in my opinion, is more frequent in children suffering from acute endocarditis than in adults. On the other hand, not a few cases of chronic endocarditis originate in a very mild grade of acute valvular inflammation, which may be, though itself mute, reinforced by a rheumatic diathesis. Indeed, acute endocarditis may be the sole expression of rheumatic disease. Not less than one half of all cases of organic valvular disease are caused by rheumatism, and more than one half of the total number occur between *twenty* and *thirty* years of age. Acute endocarditis complicating other acute infectious diseases than rheumatism (*e. g.* measles, chorea, pneumonia) may also be followed by the chronic variety; but it is quite questionable whether this occurs as frequently as in the case of acute endocarditis of rheumatic origin.

mild grade); or there may be a displacement of the mitral segments in the direction of the auricle, thus creating incompetency at this orifice. There is to be observed in many instances a marked degree of fatty degeneration of the papillary muscles, which also exhibit more or less flattening. Again, *secondary dilatation* may produce relative insufficiency at the mitral orifice. When incompetency has been established here, impeded pulmonary and general venous circulation, together with the secondary lesions in the left auricle, pulmonary vessels, and right ventricle that are characteristic of mitral incompetency, are the necessary result. The blood-current through the mitral ring may be retarded, owing to the simultaneous influx into the left ventricle from the aorta, thus causing pulmonary congestion without organic change in the segments.

Special Etiology.—(1) *Acute Endocarditis.*—Incomplete resolution of the acute form of endocarditis leads to progressive chronic valvular disease. In the young it is caused with comparative frequency by rheumatic endocarditis. Aortic regurgitation may also arise, though rarely, in the course of acute endocarditis, as, for example, when the latter is attended with destructive ulceration. Such instances usually terminate in speedy death.

(2) *Chronic Infectious Irritants.*—I have found syphilis to be a factor (though rarely the sole cause) in a considerable percentage of cases. Aortic regurgitation is a frequent complaint in sailors and soldiers, among whom it is worthy of notice that syphilis is particularly common.

(3) *Chemical Irritants.*—(*a*) *Uric Acid.*—In chronic and irregular forms of gout the irritating qualities of uric acid give rise to interstitial endocarditis and arterial sclerosis. It is quite probable that chronic rheumatism has a similar influence, though brought about in a somewhat different manner. (*b*) By favoring the accumulation of uric acid in the blood, *lead-poisoning* may be indirectly responsible for the disease. (*c*) *Alcohol* by its persistent irritant action may excite chronic valvulitis.

(4) *Augmented Aortic Tension.*—The excessive functional activity of the heart occasioned by the immoderate use of cardiac stimulants (alcohol) tends to raise the blood-pressure above the normal point, and thus sclerotic endocarditis may be developed very slowly. The effect of *occupation* in causing this disease, by increasing the vascular tension, is more notable than in the case of alcohol, though both of these factors are found not infrequently to be present in the same case. It is undeniably true that strong-bodied men in the middle period of life and engaged in such occupations as entail strain—"not a sudden, forcible strain, but a persistent increase of the normal tension to which the segments are subject during the diastole of the ventricle" (Osler)—are the most frequent sufferers from aortic incompetency.

(5) From personal observation I feel convinced that chronic endocarditis (affecting the aortic valves) may be secondary to *aortic endarteritis* as the result of direct extension. It must be borne in mind, however, that arterio-sclerosis is also often secondary to chronic valvulitis.

(6) Relative insufficiency is caused, in rare instances, by pronounced dilatation of the ascending portion of the arch near to the valve, or by an aneurysm just beyond the aortic orifice.

Among the more effective predisposing factors are *age* and *sex*. The disease occurs much more often in males than in females, chiefly on orifice. The cusps of the diseased aortic valves sometimes adhere to the intima of the aorta, and laceration of the semilunar segments, which are the seat of diseased processes (particularly ulceration), is sometimes found *post mortem*, and may be the chief factor in determining the development of the condition. This accident may, though rarely, occur as a result of a severe straining effort in the case of valves previously healthy. Occasionally, also, the principal factor in the production of this valvular lesion is a congenital malformation of the segments whereby they are rendered very prone to chronic endocarditis in consequence of the undue strain to which they are subjected. The lesions that give rise to stenosis may coexist with simple aortic incompetency, and, though the latter condition frequently occurs alone, stenosis is often combined with regurgitation.

Mechanical Influence of the Lesion.—The reflux current passes from the aorta backward through the imperfectly closed semilunar valve into the left ventricle during the diastole of the heart or while the left ventricle is being filled by the normal blood-flow from the auricle. It is clear that over-distention of the left ventricle must result at once from two simultaneous influx currents of blood, with a tendency to an increasing dilatation, especially since the lesion itself is steadily progressive. To expel the increased amount of blood from the left ventricle demands increased cardiac power, and the over-exertion causes *dilatation*, followed by a compensatory *hypertrophy*. Dilatation and hypertrophy of the left ventricle develop *pari passu* until the left ventricle reaches enormous dimensions, forming the *cor bovinum*, which weighs 1000 grams or more (30 to 50 ounces). Under these circumstances the arterial system is overfilled at each ventricular systole. In the very early stage the reflux of blood from the aorta into the ventricle tends to lessen the volume of the circulating medium in the arterial tree, but this depleting influence is successfully counterbalanced by the augmented column of blood thrown from the ventricle during cardiac systole. Hence the requirements for bodily nutrition are, for a longer or shorter time, satisfied. The abnormally large amount of blood that is thrown into the arteries with undue force subjects them to increased tension, and as a result arterio-sclerosis, leading sometimes to atheroma, is commonly developed, and presents its ulterior dangers (aneurysm, apoplexy). The coronary arteries are similarly involved, their caliber being reduced, and particularly at the point of origin. Soon or late the blood-supply to the heart-muscle may become inadequate, and nutritional disturbances now manifest themselves in fatty and fibroid degeneration of the cardiac muscles; these pathologic changes are attended with *secondary dilatation*, which soon predominates over the hypertrophy. The imperfect blood-supply to the ventricular tissue may be accounted for, in great measure, by the narrowed lumen of the coronary vessels, and also in part by the inelasticity of the walls of the latter and by the inefficiency of the aortic recoil. Furthermore, it is to be recollected that, in obedience to the laws of nature, overuse of any single group of muscles, while productive of marked hypertrophy in the first instance, is followed eventually by atrophy and loss of power. In consequence of the increased tension to which they are constantly subjected the *mitral leaflets* may become the seat of sclerotic endocarditis, and this may lead to the development of mitral insufficiency (usually of mild grade); or there may be a displacement of the mitral segments in the direction of the auricle, thus creating incompetency at this orifice. There is to be observed in many instances a marked degree of fatty degeneration of the papillary muscles, which also exhibit more or less flattening. Again, *secondary dilatation* may produce relative insufficiency at the mitral orifice. When incompetency has been established here, impeded pulmonary and general venous circulation, together with the secondary lesions in the left auricle, pulmonary vessels, and right ventricle that are characteristic of mitral incompetency, are the necessary result. The blood-current through the mitral ring may be retarded, owing to the simultaneous influx into the left ventricle from the aorta, thus causing pulmonary congestion without organic change in the segments.

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Among the more effective predisposing factors are *age* and *sex*. The disease occurs much more often in males than in females, chiefly on account of the fact that a greater percentage of the former than of the latter are engaged in occupations that are causally related to the disease. As to age, a preponderating proportion of the cases arise during advanced middle life, and a comparatively smaller number at a more advanced period than in young adult life.

Symptoms.—So long as the hypertrophy of the left ventricle successfully overcomes the otherwise injurious consequences of the valvular defect the harmonious balance of forces is maintained, and there is an almost entire absence of symptoms. I have observed, moreover, that compensation does not fail so early in young subjects as in those more advanced in years, or at a period of life when aortic incompetency is often a sequel of atheroma combined with hypertrophy and dilatation of the left ventricle. With the development of marked hypertrophy severe muscular exertion and strong mental excitement will, by exciting over-action of the powerful heart, bring on a train of symptoms as *throbbing headache*, *vertigo*, and *tinnitus aurium*. The clinical manifestations of arterial *anemia*, particularly of the brain, and also those of general *arterio-sclerosis*, frequently coexist. The patient's countenance exhibits *pallor*, and he complains of *headache*, *flashes* of *light* before the eyes, and *dizziness*. Dilatation of the peripheral vessels often leads to hot flushes and drenching sweats. Cases exhibiting the latter symptoms have been mistaken for *phthisis*. *Dizziness* is often distressing, and is most marked upon rising quickly from the recumbent to the erect posture. Shortness of breath may come on early, but this rarely happens except upon inordinate exertion or great mental excitement—conditions that cause strong cardiac action and prohibit the discharge of blood from the left auricle into the left ventricle, thus causing *pulmonary congestion*. Oppression in the precordial region and cardiac palpitation are commonly present, as is a *dull aching pain;* the most constant seat of the latter is the precordia, but it radiates not infrequently to the shoulders, and thence down the arms, particularly the left. Genuine *angina pectoris* may be a concomitant. I have seen instances of aortic regurgitation in which severe pain was located in the left shoulder-joint, the condition simulating rheumatism.

Following immediately upon **failure of compensation** the cardio-pulmonary circulation is retarded, and there is increased *dyspnea*, the latter symptom being greatly intensified by undue exertion and at night. There may be *cough*, and not rarely *hemoptysis*, though less frequently than in simple mitral disease. Later on, *general venous congestion* of a moderate grade follows pulmonary congestion, and the dyspnea now becomes severe. It is nocturnal, and often compels the patient to assume a semi-erect posture in bed. In the later stages the symptoms, particularly those of venous stasis as shown by cyanosis and malleolar dropsy, are due to mitral incompetency, followed by failure of compensation. Marked enlargement of the liver due to passive congestion may now ensue and give rise to the suspicion of a new growth. *Edema* of the feet rarely goes on to general anasarca. In aortic incompetency a higher grade of *symptomatic anemia* is reached than in any other cardiac lesion—a recent blood-count showing 2,800,000 red corpsucles to the c.mm. Hence slight edema of the feet may be due solely or in part to anemia. The intercurrence of acute endocarditis, as evidenced by prostration and irregular fever, is observed, and not infrequently as a terminal condition. The symptoms of cerebral, splenic, and renal *embolism* may arise. Probably ***sudden death*** ensues, as the result of involvement of the coronary arteries, with greater frequency in this than in all other forms of valvular disease combined; and yet this accident is by no means frequent. Instances of aortic incompetency, in which *nervous phenomena*, as peevishness, irritability, or melancholia, manifest themselves, are too common to be looked upon as mere coincidences. Many patients are led to commit suicide because of their cardiac lesion when other and erroneous explanations are given to account for their acts.

Physical Signs.—*Inspection* brings to light an enlarged area of the apex-beat; this is displaced downward, being visible in the sixth and seventh interspaces and to the left, and most marked between the mammary and anterior axillary lines. The entire precordial zone may be distended, particularly in young subjects, and the systolic pulsation is usually more or less heaving in character. The carotids throb forcibly, as do the temporals, brachials, and radials, though less violently. These abnormal pulsations are due chiefly to hypertrophy of the left ventricle, though frequent factors of lesser influence are associated—an arterio-sclerosis and a regurgitant blood-stream from the aorta into the left ventricle. The impulse becomes diffused and wavy with the progressive enfeeblement of the left ventricle, and venous pulsation due to tricuspid insufficiency may be associated with arterio-pulsation later in the affection. Epigastric throbbing may also be noticed, and on gently rubbing a spot upon the forehead an alternate paling and blushing appear (*Quincke's capillary pulse*); this may also be noted in the finger-nails. It is not peculiar to aortic insufficiency, however, and may be observed in cases of decided neurasthenia and in anemia. Very rarely the pulse-wave is propagated from the capillaries to the veins of the hand and back of the foot, giving rise to a visible venous pulsation. L. Webster Fox informs me also that the retinal vessels are often seen to pulsate in this disease.

On *palpation* a forcible heaving impulse is usually felt. When, however, dilatation predominates over hypertrophy, the impulse is weak and undulating. A diastolic thrill just to the left of the mid-sternum may be detected in many instances, and a presystolic thrill is also discoverable very rarely. The pulse is characteristic; it is quick, jerking, and full, but, upon striking the finger, recedes abruptly, and is known as the *Corrigan* or *water-hammer pulse.* This sudden collapse of the pulse is most decided when the arm is held in a vertical position. Its distinctive characters are not always appreciable after compensation is lost. A glance at the sphygmographic tracing will show a sudden rise and fall, with absence or delay of the secondary wave (*vide* Fig. 48).

Percussion.—Cardiac dulness is coextensive with the impulse, extending downward to the eighth rib, and to the left as far as, or even beyond, the anterior axillary line. Later, enlargement of the left auricle may cause dulness upward and to the left of the sternum. Enlargement of the right ventricle causes an increase of dulness to the right. When the dilatation exceeds the hypertrophy the area of dulness will be much extended transversely and slightly upward, the apex now being more rounded.[1]

On *auscultation* a diastolic murmur with its seat of greatest pronunciation is audible at, or a little below and to the left of, the aortic cartilage and is transmitted down along the left edge of the sternum; this is produced in the left ventricle. From the xiphoid it may be transmitted to the left as far as the spinal column. It may be heard in the vessels of the neck and, very rarely, in the radials. A. Borgherini affirms that the special direction taken by the regurgitant current determines largely the variable position of the murmur and the variable size of the heart. The *rhythm* of the murmur can be most readily determined by auscultating over the base, for while the pulmonic second sound is usually audible at the apex (the murmur appearing to follow it), it is not so when, as sometimes happens, the murmur is quite loud. The first sound is often dull, indefinite, and widely diffused, owing to hypertrophy of the left ventricle. In *quality* this murmur is usually soft, blowing (long-drawn), and frequently musical; sometimes, however, it is somewhat rough and loud. *Associated murmurs.*—In most instances a systolic murmur, brief and harsh in character and transmitted into the vessels of the neck, is also discovered over the aortic region (*double aortic*). The presence of the murmur with the first sound is not diagnostic of actual aortic stenosis. It is more often due to a mere roughening of the semilunar segments or of the intima of the aorta. In advanced cases a soft systolic murmur is commonly heard at the apex; it is readily distinguished from the diastolic murmur by its rhythm, and is occasioned in most instances by a relative mitral incompetency. Still another murmur, of rare occurrence, is rolling in character and generally presystolic in time, and may be heard at the apex over a limited surface-area. This may be accounted for by the presence of excessive dilatation of the left

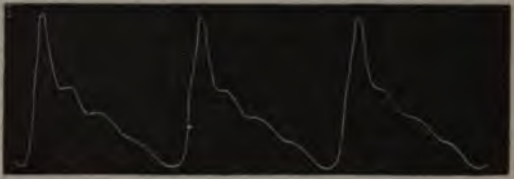

FIG. 47.—Normal pulse-tracing.

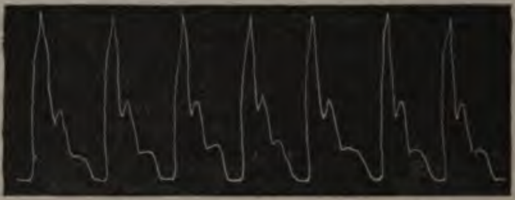

FIG. 48.—Pulse-tracing in a case of aortic regurgitation (William Hoffman).

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[1] A dilated aorta with thickened walls—a condition sometimes associated with aortic regurgitation—may also give rise to abnormal dulness under the manubrium and to the left of the sternum.

ventricle, in consequence of which the mitral leaflets must remain free in the blood-stream during the diastole, and here they set up vortiginous movements that cause the presystolic (Flint) murmur. Duroziez discovered a double murmur in the arteries (femoral), which is quite frequently present, but this may be noted occasionally in the absence of aortic regurgitation. Traube has described another arterial phenomenon —a systolic sound in the leg, somewhat resembling a heart-sound, but exceedingly short and sharp. It is probably due to sudden systolic distention of vessels that were previously empty.

The **diagnosis** demands the presence of a diastolic murmur, the signs of left ventricular hypertrophy, the peculiar arterial pulsations, and the characteristic water-hammer or Corrigan pulse. The diastolic murmur may be absent.

For the *differential diagnosis* see Aneurysms of the Arch, Hypertrophy, Dilatation of the Heart, etc.

AORTIC STENOSIS.

Definition.—A narrowing or stricture of the aortic orifice, due to thickening or adhesion of the valve-segments, and causing an obstruction to the flow of blood into the aorta.

Simple aortic stenosis may be met with, though it is a great rarity. Its development is soon followed by more or less valvular incompetency, and hence these affections often coexist. It may be secondary to aortic insufficiency; but this is rare, the conditions in the latter disease being unfavorable to the development of the former.

Special Etiology.—Rarely rheumatic endocarditis, and still less commonly other forms of acute endocarditis, cause union of the semilunar segments, with resulting stenosis. The most common immediate causative factor is a *slow sclerosis of the aortic valve*, followed by calcareous deposits. The more or less immobile, rigid valves obviously narrow the aortic orifice and oppose a barrier to the outflowing blood-current from the left ventricle into the aorta. The aortic ring may be the seat of changes similar to those just described, resulting in a moderate grade of stenosis, though the leaflets themselves remain intact. The lesions are most frequently to be regarded as a part of the general process of arterial sclerosis, which is most marked in the region of the thoracic aorta; and sometimes, as Peter contends, they are distinctly secondary to sclerotic changes at the root of the aorta. The coronary arteries may be the seat of changes similar to those noted in aortic regurgitation. The condition is also rarely congenital. *Males* who have reached *advanced years* are especially prone to aortic stenosis, for the reason that atheromatous processes belong peculiarly to that sex and period of life.

Mechanical Influence of the Lesion.—To propel the normal volume of blood through the constricted aortic orifice requires increased strength on the part of the left ventricle, and, as a consequence, the latter hypertrophies. This hypertrophy develops very slowly, and keeps pace with the progress of the valvular lesions. The undue ventricular tension sometimes induces more or less sclerotic change in the mitral valves. Hypertrophy of the left ventricle eventually gives way to extreme dilatation, and also to relative mitral incompetency with its unfavorable influence, first upon the pulmonary and, secondly, upon the general venous circulation.

Symptoms.—The symptoms date from the commencement of failure of compensation, often many years after the onset of the disease. Their first appearance will be found to follow some unusual muscular effort or the operation of some depressing influence, as the too free use of tobacco or alcohol. They are due to disturbances of circulation arising from a gradual secondary dilatation of the left ventricle, which is now unable to propel the normal quantity of blood into the arterial tree. Hence *anemia*, especially of the brain and peripheral parts of the body, becomes pronounced, and is evidenced by such symptoms as *syncope, dizziness, headache*, and *pallor*. Since aortic incompetency usually manifests itself secondarily, the clinical features of both affections are sooner or later variously commingled. In cases in which mitral lesions develop they are overcome by compensatory enlargement of the right ventricle: the latter chamber may at a later period become dilated, in which event tricuspid regurgitation and the symptoms of general venous engorgement appear. As in the case of aortic regurgitation, so in an aortic constriction, *slight edema* of the feet is common as a terminal symptom; marked dropsy, however, is uncommon. From the fibrous deposits on the segments, as well as from any small clots behind the valves, *emboli* are apt to become dislodged by the forcible blood-stream. and be conveyed to the brain (cerebral embolism), to the spleen (splenic embolism), to the kidneys (renal embolism), or to other organs.

Physical Signs.—*Inspection.*—The apex-beat is gradually displaced downward and to the left, owing to left ventricular hypertrophy. It is, as a rule, slow, forceful, and heaving, but less frequently it may be lacking in strength. It may be enfeebled, diminished in area, or even absent, owing to associated emphysema.

Palpation discloses the forcible and heaving impulse-beat, unless emphysema be present, when the heart and its movement may be concealed and the apex-beat become impalpable. A marked systolic thrill, with the seat of greatest intensity in the aortic region, is quite generally present. I have frequently felt this thrill in the apex region, though not so intensely as at the base. The pulse, in this disease, is small, regular, not compressible, and of normal or slightly lessened frequency. The sphygmographic tracing shows slowness of the ascending curve and a gradual formation of the descending line (*vide* Fig. 49).

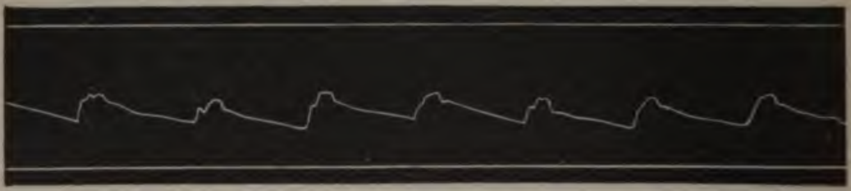

FIG. 49.—Sphygmogram of aortic stenosis, from a man aged sixty years.

when the right heart fails a lessened amount of blood reaches the left ventricle, and hence an abnormally small amount finds its way into the aorta; this fact explains the presence of the low arterial tension late in the disease. Hypertrophy of the left ventricle in this disease has also been attributed in part to the augmented tension in the general capillary vessels that is occasioned by the venous stasis.

Special Etiology.—(*a*) *Rheumatic endocarditis* is the most frequent cause, though mitral regurgitation also results less frequently from acute endocarditis due to other causes. (*b*) It may be a part of a *general arterio-sclerotic process*, this group of cases being caused, not rarely, by syphilis and alcohol. (*c*) *A diseased condition of the columnæ carneæ* or *chordæ tendineæ*, if it contracts them or weakens their structures so that the free edges of the segments pass beyond the plane of the orifice, produces insufficiency. (*d*) It rarely arises in the course of *aortic valvular disease* (a secondary mitral affection), and is then excited mainly by undue tension of the blood in the left ventricle. Here the lesion is of a mild grade, as a rule. (*e*) It is frequently occasioned by *enlargement of the left auriculo-ventricular ring*, resulting from excessive dilatation of the left ventricle, as in aortic incompetency, aortic stenosis, long-continued fevers, and the graver anemias (relative incompetency). (*f*) *Ulcerative endocarditis*, either by perforating or producing rupture of the valve-curtains or by destroying the chordæ tendineæ, may bring about mitral incompetency. Among *predisposing factors* age and sex are worthy of special mention, the incompetency occurring with greatest relative frequency in young adults (from twenty to thirty years of age, according to Ashton's figures), and somewhat more commonly in males than females.

Symptoms.—*During Compensation.*—In healthy persons the compensatory forces keep pace with the valvular lesions for an indefinite and usually lengthy period, during which time there may be an entire absence of symptoms. When present they are dependent upon disturbances of the cardio-pulmonary circulation that are occasioned by trivial causes, such as excitement, going up stairs, or other forms of active physical exertion. Under these circumstances the force of the regurgitant current is increased (by the hypertrophied left ventricle), thus producing more or less *pulmonary congestion* that may proceed to edema of the lungs or hemoptysis. The condition is usually a temporary one, and is attended by *dyspnea*, *palpitation of the heart*, a *short, hacking cough*, and *expectoration* of a frothy serum that may be blood-stained. The relation existing between the severity of the dyspnea and the degree of active physical exertion is positive and vital. Shortness of breath may be the sole feature during a long period. The rational symptoms rarely warrant a suspicion of the existence of mitral disease until compensation has failed, but the patient's appearance often indicates heart-disease. The *face* is pale and the features peaked, the eyes, lips, and ears are dusky, and the minute vessels of the cheeks are prominent. Clubbing of the finger-nails is observed most frequently in the young.

After Failure of Compensation.—Failure of compensation implies failure of the right ventricle to cope efficiently with the augmented tension in the pulmonary circulation, with accompanying congestion of the lungs, followed by engorgement of the systemic veins. The latter process begins at the right heart and proceeds toward the periphery, involving the viscera, mucous membranes, and extremities until it is universal. The *pulmonic symptoms* above detailed are now more marked, particularly the dyspnea (which may be constant), cough (with expectoration of alveolar epithelium containing brown pigment-granules), and cardiac palpitation with arrhythmia. *Pain* is rare unless stenosis coexists. *General venous engorgement* manifests itself by an enlargement of the liver and of the spleen, in the features of gastro-intestinal catarrh, in hemorrhoids, in marked cyanosis of the surface, and in the passage of a scanty albuminous urine containing tube-casts and blood-corpuscles. Dropsy follows, beginning in the feet and progressing upward, until finally the trunk and the serous sacs are involved. By stimulation the heart may be reinforced, and all of the unfavorable symptoms disappear in consequence, but this is not for long, as a rule. I have at present under observation a case in which not less than half a dozen instances of broken compensation have occurred at intervals of six to eight months, all of which have been successfully overcome.[1] In all cases, however, there comes a time when compensation cannot be restored, and the end is reached by an uninterrupted downward course.

Physical Signs.—*Inspection.*—The precordia is prominent, particularly in children, and the area of the apex-beat is enlarged, later becoming diffuse and wavy. It is carried to the left and downward, corresponding with the degree of hypertrophy of the left ventricle. A pulsating epigastrium is in frequent association, particularly after dilatation of the right ventricle appears. With the failure of the right heart also come wavy pulsations in the cervical veins, and occasionally a mild grade of jaundice.

Palpation sometimes discovers a thrill at the seat of the apex-beat, that is synchronous with the first sound. The impulse during the stage of full compensation is forceful and heaving, but with the beginning of failure of compensation it grows feeble and irregular, and late in the affection is excessively weak and arrhythmic. The pulse bears a definite relation to the apical impulse; it is commonly regular and full during the compensatory period (though at times the tension is slightly lowered), but becomes small, easily compressible, and exceedingly irregular during the period of broken compensation. One meets with cases in which irregularity appears during the period of fair compensation.

Percussion.—The dull area is increased to the left, extending frequently to the anterior axillary line; and also to the right, frequently from ½ to 1 inch (1.2–2.5 cm.) beyond the right sternal margin. Dilatation of both ventricles exerts a widening influence; hence cardiac dulness is increased more laterally than vertically.

Auscultation reveals a systolic murmur, with greatest intensity at the apex (see Fig. 50). Occasionally, this murmur is also conducted to the tricuspid and pulmonary valves. It is rarely loudest in the fourth or third space in the vertical nipple line. Balthazar Foster first called attention to the fact that the murmur of mitral regurgitation may be loudest at the base of the heart, and at times audible only in that situation—an occurrence that has since been confirmed. It is sometimes audible in the recumbent posture and inaudible in the erect. From the apex it is transmitted to the left as far as the angle of the scapula, with progressively diminishing clearness. It has a blowing quality, and frequently ends in a musical tone. Loudness implies strength of contraction (Broadbent). Over the third left costal cartilage, and frequently at the apex, there is heard the accentuated pulmonic second sound, due to the increased tension in the pulmonary vessels that is engendered by the hypertrophy of the right ventricle. *Combined murmurs* may be heard, and not infrequently a rough, rolling, or rumbling presystolic murmur is detected. A frequent late occurrence is the secondary dilatation of the right ventricle, causing relative tricuspid insufficiency with its characteristic soft, low-pitched, systolic murmur, heard best at the ensiform cartilage. A spurious diastolic murmur may be noted, though rarely, when the sounds are timed with the pulse. This is due to a weak systole that fails to cause a radial pulse.

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[1] Neglect of hygienic precautions and intercurrent complaints of various sorts often determine the occurrence of failure of compensation.

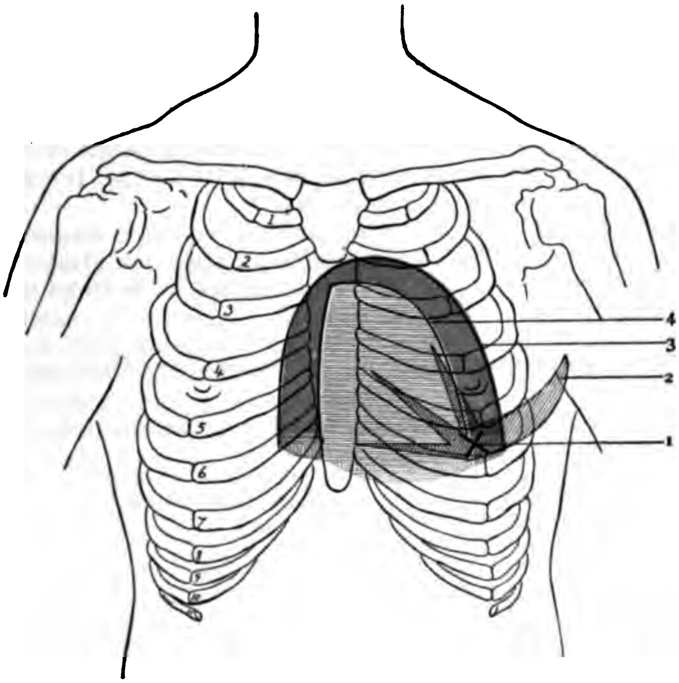

FIG. 50.—1, Seat of greatest intensity; 2, direction of chief transmission; 3, boundary line of relative dulness; 4, boundary-line of absolute dulness (modified from Sahli).

Diagnosis.—In the presence of the following group of features the diagnosis is set at rest: A marked broadening of the area of cardiac dulness; a systolic, *apical* murmur that is conveyed to the left axilla and may be heard even at the back; and a decided accentuation of the pulmonary sound. Obviously, the latter sound becomes feeble after dilatation of the right ventricle has occurred. A systolic thrill is of the highest diagnostic importance, but is unfortunately absent in perhaps a majority of the cases. Free regurgitation through the mitral orifice may be safely inferred when the following signs are concurrent:

(*a*) An absence of the sound of mitral-valve tension, a murmur replacing the first sound; (*b*) accentuation of the pulmonic second sound; (*c*) an enlarged area of the left cavity; (*d*) an enlarged area of the right cavity (Sansom).

Differential Diagnosis.—There are two organic lesions of the heart that are sometimes mistaken for mitral incompetency, since both are accompanied by a systolic murmur—the one *aortic stenosis*, and the other *tricuspid regurgitation.* How to distinguish mitral from tricuspid incompetency is a question that will receive due attention when the latter disease is considered. *Aortic stenosis* generates a systolic murmur, but it is loudest over the base, and is transmitted through the great vessels of the neck; while the mitral systolic is most intense over the apex and is transmitted far to the left. In mitral incompetency the pulmonary second sound is accentuated; in aortic stenosis it is not. In mitral incompetency both ventricles are enlarged, as shown by percussion and other signs; in aortic stenosis the left is chiefly enlarged during almost the entire course. In mitral incompetency a thrill, most marked over the apex-beat, may be felt; in aortic stenosis a thrill, rough and having its chief seat at the base, is common. Additional points of distinction are furnished by the contrasting factors of the pulse, the age of the patient, and other etiologic influences.

Functional and other harmless murmurs are often confounded with mitral insufficiency. The considerations on which the greatest dependence is to be placed in the differentiation are to be found in the subjoined table:

MITRAL INCOMPETENCY.	FUNCTIONAL AND HARMLESS MURMURS.
History.	
Previous history of rheumatism or other disease causally related.	History of causative factors of one or other form of anemia, of debility, or of Graves' disease.
Frequently there is definite knowledge of rheumatism and organic heart-disease, in combination in the same individual.	No such association.
Physical Signs.	
Inspection.—Dusky lips, ears, etc.; later wavy pulsation in veins of neck.	Pallor of skin and mucous surfaces common.
Palpation.—Finger-tips placed over apex-beat forcibly lifted. Pulse-tension somewhat lowered and not prolonged. Impulse displaced.	Finger not lifted by the impulse, which often cannot be felt. Pulse-tension prolonged and arterial pressure increased generally. Impulse not displaced.
Percussion.—Evidence of dilatation of both ventricles.	Dilatation of right auricle, but only in about one-half of the cases, giving rise to dulness above or to the right of the right edge of sternum.
Auscultation.—A systolic apex-murmur (often musical), with characteristic area of transmission. This murmur, unlike the functional, is often heard behind, between the spine and the scapula.	Soft systolic murmur at apex (often late—systolic rarely transmitted to axilla), usually preceded by or associated with a basic systolic murmur and a venous hum in the veins of the neck.

To differentiate the murmur of *relative mitral incompetency* is difficult, though in many instances it can be accomplished with reasonable certainty. It rests upon two points: (*a*) the character of the murmur, which is, as a rule, softer and less intense than that due to valvular lesions; and (*b*) the antecedent history of the patient. Thus, relative insufficiency of the mitral segments probably exists in patients in the middle period of life, in whom the previous history furnishes such etiologic factors as chronic gout, syphilis, or alcoholism; or in persons who exhibit arterio-sclerosis or organic disease of the aortic valve and an apex-systolic murmur. On the other hand, if the signs of mitral regurgitation occur in a younger subject or in one who has been afflicted with acute rheumatism, it is highly probable that the mitral-valve segments are the seat of chronic endocarditis of rheumatic origin. Again, if present in chronic *renal* disease, with concurrent symptoms of high arterial tension and of left ventricular hypertrophy—accentuation of the aortic second sound, a mitral systolic murmur—it is to be ascribed to relative insufficiency. *Compression of the edge of the left lung* by the ventricular systole may produce a spurious murmur. I believe that a rare sequel of mitral incompetency is mitral stenosis, owing to the contraction of the mitral orifice, with, in some instances, cohesion of the free edges of the cusps.

MITRAL STENOSIS.

Definition.—Constriction of the left auriculo-ventricular orifice, due to either thickening or adhesion of the segments. With few exceptions adhesions of the free borders of the valve or of the chordæ tendineæ obtain.

Special Pathology and Etiology.—It is to be recollected that the constriction may be almost inappreciable, and yet an uneven, roughened surface be presented, producing a murmur as the blood-stream enters the ventricle; on the other hand, a high degree of constriction may be encountered. Thus, in the *funnel-shaped* form of mitral stenosis the aperture may be so small as scarcely to admit the passage of a goose-quill. When moderate in degree the tip of the index finger is admissible; in the *button-hole* form the slit may be so narrow as not to allow an object larger than a shirt-button to pass through it. The *funnel* variety is common in children, and is occasionally a congenital condition (possibly hereditary), while the button-hole variety is comparatively rare in childhood. In adults, however, the funnel-shaped constriction is rare, while the button-hole valve is quite common; in 62 *postmortem* examinations only 3 showed funnel-form contraction (Hayden and Fagge). Mitral stenosis is, as a rule, dependent upon a mild or limited endocarditis that is usually of rheumatic origin. It is more common in *young adults* and in *children* after the fifth year than in older persons, and a greater incidence is shown in *females* than in males, for the reason that the affections that are causally related to endocarditis are more frequent in females (rheumatism, chorea, chlorosis). The endocarditis of measles and scarlatina may also lead to narrowing of the mitral orifice, and I quite agree with Osler in the belief that whooping-cough, owing to the great strain that it imposes upon the heart-valves, may be account-able for certain cases. In adults *arterio-sclerosis* and *chronic nephritis* may act as causes. Ball-thrombi have been found in the auricle.

Mechanical Influence of the Lesion.—On account of the obstruction of the blood-stream at the mitral orifice during diastole the task of the left auricle becomes greater than normal, and in consequence of this its walls hypertrophy. They may be found to be one-fourth or even one-half inch (1.2 cm.) in thickness, the normal thickness being only three-twentieths of an inch (3.7 mm.). Dilatation of the auricle comes on early, since this chamber cannot take on much hypertrophy owing to lack of muscular structure, and in the later stages its walls become extremely thin. For a varying period of time the increased power due to hypertrophy of the left auricle and the increased resistance to the circulation that is the result of the mitral lesion are exactly balanced. At a comparatively early period, however, the auricle can no longer maintain this equilibrium; and then, owing to retardation of the current from the pulmonary veins to the auricle, the vascular tension in the lungs and right ventricle is increased. The right ventricle, in seeking to overcome the obstruction, becomes greatly hypertrophied and dilated, and late in the disease tricuspid incompetency supervenes with its usual sequences. The hypertrophy of the latter chamber counterbalances the lesion during the greater part of the period of compensation. For a brief time the left ventricle exhibits no abnormal proportions. Later and at autopsies its cavity is found smaller and its walls thinner than the normal, these conditions being due to its abnormally light labor. The apex of the heart is formed almost exclusively by the greatly enlarged right ventricle. If the left ventricle be hypertrophied, it is owing to the existence of associated mitral incompetency.

Symptoms.—The subjective symptoms are scanty and of slight value in forming the diagnosis. During the period of full compensation there may be an entire absence of symptoms except on going up stairs or on attempting some unusual muscular effort, when *dyspnea* appears. Fragments of fibrinous coagula dislodged from between the musculi pectinati of the auricle or swept from the valves may give rise to the phenomena of *cerebral embolism* (aphasia and hemiplegia). The same conditions may arise, and in the same way, from recurring endocarditis, to which such patients are specially liable. The patient in well-marked cases presents an *anemic* appearance: a *stitch-like pain* in the apex-region is frequently present, and active exertion, by overtaxing the left auricle, induces *cardiac palpitation* and *dyspnea*.

After failure of compensation the symptoms referable to the pulmonary system are almost identical with those manifested in mitral incompetency. Owing to the pulmonary engorgement the *dyspnea* is constant, and is increased by exertion. After severe or prolonged physical exercise *congestion*, followed by *edema* of the lungs, may supervene, attended by a copious blood-stained, serous *expectoration*. True *hemoptysis* may arise from time to time. The increased tension in the pulmonary vessels being practically constant, sclerosis, followed by atheromatous degeneration of their walls, is a frequent occurrence, and may accidentally result in *pulmonary apoplexy*. Intercurrent *febrile attacks* (due usually to recurring endocarditis) are common, particularly in the later stages, and are attended with marked aggravation of the circulatory disturbances.

Mitral stenosis differs from mitral incompetency in that *general anasarca* is rare, though marked enlargement of the liver and other evidences of portal congestion (including ascites) are commonly present.

Physical Signs.—*Inspection.*—The apex-beat is not displaced unless there be excessive enlargement of the right ventricle or associated hypertrophy of the left. There is usually present a visible pulsation in the second left intercostal space, and sometimes in the third and fourth interspaces, occasioned by increased tension in the pulmonary artery; and there is also a diffuse impulse along the right border of the sternum. Epigastric pulsation is common. A prominence having its seat over the fifth and sixth left costal cartilages and the lower half of the sternum is observed, particularly in children. After failure of compensation the impulse is feeble and undulates, with engorgement and pulsation of the jugular veins.

Palpation discovers a presystolic thrill in a great proportion of cases. In certain instances active physical exertion may render this appreciable, or when in the recumbent posture on the left side the elevation of the arms may accomplish the same result. It is, however, absent in rare instances before failure of compensation occurs, and more frequently by far after the latter event. This fremitus is best felt over the third and fourth (less frequently the fifth) interspaces, just within the nipple, and during expiration. It commences after the second sound (during the diastole) as a purring fremitus, increasing steadily in volume and intensity, and terminates abruptly with the severe shock of the new impulse. The fremitus and systolic shock are pathognomonic, and may be relied upon in the absence of the murmur. The heart's impulse is most forcible over the lower portion of the sternum and along the right border, being due to the enlarged right ventricle; in a smaller proportion of cases, in the third, fourth, and fifth interspaces to the left of the sternum. The radial pulse is small, compressible, and markedly irregular as the propulsive power of the right ventricle diminishes. The sphygmographic tracing is notably irregular (*vide* Fig. 51).

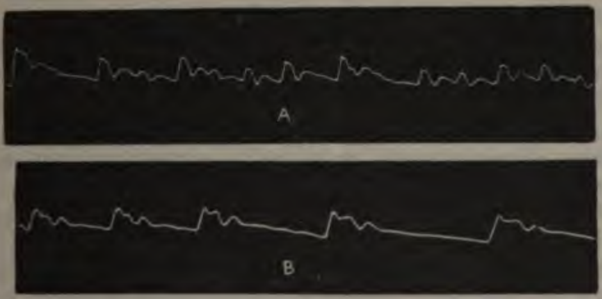

FIG. 51.—Sphygmograms in a case of mitral stenosis treated by extract of convallaria, and subsequently by digitalis: *A*, before treatment, showing the interpolated pulsations; *B*, after treatment (Sansom).

Percussion shows an extension of heart-dulness to the right, frequently 5 centimeters (2 inches) beyond the sternal margin, as a result of hypertrophy of the right ventricle, and upward as high as the second rib on either side of the sternum. Increase in the cardiac dulness to the left also occurs not infrequently, and is attributable to excessive enlargement of the right ventricle, though more often of the left ventricle in consequence of associated mitral insufficiency.

Auscultation reveals a rough, presystolic murmur, which may be characterized as churning or rolling, acquiring increased intensity. It occurs synchronously with the thrill. Its point of greatest pronunciation is just above and about one inch within the normal apex-beat. The area of transmission is generally quite limited, not exceeding a couple of inches in any direction. Griffith, however, has shown that the murmur is not seldom widely transmitted. This murmur sometimes exhibits atypical characters: it may be brief and low-toned, and may be audible on one occasion and then disappear for a considerable period. After the right ventricle becomes weak, the murmur may absent itself either temporarily or permanently, and lose its peculiar termination. In most cases the clear, accentuated first sound is retained, even though the murmur disappears. Improvement in the muscular power of the heart as the result of judicious treatment may cause the murmur to reappear, and I have seen such an occurrence in a case associated with mitral incompetency at the Philadelphia Hospital. For purposes of diagnosis, nothing is so vitally important as the time or rhythm of the murmur, and in his examination the observer must therefore palpate the heart, and not the radial pulse, while practising auscultation. The finger as well as the ear will thus become sensible of the systolic shock which replaces the cardiac impulse, and it will be noted that the murmur terminates at the same moment. In cases in which the impulse cannot be felt the finger should be placed over one or other carotid, since here the pulse is practically synchronous with the systole. In most cases the murmur occupies only the latter half of the diastole, though occasionally the whole of it. In some cases it is heard in the middle of the long pause. Owing to the presence of right ventricular hypertrophy the pulmonic second sound is greatly accentuated, being distinctly audible at the apex, while the aortic second sound is often absent or feeble. Reduplication of the second sound is not rare, and is characteristic.

Secondary Murmurs.—As previously pointed out, the murmur of mitral stenosis may succeed that of mitral incompetency, but this is comparatively rare. Mitral stenosis may follow aortic valvular disease, but in the vast majority of instances it is a primary affection. Secondary murmurs are not uncommon, however. Among these the bruit of *mitral incompetency* is relatively frequent. After compensation is ruptured the murmur of *tricuspid insufficiency* usually becomes audible at the lower end of the sternum and persists until the end.

In so-called "*relative mitral stenosis*" associated with primary dilatation of the left ventricle, which holds the orifice open, there occurs also a mitral regurgitant murmur, while that of stenosis may be absent.

Diagnosis.—The distinctive features of mitral stenosis are—(1) A presystolic thrill at the apex. (2) An increase in the precordial dulness upward and to the right. (3) A murmur which (*a*) has its seat above, yet near, the normal apex-beat; (*b*) is usually localized; (*c*) is presystolic in time, terminating abruptly with the systolic shock (sharp impulse); and (*d*) is rough and vibratory in character. (4) A marked accentuation of the pulmonic second sound.

Differential Diagnosis.—When the murmur of mitral stenosis is very brief, it is difficult to eliminate a *mere roughening* without valvulitis. In the latter condition, however, there is no increase in intensity of the murmurs on exertion or when the arms are uplifted, they are not vibratory in character, and there is no right ventricular hypertrophy. From simple mitral stenosis the lesion of *mitral incompetency* is easily distinguished by its systolic rhythm, greater area of transmission, and by the soft, more blowing character of its murmur. A combination of the two lesions, however, is a more frequent occurrence than that of pure mitral stenosis; and under such circumstances it is with great difficulty that the two murmurs are separated. The presence of the systolic murmur is distinguishable by its synchronism with the impulse or carotid pulse, and by its area of transmission to the left as far as the axilla. If now the stethoscope be applied just above and to the right of the normal apex, a limited superficial area will be found where a presystolic murmur is distinctly heard. Points can also usually be found where a continuous bruit, covering a portion of the period of diastole and of systole, is audible.

In *aortic regurgitation* the presence of a presystolic thrill and murmur has rarely been recorded, and Fisher, Phear, and others have called attention to their presence in adhesive pericarditis as well as in simple dilatation. When, as is usual, a purely diastolic murmur is also present in the aortic area, together with strong correlative evidence of aortic regurgitation, the diagnosis of mitral stenosis must be made with extreme caution and reserve.

TRICUSPID INCOMPETENCY.

(*Tricuspid Regurgitation.*)

Definition.—An imperfect closure of the tricuspid valve, due either to a dilatation of the right ventricle that is secondary to mitral or lung-disease, or, less frequently, to an inflammatory shortening of the valves.

Pathology and Etiology.—As a primary disease tricuspid incompetency is rare. It, however, is not uncommonly due to chronic organic changes, though originating in fetal endocarditis. After birth this variety is most common during childhood, and the frequency of occurrence is in inverse ratio to the age. At any period of life, however, chronic affections of the lungs or organic disease of the left side of the heart may, by augmenting the tension in the right ventricle, produce chronic interstitial changes in the tricuspid segments. These latter, however, are usually of mild grade. I have observed in autopsied cases of chronic bronchitis associated with emphysema, and in pulmonary tuberculosis, that the chief reason why extensive lesions of these valves are seen so rarely is to be found in the fact that dilatation of the right ventricle is soon followed by relative insufficiency, and thus the strain is in great part removed from the valves themselves. And yet, according to the statistical studies of Byron Bramwell, the tricuspid valve is implicated in 50 per cent. of all cases of acute endocarditis, notwithstanding the rarity of sclerosis of these segments. He suggests that the acute form frequeutly results in cure because of the relatively diminished right intraventricular tension. In rare instances one of the leaflets has been ruptured by straining. The relative tricuspid insufficiency, produced in a manner analogous to relative mitral insufficiency, is an exceedingly common secondary condition in affections of the lungs and heart that cause hypertrophy and dilatation of the right ventricle (mitral incompetency and stenosis, emphysema, sclerosis of the lung).

Physiologic Pathology.—In tricuspid leakage every systole of the right ventricle is accompanied by a reflux of venous blood through the imperfectly closed tricuspid orifice into the auricle, and thence into the veins. This causes venous stasis and gives rise to visible pulsation, and in this manner the engorged pulmonary circulation is relieved to some extent. A necessary unfavorable consequence, however, on account of the reflux current from the right ventricle, is the lessened blood-supply to the pulmonary arteries, even though the latter are found to be engorged. The already hypertrophied and dilated right heart now undergoes further enlargement in the same manner as in the hypertrophy of the left ventricle following mitral incompetency, though not to the same extent. In mitral incompetency the right ventricle compensates the mitral lesion after failure of the left auricle, but there can be no such effective compensatory mechanism after failure of the right auricle in tricuspid incompetency, since the right heart is not reanimated by a fellow as is the left. The blood-stream flowing into the right ventricle during the period of diastole, however, is abnormally large, owing to moderately increased tension. When the right ventricle fails to maintain the pulmonary circulation, progressive dilatation of its chamber occurs, with a proportionate thinning of its walls until its dimensions are enormous.

Symptoms.—In most instances the indications of the primary or causal affection must be noted, though these are often more or less screened by the more characteristic features of the disease under consideration. The symptoms of tricuspid incompetency point to passive congestion of the lungs and engorgement of the systemic veins, and they have been described in connection with mitral lesions. *Cardiac dropsy* is common, though present in by no means all cases. Frederick Taylor[1] contends that *ascites* is absent frequently, because the liver acts as a diverticulum to accommodate the excess of venous blood.

Physical Signs.—*Inspection.*—Venous pulsation, caused by the backward blood-wave from the right ventricle at each systole, is a pathognomonic sign. It is confined to the lower portion of the jugular veins so long as the valve that lies above the jugularis remains closed, but soon this yields, and then the veins seem to pulsate through their entire course with each cardiac systole. This is best seen when the patient is in the semi-recumbent posture, and is more marked in the right than in the left side. The subclavian and axillary veins may also be seen to pulsate, but rarely. The veins appear to be everywhere engorged, producing a cyanosis that is more noticeable if the breath be held when in full respiration. Tricuspid incompetency may be shown by pressing on the vein with the finger rather firmly, commencing just above the clavicle and passing upward, thus emptying it of blood. If, now, the right ventricle be capable of producing a return wave sufficiently powerful to overcome the valve in the external jugular, pulsation is seen to take place—also from below—in the vessel slowly and increasingly until the vein, as far as the point compressed, becomes filled. The vein fills "by jets synchronous with the heart-beat" (Sansom). Again, an impulse may be communicated to the jugulars from the underlying carotid artery; if this be the true explanation in any given case, the light pressure upon the vein below does not arrest the pulsation above, as is the case in tricuspid incompetency. Not rarely there is noticeable a feeble presystolic venous pulse, due to the weaker contraction of the right auricle as compared with that of the right ventricle (*anadichrotic venous pulse*). The area and seat of the apex-beat vary with the nature of the primary affection: in mitral incompetency, for example, the beat is displaced to the left and downward, while in uncomplicated mitral stenosis no appreciable displacement occurs. To the right of the sternum an undulatory pulsation is seen, due to contraction of the right auricle and ventricle, but this is not characteristic, since it may take place in simple mitral stenosis without tricuspid regurgitation. Epigastric pulsation is almost invariably observed.

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[1] *Lancet*, Nov. 22, 1890, p. 1126.

Palpation detects the heaving impulse of the right ventricle in the upper epigastric region. Rhythmic expansile pulsation of the veins of the liver is quite diagnostic and is usually detectable. To obtain this sign the patient should lie on the back with the arms raised, and the examiner should place the palm of his left hand over the right midaxillary region, and that of the right hand over the upper abdominal region. He will thus be enabled to feel an expansile pulsation of the liver synchronously with the ventricular systole. This is to be carefully distinguished from mere systolic depression of the organ due to the impulse of an enlarged right ventricle, transmitted through the diaphragm and left lobe of the liver to the epigastrium.

Popoff and others have also noted an inequality in the radial pulses in tricuspid regurgitation. This is probably due to the pressure of the enlarged auricle.

Percussion.—The extent and form of precordial dulness are variable according to the nature of the causative disease, but a dulness extending far beyond the right edge of the sternum is especially characteristic.

Auscultation.—A systolic murmur having its seat of greatest intensity at the base of the ensiform cartilage (*vide* Fig. 52) is almost constantly audible. The area in which it is best heard varies according to the intensity of the murmur. It is clearly conveyed to the left one inch beyond the left sternal margin, and to the right and upward for an equal distance beyond the limit of cardiac dulness. It is soft in character, short, and often faint. If the heart be weak, it may be absent. Additional murmurs, due to primary lesions, are often heard, and usually at other orifices. The pulmonic second sound is accentuated.

Diagnosis.—I believe that the most valuable symptom for diagnosis is the venous pulse, whether observed clearly in the neck or determined positively by bimanual palpation of the liver, as before described. Either of these signs alone suffices. The murmur is generally audible, and when so is a most valuable aid to the diagnosis. The **differential diagnosis** between mitral and tricuspid regurgitation is easy when either exists alone, if it be remembered that the seat of greatest pronunciation, the area of transmission, and the acoustic character of the respective murmurs are widely different. But it is extremely difficult to discern precisely a faint tricuspid murmur when it develops secondarily to the more pronounced murmur of mitral incompetency. If a careful observation of the murmur fails to establish the diagnosis of tricuspid insufficiency, as sometimes is the case, absolute reliance should, in my opinion, be placed upon the venous pulse when present, and the absence of the latter sign should exclude this disease.

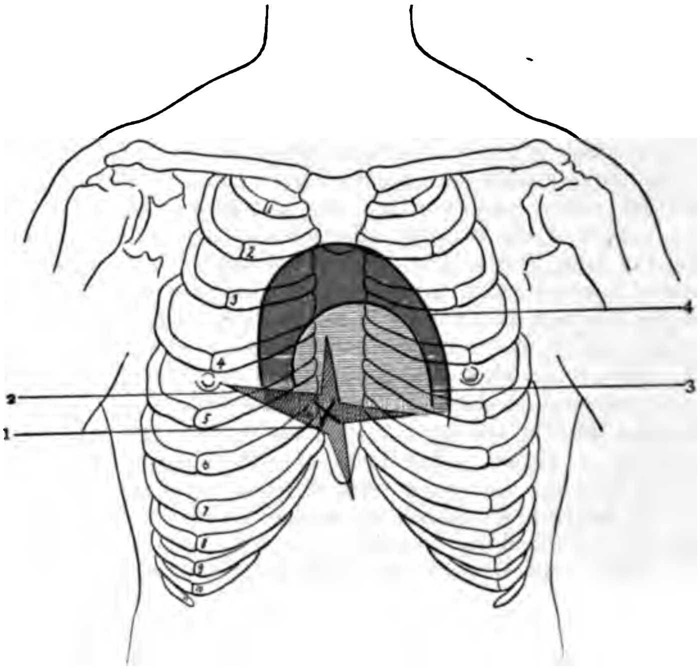

FIG. 52.—1, Seat of greatest pronunciation; 2, chief direction of conveyance; 3, boundary-line of absolute dulness; 4, boundary-line of relative dulness (modified from Sahli).

TRICUSPID STENOSIS.

THIS is a rare condition, occurring as a congenital and an acquired disease with about equal frequency. As a primary, independent disease tricuspid stenosis is very rare, being usually seen in association with organic disease of the left side of the heart. The lesions of mitral and tricuspid stenosis are observed to be combined most frequently, while those of tricuspid stenosis and aortic insufficiency coexist less frequently. The morbid changes are practically identical with those of mitral stenosis, the right auricle becoming dilated, and this being followed by general venous stasis. The effect of tricuspid stenosis upon the right ventricle is the same as that of mitral stenosis upon the left ventricle. The right ventricle, however, is usually hypertrophied, owing to the obstruction in the pulmonary circulation that results from the combined valvular deficiencies.

Special Etiology.—The fact that mitral and tricuspid stenosis frequently have a common cause, acting concurrently, can scarcely be doubted in view of their frequent association and pathologic identity. Hence the statement that *rheumatic antecedents* are furnished by the history in from 30 to 40 per cent. of the cases of tricuspid stenosis need excite no surprise. As in mitral stenosis, so in tricuspid, *sex* is a potent factor, the statistics of Bedford, Fenwick, Herrick, and of Leudet (which embrace a total of 160 cases) showing a ratio of 5 to 1 in favor of the female sex.

Symptoms.—The symptoms are those of the combined affections.

Physical Signs.—*Inspection* sometimes reveals a feeble venous pulse in the jugulars, due to right auricular systole, and hence presystolic in time. *Palpation* may detect a presystolic thrill over the body of the right ventricle. *Percussion* may enable the observer to indicate the enlarged right auricle. *Auscultation* gives usually an audible presystolic rolling murmur, which is best heard over the lower sternum and along its right border. The above physical signs are to be relied upon in *uncombined* cases, which are exceedingly rare. On the contrary, it is difficult in the extreme to differentiate the signs of tricuspid stenosis from those of the lesions with which it is almost uniformly associated—viz. mitral stenosis and aortic insufficiency.

PULMONARY INCOMPETENCY.

(*Pulmonary Regurgitation.*)

THIS is an exceedingly rare complaint that results from acute (malignant) or chronic endocarditis after birth; it is also rarely due to a congenital malformation. In the latter form union of two of the segments is often observed; in the former, the usual sclerotic processes, with the occasional adhesion of one or more segments with the pulmonary artery wall, may be noted. The effect of the lesion is to cause hypertrophy and dilatation of the right ventricle. The *physical signs* furnish no diagnostic characteristics. There is developed a diastolic murmur which is most audible in the second pulmonary interspace, and is transmitted to the lower sternal region, simulating the murmur of aortic regurgitation. The water-hammer pulse and marked hypertrophic dilatation of the left ventricle are present in the latter complaint, however, and are absent in pulmonary regurgitation. In pulmonary insufficiency, on the other hand, hypertrophy and dilatation of the right ventricle ensue. Preble reports a case of *relative insufficiency* of the pulmonary cusps; at the autopsy aortic and mitral insufficiency were also found.

PULMONARY STENOSIS.

A QUITE frequent form of *congenital malformation* of the heart is the narrowing of the pulmonary orifice. In the rarest cases it is of *post-natal* date, and may result in induration, contraction, and fusion of the segments. In one of Osler's cases the orifice "was only two millimeters in diameter, with vegetations of acute endocarditis on the segments." I saw one case in which the pulmonary artery near the valve was contracted to one-half its normal caliber. *Myocarditis* with resulting contraction of the conus arteriosus may cause pulmonary stenosis, and some of the cases that originate during adolescence and later in life are due to *atheromatous* change, while others possibly are the result of *chronic endocarditis*, direct *violence*, and *ulcerative endocarditis.* The lesion is compensated by an hypertrophy of the right ventricle, following which dilatation and tricuspid incompetency may appear.

Symptoms.—*Cyanosis* and *distention of the systemic veins* are observed.

Physical Signs.—A systolic *thrill* may be felt at times over the base. There is considerable enlargement of the right ventricle, as elicited by *percussion* and *palpation*, and a *systolic murmur* of greatest distinctness is audible, as a rule, in the third left space near the sternum. It is *harsh*, superficial, and transmitted a short distance upward and to the left. Occasionally this murmur is heard best at the aortic valve, but it is never conveyed to the vessels of the neck, and hence is easily distinguished from the aortic systolic murmur. Its harsh character and loudness would serve to obviate confusion with *functional* or *anemic murmurs* that are sometimes heard here. The pulmonic second sound is weak, and, not rarely, there is a diastolic murmur of the same character, indicating *pulmonary regurgitation.* Broadbent asserts that a *temporary* systolic murmur due to severe exertion may be observed, and I have noted a systolic murmur in the pulmonary area in young adults of remarkably vigorous build and unusual endurance. Sansom holds that disease of the pulmonary artery (contrary to other forms of organic heart-disease) predisposes markedly to pulmonary tuberculosis. I have at present under my care a tuberculous patient in whom there is a double murmur audible at the pulmonary orifice.

COMBINED FORMS OF CARDIAC DISEASES.

IT may be asserted safely that in more than one-half of all the cases combined lesions or murmurs are exhibited before the fatal termination. As I have already stated, stenosis of an orifice when due to valvular disease is associated with incompetency of the corresponding valve. Thus aortic stenosis is constantly combined with or followed by aortic incompetency, and in like manner mitral stenosis by mitral incompetency. The association may also have reference to lesions at two or more different valves; and according to the table of F. J. Smith, the relative frequency of the chief murmurs found in combination is as follows:

Aortic diastolic and systolic and mitral systolic,	16.55 per cent.
Aortic stenosis and mitral stenosis,	6.12 "
Aortic diastolic and mitral systolic (common in children),	5.21 "
Aortic diastolic and systolic and mitral presystolic and systolic,	3.77 "

When two lesions coexist at the same valve, the one may compensate, in part at least, for the other, as, for example, in the case of aortic stenosis in association with aortic regurgitation. Here the stenotic deficiency lessens the reflux current from the aorta into the left ventricle during the diastole; hence the latter receives a correspondingly diminished amount of blood. During the contraction of the ventricle the distending force in the aorta is diminished, both on account of the narrowing at the aortic orifice and the relatively lessened contents of the hypertrophied ventricle. Similarly, in dominating mitral incompetency an associated mitral stenosis by lowering the strength of the regurgitant current renders the conditions more favorable. Relative insufficiency at the mitral valve, following aortic insufficiency, may prove salutary by preventing over-distention of the left ventricle, and also the over-filling of the arterial tree and the possible rupture of a blood-vessel. On the other hand, when mitral incompetency is secondary to aortic stenosis, the latter defect may hasten the unfavorable tendencies in the former.

Relative tricuspid incompetency, secondary to mitral disease (a frequent combination), usually results in the development of a serious impediment to the systemic venous circulation, and often heralds a speedily fatal issue. It is probable that in advanced mitral disease the occurrence of a slight leakage at the tricuspid valve may be the means of obviating disastrous consequences to the right ventricle in case of undue strain.

Physical Signs.—These are confusing, but a systematic analysis often leads to the correct inference. That one of the valvular lesions predominates over all others is a fact of paramount importance for the solution of these cases. The chief lesions can usually be determined by noting the *seat*, the area of transmission, and the character of the most pronounced murmur; and more important still is the *correct timing* of any murmurs that may be audible. When a murmur occupies both the aortic and mitral areas the student will note two points of maximum intensity, and that each grows weaker as the stethoscope is moved toward the mid-præcordial region. The secondary alterations in the heart frequently coincide with the predominating murmur, and observers should recollect the familiar fact that mitral murmurs are often secondary to aortic, and that tricuspid murmurs point to accompanying mitral disease. Unquestionably, a single observation of these cases, however carefully made, is often profitless.

Complications of Valvular Disease.—Most of these have already been spoken of at sufficient length, but to restate them collectively in this connection may prove useful to the student and physician. They are—(1) acute endocarditis (including the ulcerative form); (2) acute pericarditis; (3) pleurisy; (4) pneumonia; (5) nephritis, followed by uremia; (6) local or general arterial sclerosis; (7) chronic gastric or intestinal catarrh with intercurrent acute attacks; (8) embolic processes; (9) angina pectoris; (10) edema of the lungs; (11) hysteria, neurasthenia, epilepsy, and insanity; (12) rupture of the skin of the extremities in consequence of excessive edema, with erysipelatous inflammation; (13) synovitis, a not uncommon complication, fever, swelling of one or more of the joints, and pain are the usual symptoms. The muscles of the extremities may also be involved simultaneously. It is highly probable that these manifestations are to be ascribed to rheumatism, though they are also met with in ulcerative endocarditis. (14) Febrile paroxysms occur at varying intervals of time, and are due to a variety of causes, as rheumatism, acute endocarditis, and pericarditis. Ulcerative endocarditis may also occur and be attended with an irregular type of fever.

Course and Duration.—When valvular disease consists in rupture of a segment the course is brief and usually proves quickly fatal. Apart from these exceptional instances the duration is measured by months, or more often by years or even decades. Statements applicable to all cases cannot be made, however, owing to the wide differences in different cases. Among the circumstances affecting the duration I would mention in particular the patient's mode of life, the hygienic conditions under which he lives, his occupation, mental condition, and the severity of the morbid processes. Every experienced physician has doubtless met with a small class of cases that have terminated fatally in from six months to a year, having developed in that period all of the serious phenomena and complications of the more chronic forms of organic heart-disease. In the preponderating proportion of cases, however, the course is exceedingly slow, and often cases have existed many years before they have finally been recognized. In numerous instances the patient follows his usual avocation, which may even be laborious, for years, and without discomfort. In other cases the symptoms, as dyspnea on exertion, are so slight as not to excite suspicion.

Facts such as these render it obvious that while the period of compensation is long, its exact limits are indeterminable. In 12 instances of chronic endocarditis that have developed under my observation (some having lasted ten or twelve years) only 3 have reached the stage of broken compensation. The *progress after failure of compensation* is more definitely known, since frequent opportunities for observation are afforded. At this time the cases also exhibit wide differences respecting their duration; in my own experience they have varied from two to three months to as many years (rarely even longer), depending much on the patient's mode of living. The course may be shortened by severe external injury, intercurrent acute illness (especially febrile disease), vicious habits, straining efforts, and the like.

Prognosis.—The detection of a cardiac murmur should not alone lead to a gloomy prognosis. Says Osler: "With the apex-beat in the normal situation and regular in rhythm, the auscultatory phenomena may be practically disregarded." Individual cases require separate and careful consideration. It is well not to advance positive assertions until all the circumstances that may influence the prognosis of any given instance have been well weighed. Observation of a case for some weeks and months enables the physician to speak with greater confidence and knowledge concerning the probable outcome. Prior to the occurrence of disturbances of compensation the prognosis is measurably favorable. After this pivotal event the prognosis as to life becomes wholly unfavorable in direct proportion to the extent of the degenerative changes of the myocardium. Disturbances of compensation that are attended with marked arrhythmia, urgent dyspnea, and general dropsy may admit of complete relief. Later, restoration of the balance of forces becomes only partial, and finally the above-mentioned symptoms become more pronounced; Cheyne-Stokes' breathing may then develop, and after a prolonged and distressing struggle for breath the patient succumbs. Death may also occur suddenly from cardiac paralysis. Among ominous and yet common *complications* and intercurrent affections may be cited again extensive edema of the lungs, pneumonia, typhoid fever, embolic processes, ulcerative endocarditis, acute endocarditis, obstinate gastritis, and nephritis. On the contrary, *favorable indications* are sound general health, good external conditions (absence of poverty, hunger, etc.), strong and regular action of the heart, absence of arterio-sclerosis, of excessive hypertrophy, of rheumatic antecedents, and any vices of life. *Age* influences the prognosis to some extent. In children under ten years the lesions are usually somewhat more rapidly progressive than in adults, and the compensatory hypertrophy is developed with corresponding rapidity; hence the period of failing compensation is reached earlier. This may be said to be a broad general rule, and I have found that it is one to which there are many exceptions. Among other reasons for the more gloomy prospect when heart-disease occurs in young children are the following: the mitral valve is generally implicated, the liability to rheumatic intercurrences is great, and children, unless carefully controlled, overtax at play the reserve cardiac power when indulging in running and other forms of exercise. After the twelfth year the prognosis becomes more favorable. *Sex* is also a modifying prognostic factor, women bearing valvular lesions better than men, apart from the influence of childbearing, though even this is an influence the significance of which has been greatly magnified by many writers. To explain the more favorable outlook in women we have two main facts—viz. a less laborious as well as a more quiet life, and a diminished liability to arterio-sclerosis and involvement of the coronary vessels. The particular valve involved has some influence on the prognosis.

Aortic regurgitation gives, on the whole, a rather favorable prognosis, particularly in those cases that begin in early adult life, granting, of course, that the patient regulates wisely his manner of living. Under such circumstances the lesion may be compensated for many years or even decades. The increased vigor of the left ventricle as compared with the right is conducive to longevity in this disease. After failure of compensation, the prognosis is less satisfactory in aortic regurgitation than in mitral regurgitation, since restoration of compensation is not as readily accomplished in the former as in the latter variety. In the lesion under consideration a chief danger arises from associated arterio-sclerosis—a rather frequent occurrence in advanced life—and from implication of the coronary arteries. Much depends upon the condition of the latter vessels. When their lumen is narrowed starvation of the heart-muscle quickly ensues, followed by myositic degeneration. Blocking of one of the branches of the coronary artery is the most frequent cause of sudden death in this affection. In *aortic stenosis* equally favorable predictions are warrantable when the disease is uncomplicated.

Mitral regurgitation, when a primary lesion, is propitious, except in the very young, and not infrequently the progress of the morbid process is apparently arrested. In a considerable proportion of cases the disease does not materially shorten the life of the sufferer. In a larger percentage, however, there is special liability to a renewal of the causative affections (*e. g.* rheumatism) and to pulmonary conditions of serious import, producing exacerbations and permanent aggravations of the disease. The gravity of these intercurrent complaints is also increased by the existence of the cardiac lesion. Failure of compensation at once renders the prognosis decidedly unfavorable. In *mitral stenosis* compensation of the right heart fails somewhat earlier than in mitral insufficiency, and hence the accidents and conditions referable to the lung (diffuse pulmonary apoplexy, edema) are not so long delayed as in the latter disease: this is also true of the later, more serious manifestations. I have learned by experience that mitral stenosis is better borne by women than by men, and better during adolescence and early adult life than during more advanced years. The congenital forms are comparatively benign. It should not be forgotten that mitral stenosis causes sudden death more frequently than any other form of organic disease of the heart except aortic regurgitation. *Tricuspid incompetency*, whether secondary to disease of the lung or of the left side of the heart, is extremely grave. It is usually indicative of dilatation following hypertrophy of the right ventricle. Compensatory hypertrophy, however, can be re-established, and sometimes repeatedly.

Treatment.—This falls naturally into three subdivisions: (1) prophylaxis; (2) management during the stage of compensation; (3) treatment of the stage of non-compensation.

(1) **Prophylaxis.**—It can scarcely be doubted, as shown by the statistics of Sibson, that complete rest and protection of the surface during an attack of acute articular rheumatism lessen the average percentage of cases in which acute endocarditis develops. When the latter complication occurs in acute rheumatism the patient should keep to his bed for some time after all rheumatic symptoms have disappeared (two to six weeks) or until the improvement in the cardiac condition has ceased absolutely. This precautionary measure will often lessen the extent of the ensuing chronic endocarditis, and also increase the proportion of perfect recoveries. Suitable dietetic and medicinal treatment must necessarily be combined. When the physician is cognizant of hereditary predisposition to organic heart-disease, or has to deal with the arthritic diathesis (gouty or rheumatic) or the alcoholic habit, he can frequently, by timely advice and hygienic suggestions, direct his patient to adopt measures that will obviate the occurrence of valvular disease. All persons predisposed by heredity or otherwise should be told of the probable effect of muscular strain, alcohol, and other exciting factors; too often, however, when he sees his patient for the first time the physician is confronted by an incurable malady.

(2) **Management during the Stage of Compensation.**—Three main objects are to be accomplished: (*a*) The avoidance of every agency that tends to aggravate or maintain the lesion or lesions. Under this head the detection and removal of all causal factors is imperative. Thus, if the patient's avocation entails undue muscular effort, it must be abandoned; violent exercise, as running up flights of stairs, heavy lifting, or straining at stool, is also dangerous and must be prevented. If alcohol has been a factor, it must be discontinued; if syphilis, it must be treated specifically. If there be present a rheumatic or gouty taint of the system, it must be overcome as far as possible by special measures. Fatigue and exposure must be avoided, particularly if the patient be comparatively young. Emotional excitement and mental over-exertion injuriously affect the cardiac lesion; therefore tranquillity of mind should be insisted upon, though moderate and systematic mental exercise has no risks for the patient. In the case of children at school careful supervision of their studies as well as of their recreative exercises is essential. Fright and sudden emotion must be avoided if possible. The use of tea, coffee, and tobacco should be rigidly prohibited. In mitral disease, bronchitis is to be especially guarded against.

(*b*) The *diet* of the patient demands careful regulation. Only a very moderate amount of food, composed for the most part of readily digested albuminous articles (milk, eggs, the lighter forms of meats, and stewed fruits), is to be taken, since overloading the stomach will disturb the action of the heart; particularly is this true at night. The carbohydrates may be allowed only in limited quantities, since they are apt to decompose and form gases that distend the stomach and intestines. For the same reason the coarser and more indigestible food-stuffs should be avoided. Small meals at brief periods is a plan of feeding that I can highly commend. The amount of liquids taken should not exceed the actual requirements of the patient, inasmuch as over-filling of the blood-vessel system increases the work of the already overburdened cardiac forces. Alcoholic beverages should not be used as a rule; but if the patient has been moderate in the use of alcohol, and particularly if he be advanced in years, light wines may be allowed in moderate quantity to aid digestion. (*c*) *Carefully regulated exercise* is beneficial, but it must be gentle and should be taken out of doors. As before intimated, a good general muscular development is an aid of no mean value to the conservative powers of the heart. Oertel, with a view to assisting the compensatory forces of the heart, has recommended graduated physical exercise; he advises that patients be instructed first to ascend low elevations, and later mountains of a considerable height, the object being to bring about full compensation. Great caution is to be exercised by the physician, however, since this method has been found to be inapplicable to a large percentage of cases. Cardiac distress, palpitation, and dyspnea are complained of by this large group of patients if other than the gentlest forms of exercise be undertaken. With respect to exercise, then, the sensations and experiences of each patient must be consulted before the physician can advise judiciously. Woollens should be worn next to the skin during both the warm and the cold seasons. The skin should be kept clean by daily sponge baths, and if these be followed by friction of the surface, the bodily nutrition will be improved and the liability to intercurrent attacks of bronchitis greatly lessened. The bowels should be moved each day, and usually the use of stewed fruits suffices to accomplish this end; if not, salines, Rochelle or Carlsbad salts, and the bitter waters (Friedrichshall, Hunyadi-Janos) must be brought into requisition. In winter a warm climate may prove advantageous, though long journeys are often illy borne, owing to the fatigue induced thereby. If the patient becomes anemic or his nutrition is notably impaired, a suitable change of air,[1] or the use of quinin, arsenic, small doses of mercury, and cod-liver oil, is to be recommended. Digitalis should not be employed when compensation can be preserved in other ways. We should train the heart up to the amount of work required of it (Brunton).

(3) **Treatment of the Stage of Non-compensation.**—The principal object to be kept in view in this stage is the reinvigoration of the exhausted cardiac muscle, and thus to relieve the impeded circulation. *Sudden death* may, though rarely, occur from the blocking of a branch of the coronary artery or from acute dilatation. Failure of compensation, however, *begins gradually* as a rule, the condition often existing without marked or characteristic symptoms; but its early recognition is important from the stand-point of therapy. Increased dyspnea on exertion, and nocturnal seizures of shortness of breath and irregular action of the heart (*arrhythmia*), are among the earliest clinical features. The latter symptom may have been present before, and particularly during active exercise in mitral disease, but now it is more marked, and may be constant. The patient's nutrition often suffers, and he is pale and rather feeble. Absolute quiet, liberal feeding with suitable food, and iron may in a little while restore the impaired cardiac tone. If this treatment fails, by the end of a fortnight a small dose of digitalis should also be exhibited (5 minims—0.333—of the tincture three times daily); the latter should be promptly withdrawn upon the disappearance of the symptoms. *Decided indications* of lost compensation are marked dyspnea and arrhythmia; the canter rhythm; an irregular, small, compressible pulse; and cyanosis, with or without the presence of dropsy. The object now is the maintenance of the blood-pressure at an adequate height by the following means: (*a*) *Absolute rest in bed.* This diminishes greatly the work of the heart, and thus enables it to regain largely its former vigor. Rest joined with massage and careful yet liberal feeding and attention to the bowels will often restore disturbed compensation in from one to two weeks. In 4 cases recently treated at the Medico-Chirurgical Hospital this method succeeded admirably.

(*b*) *Cardiac stimulants and tonics.* Of these, when occasion demands, the most important is digitalis, and this may be tried in any case in which dilatation exists. By stimulating the pneumogastric, by increasing the blood-supply to the heart-muscle, by causing the systole to be more complete and the period of diastole to be lengthened, digitalis becomes an invaluable aid to the nutrition of the cardiac muscles. In addition, the heart contracts more regularly and the blood-pressure in the peripheral circulation is raised. As a result of the use of this drug the tissue-calls upon the cardiac forces from the outlying portions of the body are satisfied and the reserve energies of the heart-muscles are maintained.

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[1] Observation and experience have confirmed my belief that sea-air during the warm season and high altitudes at all times are injurious in their effects in valvular disease of the heart.

In *mitral disease* the influence of digitalis is most beneficial, the pulse becoming slower, of better tension, and more regular while the urine increases in amount. In mitral incompetency its good effects are ascribable in part to the powerful contractions of the left ventricle, whereby the normal blood-stream from the ventricle to the aorta is greatly increased. On the contrary, the patient's condition is occasionally aggravated by the drug, because "the leak is increased as much as the normal flow" (Hare). Digitalis exercises its most beneficial influence by rendering the systole of the right ventricle more energetic, the blood-pressure being raised in the pulmonary circuit and left auricle; this fills the left ventricle better during diastole and "resists reflux through the mitral orifice in the systole" (Broadbent). In mitral stenosis digitalis, by lengthening the period of diastole, allows time for the blood to pass from the auricle through the narrowed mitral orifice into the ventricle. Slight toxic effects may sometimes result from digitalis, the pulse becoming thread-like and irregular, and the urine scanty. Under these circumstances the drug should be discontinued.

In *aortic regurgitation* digitalis exercises as great, if not as wide, an influence as in mitral disease: the theoretic view, however, that by prolonging the diastole digitalis causes overfilling of the left ventricle rests on too slender a foundation to be regarded as a valid objection to its use. It may, however, produce excessive hypertrophy, in which case it should be promptly withheld. The symptoms due to secondary mitral regurgitation digitalis meets by reinforcing the ventricles, particularly the right. In all forms of organic heart-disease, though most frequently in aortic regurgitation, nausea and vomiting sometimes follow the administration of digitalis: when this is the case it should be stopped and other cardiac stimulants substituted or the dose reduced to the point of tolerance, when it may be continued if adequate to maintain a proper effect. When secondary dilatation comes on in aortic stenosis digitalis is needed to increase left ventricular power. The dose is to be calculated according to the degree of existing dilatation. When tricuspid incompetency is secondary to mitral disease striking results are obtained from the use of digitalis (*supra*); but when it exists alone—*e. g.* following emphysema or cirrhosis of the lung—digitalis often fails. The cardiac contractions, if they have previously been irregular, may become somewhat more regular, but the precordial distress will often be increased, while the circulatory disturbance, as evidenced by the objective signs, will remain unrelieved. If *dropsy be slight* or absent, 10 minims (0.666) of the tincture or 2 to 3 drams (8.0–12.0) of the infusion, three or four times daily, will suffice. If symptoms of *decidedly unfavorable import* be present, including marked dropsy, the dose should then be larger (of the tincture, minims x to xv—0.666 to 0.999; of the infusion, ℥ss—16.0—every two or three hours) for two or three days, when the dose must be diminished or given at longer intervals. Quantitative estimations of the urine should be made during the use of the drug, and if the effect be good the daily amount will often be greatly increased; if bad, there will be a diminution rather than an increase in the amount. There are not a few patients in whom the symptoms of commencing failure of compensation recur as soon as the drug is discontinued. To such digitalis may be administered continuously or until toxic symptoms are manifested. I believe that the solid preparations (powder and extracts) can be taken for longer periods than the liquid forms without exciting untoward symptoms. This suggestion should be followed particularly in cases that are seen at long and irregular intervals of time. Evidences of fatty degeneration and atheroma are not contraindications to its use, but are signals for the observance of extreme caution. It should, however, be a rule never to be broken to discontinue the digitalis when the symptoms of disturbed circulation have vanished. When it fails of its effect or is not well borne, and when, as often happens, the arrhythmia is not favorably influenced by it, the physician is compelled to resort to other cardiac stimulants. These are numerous, and, whilst their good effects are not comparable to those of digitalis in every respect, some of them seem to meet certain indications that are not met by this drug. Among the more important are nitroglycerin, strophanthus, strychnin, cocain, spartein, and caffein. Nitroglycerin in small doses is at the same time a cardiac stimulant and an arterial relaxant, and hence is more often useful in aortic than in mitral valvular disease. In larger doses, when left ventricular hypertrophy is excessive, as may occur when general arteriosclerosis is associated with aortic regurgitation and also (though rarely) aortic stenosis, it is highly useful, widening the blood-paths, and causing less powerful contractions of the heart. Strophanthus should be employed in instances in which digitalis must be interrupted, since the action of these two remedies upon the heart-walls is very similar. The tincture is usually employed, the dose (varying with the indications of each case) being from 4 to 10 minims (0.266–0.666) every three or four hours, and in controlling the irregularity or intermittency of cardiac action it is sometimes better in its influence than digitalis. Many cases of marked arrhythmia will not yield to either when but one is given; and in such I have occasionally obtained good results from digitalis and strophanthus in combination. Caffein citrate is also a good cardiac stimulant, but is superior as a diuretic. It should be stated that, rarely, strophanthus, like digitalis, does harm rather than good, being sometimes badly borne by the stomach. Under these circumstances I have employed, both in hospital and private practice, the following combination:

℞. Caffein. citrat., ʒj (4.0);
Strychninæ sulphat., gr. $\frac{1}{3}$ (0.021);
Spartein. sulphat., gr. ij (0.129).
Ft. capsulæ No. xij.
Sig. One every three or four hours.

The above prescription is not only a good heart-tonic and stimulant, but also an equally good diuretic. Spartein is a potent diuretic and heart-stimulant when employed in doses of gr. $\frac{1}{6}$ to $\frac{1}{4}$ (0.010–0.016) every four to six hours, and is especially serviceable in organic heart-affections when dropsy as a symptom and nephritis as a complication exist. Strychnin, when given hypodermically in full dose, gr. $\frac{1}{30}$ to $\frac{1}{15}$ (0.002–0.004), is the most efficient cardiac stimulant known to medical science. It should be employed in this manner in cases in which there is sudden failure of heart-power with the development of serious symptoms. Given in doses of average size, *per os*, its effects in chronic valvular disease are not very striking. Atropin may be advantageously combined with it.

When the indications are urgent and the above agents are not available, diffusible stimulants, as ether or ammonium, may be used until more suitable remedies can take effect. Cocain simulates strychnin in its action. The dose is gr. ¼ (0.016) every four hours, and the drug may be given with digitalis in pill-form. Later, systemic tonics are often demanded by the *anemia* and other constitutional indications, and here iron and quinin should be joined with strychnin. Unquestionably, the value of iron in full doses as an aid to the completion of the work of restoring broken compensation has been and is still scarcely appreciated by the profession at large. When iron disagrees, arsenic may be given instead. In many cases of failure of compensation the restoration of the balance of the cardio-systemic circulation can be greatly assisted by depleting the over-filled venous system. There are two ways of attaining this end:

(*a*) *Venesection.*—When the right heart is over-distended, as shown by its very feeble efforts at contraction, and the whole venous system is intensely engorged, as shown by marked cyanosis and orthopnea, bleeding directly from a vein is not only warrantable, but often imperatively demanded in order to save life. From 16 to 30 ounces (473.0–887.0) may be removed safely, and the heart's action will almost immediately be observed to grow stronger and more regular, and the pulse fuller and of better tension. As before intimated, the form of dilatation of the right ventricle that follows emphysema is disinclined to yield to digitalis. In such instances, following the suggestion of Osler,[1] I have obtained brilliant results from free bleedings.

(*b*) *Depletion by purgation* affords less pronounced relief to the heart, though it is of the greatest value in cases in which a moderate grade of cyanosis and dropsy exists. As in the case of venesection, a feeble, irregular pulse is not a contraindication to the use of purgatives, since the latter remove directly a considerable portion of the heart's burden. The purgative to be used will vary with different cases. I select at the outset Rochelle or Epsom salts, employing them after the method of Matthew Hay—*i. e.* from 1 to 2 ounces (32.0–64.0) of Rochelle or 1 to 1½ ounces (32.0–48.0) of Epsom salts, in concentrated solution, to be given from a half to one hour before breakfast. Watery evacuations (three to six in number daily) usually follow the administration of the saline; but, unfortunately, one meets with many patients in whom it produces symptoms of marked catarrhal irritation. Next to salines, the most satisfactory results have been obtained from the use of elaterium; I often combine this with podophyllin and belladonna. I have never seen good results from the use of mercurials when the object has been to procure venous depletion, but they are of service in dropsy, and particularly in ascites.

Schott of Nauheim has introduced a special treatment by baths that is applicable to most forms of valvular disease, simple dilatation, and nervous affections of the organ. The beneficial effects are principally attributable to the salt, which acts as a cutaneous stimulant, and to a slighter degree to the gaseous ingredients of the bath. Greene[2] regards the warmth and moisture as the important features. Twenty-one baths are given in one month, dropping one every fifth, fourth, third, and second days. The water contains sodium chlorid, calcium chlorid, and carbon dioxid, and the temperature ranges from 82°–95° F. (27.7°–35° C.). The first bath lasts seven or eight minutes; the time is then gradually lengthened, the temperature lowered, and the carbon dioxid increased. After the bath the patient is rubbed and allowed to rest for an hour.

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[1] For illustrative cases from Prof. Osler's wards, see article by Leufler, *Medical News*, July, 1891.

[2] *Jour. Amer. Med. Assoc.*, Oct. 15, 1898.

Artificial Nauheim baths are successfully employed in certain American hospitals at the present time. They are prepared as follows: Five pounds of sodium chlorid and eight ounces of calcium chlorid are dissolved in one half bath (30 gals.—114 liters), the temperature of the water being 95° F. (35° C.). In a few days the bath is charged with carbon dioxid by adding sodium bicarbonate (1 lb.—453.6) and HCl (½ lb.—226.8), the latter just before the bath is taken. The effects are to lower the pulse-rate, to decrease the size of the heart, to stimulate the nerves, and, indirectly, the cardiac nutrition. There is also a tendency toward improvement of the skin and an increase of the urine.

Gentle resistance exercises (consisting of all the more reasonable movements that a person naturally makes, and resisted by an attendant) form an important element of the treatment, since they tend to stimulate the muscles and nerves and propel the blood from the congested veins. The Nauheim treatment is not suitable in aortic regurgitation, aneurysm, or fatty degeneration of the heart, although the movements alone are beneficial in these conditions and may be employed to the exclusion of the baths.

Individual symptoms frequently become so conspicuous as to demand special treatment.

(1) *Dyspnea and Orthopnea.*—When these phenomena are caused by engorgement of the pulmonary vessels, the cardiac stimulants above detailed usually afford relief. Frequently the patient cannot lie down, in which case a suitable bed-rest often gives immediate comfort and support. For the severe attacks of nocturnal dyspnea (amounting sometimes to orthopnea), particularly when accompanied by cardiac palpitation, the subjoined formula has proved itself of great benefit:

℞. Sodii bromidi, gr. xv (0.972);
Tr. opii deod., ♏x–xv (0.666–0.999).—M.
Sig. To be taken in one dose at bed-time.

In the late stages of heart-disease morphin, given hypodermically, is to be preferred in combating this symptom, and is entirely free from the usual objections to the habitual use of the remedy. Its influence for good is inestimable. Dyspnea may also be produced by associated bronchitis, edema, emphysema, and hydrothorax—conditions that must be treated according to the customary rules. Frequent physical explorations of the chest should not be omitted. Hydrothorax demands aspiration, and this repeatedly in some instances.[1]

In valvular disease (particularly aortic), owing probably to coronary arterio-sclerosis, paroxysms of severe dyspnea (*cardiac asthma*) are apt to arise. These are best overcome by nitroglycerin in ascending dosage in combination with sodium bromid at bed-time, to be repeated as needful.

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[1] When the chambers of the heart are greatly dilated care must be exercised in inserting the aspirating needle, lest the left ventricle be entered.

(2) *Cough.*—Cough is common after failure of compensation, and is due to bronchitis resulting from stasis in the pulmonary vessels. In mitral disease it may come on before the rupture occurs. Beyond the treatment directed to the causal condition (the cardiac failure) nothing is needed to relieve the cough. These subjects, however, are prone to suffer from catarrhal bronchitis due to cold, and this impairs the compensatory mechanism. J. Weiss extols heroin in cases that are not relieved by the ordinary remedies.

(3) *Hemorrhage* may take place, and generally from the lungs, though it may also proceed from the nose, stomach, bowels, or uterus. In a recent case of double aortic and relative mitral insufficiency attended with marked dropsy, rather copious hemorrhages occurred from the bowel, but with apparent relief to the patient. The hemoptysis, which is a rather frequent accompaniment of mitral lesion, is rarely excessive, and is probably always beneficial. I would advise against active treatment unless the hemorrhage is actually copious in amount.

(4) *Palpitation* may be due to different causes, the recognition of which in each case is important. At times undue hypertrophy maintains a constant throbbing and distress in the precordial region, the former being distinguished by the strength of the impulse and by the full, tense pulse at the wrist. Palpitation is best met by the use of the tincture of aconite, ♏ j–iv (0.066–0.266, every four hours. With the aconite I frequently associate the bromid with excellent effect. An ice-bag to the precordia is worthy of recommendation. Unless the patient's discomfort is significant, however, this symptom does not call for active treatment. The administration of a saline purge not infrequently serves to quiet the heart. The patient may suffer from pure nervous palpitation, in which case the diet and the condition of the stomach must be carefully looked to, while for the throbbing the bromids of ammonium and sodium, together with preparations of valerian, are the most reliable.

(5) *Anginose Pains.*—These are seen in aortic incompetency accompanied by sclerotic vessels, and more especially in mitral stenosis. When dependent upon rigid blood-vessel walls nitroglycerin should be tried; if the attacks be severe, amyl nitrite by inhalation deserves a trial, and, this failing, morphin and atropin may be employed hypodermically. The latter measures, as a rule, promptly relieve the patient's suffering. Local measures alone are sometimes sufficient when the pain is only moderately intense, and the ice-bag or Leiter's coils may be tried. The sedative effect of a blister (4 by 6 in.—10 by 15 cm.) has more often proved effectual in my experience, though its use should be limited to patients whose general strength is not materially impaired.

(6) *Pain* referred to the stomach, and less frequently to the abdomen also, occasionally assumes prominence and is relieved with great difficulty. It is dependent, in part at least, upon obstinate subacute gastritis, and I have quite recently seen an instance of the sort verified by autopsy. Among many drugs tested in this case, opium alone gave relief. Usually the pain results from gaseous distention of the stomach and bowels, and is not intense, a mild laxative frequently bringing relief. Should this fail, however, trial should be made of the carminatives in combination with some antiseptic agent, as salol or creosote.

(7) *Gastric Symptoms.*—Soon after compensation is broken the ap-

pearance of mild symptoms of catarrh of the stomach may be said to be the rule, and these yield to simple measures in addition to the cardiac stimulants and laxatives already indicated. But there are not a few instances in which such symptoms as gastric distress and uneasiness, constant nausea with frequent vomiting, particularly after food, occur and assume a distressing phase. Such patients cannot, as a rule, take digitalis or strophanthus by the mouth; they sometimes, however, do well on the capsules before adduced composed of strychnin, spartein, and caffein. When the latter cannot be borne I employ hypodermically digitalin and strychnin or caffein citrate, the latter being made soluble by the addition of sodium benzoate in solution. Cases of this class reach an early fatal termination, as a rule. The symptoms may be partly due to gastric catarrh coupled with hepatic engorgement, and partly to uremic intoxication.

(8) *Nervous Symptoms.*—Insomnia and restlessness are almost constantly present at some period in the course of heart-disease, and notably in the more advanced stages. The restiveness is rendered more distressing on account of hideous dreams and cardiac palpitation on awaking. For these phenomena stimulation often answers a better purpose than sedation. Hoffman's anodyne (ʒj—4.0—well diluted), spirits of chloroform (♏xv—0.999), or ether (ʒss—2.0), taken in whiskey (℥j—32.0) are serviceable. The elixir of ammonium valerianate is also of value when given in full doses. I formerly employed sulfonal in combination with camphor monobromate when a hypnotic was required to afford sleep. Recently, the use of trional (grs. xv) in combination with sodium bromid (grs. xx) was found more satisfactory. Paraldehyd and chloralamid are among the remedies of choice in the treatment of this symptom, but I have had no experience with their employment.

In the later stages there is no objection to the use of morphin hypodermically. Headache due to uremia may frequently be a troublesome symptom in connection with sleeplessness, and in such cases morphin is the remedy *par excellence;* it is to be supplemented by free purgation and cardiac stimulants. Should the right heart be found flagging, venesection may be practised.

(9) *Dropsy.*—Among the symptoms requiring special treatment in advanced valvular disease dropsy easily assumes the lead. As above pointed out, rest with attention to the diet and state of the bowels will often restore defective compensation even when accompanied by a moderate degree of dropsy. In the severe grades of failure of the balancing forces the cardiac stimulants and purgatives before mentioned often suffice to remove the dropsy for a considerable period of time. Later, however, it becomes obstinate, and refuses to yield to any of the known methods of treatment. The therapeutic indications, so far as the symptom under consideration is concerned, are for the use of diuretics and purgatives. Diaphoretics, particularly the hot-air and vapor baths, are not to be thought of, since they tend to depress the already weakened heart. While describing the action of digitalis as a cardiac stimulant, incidental allusion was also made to its action as a diuretic. In view of the fact that it raises the blood-pressure in the peripheral vessels and capillaries by contracting their walls, and because of its stimulating

effect on the heart, digitalis in large doses becomes a most efficient diuretic in cardiac dropsy. When the renal secretion is not free under its use, or when for some good reason it cannot be taken, I have frequently found that a combination of strychnin, spartein, and caffein (*vide supra*) will excite free diuresis. Nitroglycerin may also be prescribed, especially in cases presenting evidences of advanced arterio-sclerosis. An unirritating yet highly effective diuretic mixture in these cases is the following:

℞. Potassii acetatis,	ʒj	(4.0);
Inf. digitalis,	℥ij	(64.0).—M.

Sig. ℥ss (16.0) every three hours.

Purgatives are of the utmost value. Frequently, after a few copious watery evacuations as the result of the action of hydragogue cathartics, a free discharge of urine can be established, when before the latter event it has been impossible. Salines and elaterium, with podophyllin and belladonna, are agents that have been already recommended as purgatives (to deplete the venous system), and these should be first employed in the order named. Compound jalap powder may also be combined with the elaterium. A course of calomel, followed by salines until free catharsis is set up, is valuable from time to time. Mercury is especially applicable when the liver is much enlarged and ascites is a marked feature, or when the history of syphilitic infection is obtainable. It may be combined with cardiac stimulants and other diuretics as follows:

℞. Pulv. digitalis,		
Pulv. scillæ,	*āā.* gr. xij	(0.777);
Hydrarg. mass.,	gr. xxiv	(1.555);
Ext. belladonnæ,	gr. ss	(0.0324).

M. et ft. pil. No. xij.
Sig. One every three or four hours.

When efforts at relieving the dropsy by means of medicines fail, then the most dependent parts of the body, or those most swollen, should be scarified under strict aseptic precautions. Fine silver trocars with rubber tubes attached (Southey's tubes) may be inserted and the liquid allowed to drain off in a gradual manner.

Means to Prevent Recurrence of Broken Compensation.—When the compensation has been successfully re-established, the after-treatment must be prosecuted with vigor for at least a year. The cause of the rupture of compensation is most probably fibroid and fatty degeneration of the cardiac muscle, and hence the mere restoration of the compensatory power of the heart does not imply a complete cure of the impaired muscular structure of that organ. Much can be done, however, to overcome the tendency to degeneration by the peristent use of hematinics and other tonics, as cod-liver oil and mercuric chlorid, the latter in small doses. I have obtained excellent results from the use of the following prescription in these cases:

℞. Liq. arsenici chlor.,	♏xlviij	(3.186);
Tinct. ferri chlor.,	℥ss	(16.0);
Hydrarg. bichloridi,	gr. ss	(0.0324);
Glycerini, q. s. ad	f℥iij	(96.0).—M.

Sig. ʒj (4.0) after each meal, well diluted.

This preparation may be taken indefinitely with occasional brief interruptions. The patient should lead a very quiet life, and follow rigidly all hygienic rules that tend to prevent the production of valvular disease. Appropriate *diet*, it should be emphasized, is not inferior to appropriate medication in its salutary effect. Should the faintest evidence of failure of the right ventricle manifest itself, the patient must be put to bed immediately, and the foregoing treatment is to be carried out. I am inclined to the view that the plan herein advocated not only renders the course of recurring attacks of failing compensation milder, but that, in a considerable proportion of the cases, the much-dreaded recurrence is thus prevented.

CARDIAC THROMBOSIS.

Pathology.—True cardiac thrombi are seen most frequently on the right side of the heart, in the auricular appendices, and, less commonly, in the right ventricle near the apex. They are of firm consistence, and are tightly adherent to the endocardium, considerable force being required to dislodge them. The color, while generally grayish-brown or red, varies with the age of the thrombus, being more colorless as it becomes older. Cardiac thrombi may be pedunculated or sessile, and their contour is, as a rule, more or less rounded. Recklinghausen and others have observed globular masses, the so-called "ball-thrombi," in the auricles, without the slightest endocardial attachment. They vary greatly in size, from a mustard-seed to a hen's egg, and sometimes exhibit calcareous degeneration. Cardiac thrombi may occur singly or in groups of considerable numbers. From the cavity in which they have their primary seat they may project into other chambers of the heart, or from the left ventricle into the aorta for a considerable distance. It is evident that fragments detached by the blood-stream from these cardiac blood-concretions will tend to lodge in various viscera and in the peripheral tissues, and set up embolic processes. Examined microscopically, degenerate round cells and detritus are revealed, but pus-cells are not seen. Secondary degenerative changes, and later softening, may take place in the central portions of a thrombus, and these areas may contain a reddish-brown liquid.

Etiology.—The causes of cardiac thrombosis are to be found chiefly in some previously diseased or injured condition of the endocardium, though sometimes alterations of the blood constitute a factor of considerable importance. The condition may occur in the course of both acute and chronic diseases, in which the intracardiac conditions favor the formation of a blood-clot. Hence it is seen in connection with organic diseases of the heart in which the valvular and often the mural endocardium are roughened, and the obstructive and regurgitant lesions at the various valves cause retardation in the blood-current. Chronic obstruction in the lungs may contribute to the result by slowing the circulation in the heart. Cardiac thrombosis has been observed in many of the acute affections, and almost invariably there is a loss of endocardium, due to inflammatory action (endocarditis) at some point in the cavities of the heart. This becomes the seat of the fibrinous deposit which is subsequently imperfectly organized. Among the most important of these acute primary diseases are *rheumatism*, *diphtheria*, *lobar pneumonia*, and *pyemic* and *puerperal conditions*. It may be questioned whether, given a healthy endocardium, as contended by some writers, slowing of the circulation alone suffices to cause true cardiac thrombi.

Symptoms.—These will depend very much upon the rapidity with which the thrombus is formed, as well as upon its seat and dimensions. Thrombi invariably lack definiteness, and, as their effects are largely mechanical, signs of obstruction to the cardiac circulation and failure (more or less gradual) of the cardiac muscle are developed. The *pulse* becomes weak, rapid, and irregular; *dyspnea*, *vertigo*, and attacks of *syncope* are frequent; and later *cyanosis* may appear. It is probable that at times the liquefied products of a clot may be absorbed, producing blood-poisoning. When the thrombus is formed rapidly the symptoms are suddenly developed and the course is rapid. Rarely a valvular orifice, an efferent vessel, or the coronary artery may become blocked and instant death follow. Since the right heart is the most frequent seat of these thrombi, pulmonary embolism, attended with its usual symptoms, is a common event. When portions of a clot are broken off and swept into the systemic circulation, the clinical phenomena of cerebral, splenic, or renal *embolism* are exhibited.

The **physical signs** consist of a feeble impulse with marked arrhythmia; the area of dulness is somewhat increased to the right, and often upward; and the heart-sounds are greatly enfeebled and quite irregular, with marked change in any murmurs that may previously have been audible. A systolic pulmonary murmur may rarely be engendered.

Differential Diagnosis.—It is important to distinguish true cardiac thrombi, such as are above described, from the less dense and usually darker clots that are formed either immediately before or after death. The latter may seldom show an attempt at a very low grade of organization, and may present a somewhat decolorized appearance, but they do not adhere firmly to the endocardium. Moreover, *antemortem* and *postmortem* clots, as the latter may be appropriately termed, have a different causation from true thrombi. For instance, they are apt to form in diseases in which the fibrin-factors of the blood are greatly increased, as in pneumonia. Perhaps a more potent causal element is the progressive weakening of the heart-muscle, resulting in partial expulsion of the contents of the right ventricle; the blood that remains in the chamber is merely whipped up, and the deposition of its fibrin must thus be greatly favored. Such heart-clots may be generated if the endocardium be healthy, and cannot be separated positively from true cardiac thrombi by clinical observation.

The **prognosis** is uniformly bad and sudden death may be expected.

Treatment.—Beyond measures calculated to meet the symptomatic indications nothing can be suggested.

HYPERTROPHY OF THE HEART.

(*Hypertrophia Cordis.*)

Definition.—Hypertrophy is an increase in the muscular structure of the heart, evidenced usually by an increased thickness of its walls. It is almost invariably associated with dilatation of the chambers.

Pathology.—When the two processes—hypertrophy and dilatation—coexist, they cause great enlargement of the organ. To this condition the term "*eccentric hypertrophy*" has been given. Hypertrophy without dilatation receives the name "*simple hypertrophy*," and hypertrophy with diminution in the size of the cavities was formerly described as "*concentric hypertrophy*," but this term should now be regarded as obsolete, inasmuch as the condition is due to postmortem contraction of the ventricles.

The increase in size may affect either the whole heart, one chamber on either side, one whole side, or but a single cavity (*general* and *partial hypertrophy*). The process may also be limited to a minute division of the heart (*circumscribed hypertrophy*). Owing to its important physiologic function the left ventricle is more frequently enlarged than the right, and oppositely the right auricle is more frequently involved than the left. The *weight* of the normal heart in a man of average size is approximately 9 ounces (255.0); in a woman it is 8 ounces (226.0). In bilateral hypertrophy, however, the weight of the heart may be greatly increased; hearts weighing from 15 to 25 ounces (425.0–710.0) are seen in moderate grades of hypertrophy, and those from 40 to 50 ounces (1134.0–1420.0) in extreme cases (*cor bovinum*). Measurements showing the thickness of the walls also indicate the degree of hypertrophy[1] and the exact seat of the enlargement when not general. The normal diameter of the left ventricular wall is from 8 to 12 mm. ($\frac{1}{3}$–$\frac{1}{2}$ in.); that of the right ventricle, from 5 to 7 mm. ($\frac{1}{5}$–$\frac{1}{4}$ in.); that of the left auricle, about 3 ($\frac{1}{8}$ in.), and of the right, 2 mm. ($\frac{1}{12}$ in.). Suffice it to state in this connection that under conditions of cardiac hypertrophy the normal thickness of the various cavity-walls is usually doubled, not infrequently trebled, and, rarely, even quadrupled. In cases in which there is a concomitant dilatation the walls may appear thinned, while the measurement will show them to be in reality thickened.

The *shape* of the heart is also altered according to the seat and extent of the hypertrophy. If both ventricles are enlarged, the apex is widened and appears flattened; if only the left ventricle is involved, the apex is lengthened and is more or less pear-shaped; and if the right ventricle alone is hypertrophied (as in mitral stenosis), it may form the largest part of the apex, which will be less conical than in health.

The papillary muscles and columnæ carneæ are greatly thickened, and, particularly in the eccentric form of hypertrophy, they are often decidedly flattened. In this form the septum frequently shows increased thickness—a condition that I have never observed in simple hypertrophy. The muscular trabeculæ generally assume greater prominence on the right than on the left side. The muscular structure is usually of a deeper red color and also firmer than normally. The hypertrophied left ventricle can, as a rule, be lacerated readily, while the right, as first pointed out by Rokitansky, may be tough and leathery. As the heart continues to enlarge it sinks lower in the chest-cavity; this is not, however, owing to an increase in size alone, but more particularly to an increase in weight. In hypertrophy of the heart there is a multiplication of muscular fibers, to which alone the enlargement of its walls is attributable.

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[1] Measurements should not be attempted until the *rigor mortis* has been overcome by soaking the organ in water.

Etiology.—Hypertrophy of the left ventricle (sometimes termed *general hypertrophy*) results from obstructions to the arterial circulation of whatever sort. These may be classified, according to their seat, into—(1) **Lesions of the Heart.**—(*a*) Aortic incompetency and aortic stenosis; (*b*) Mitral insufficiency; (*c*) The fibroid form of myocarditis; (*d*) Pericardial adhesions, particularly in the young. Late in life the heart may become atrophied. In such cases the adherent pericardium exerts a counter-traction force during the systole, and thus the work is increased beyond the capacity of the normal heart, with consequent hypertrophy. In valvular disease the augmented tension in the ventricle induces the hypertrophy.

(2) **Abnormal Conditions of the Blood-vessels.**—(*a*) Narrowing of the aorta—*e. g.* congenital stenosis, external pressure, and the development of an aneurysm; (*b*) General arterio-sclerosis, by raising the pressure; (*c*) Increased arterial pressure, due to contraction of the peripheral vessels in consequence of the local action of certain chemical and biologic irritants (lead, Bright's disease, gout, syphilis). Hassenfeld[1] has recently shown that hypertrophy of the left ventricle occurs only when the visceral arteries exhibit an extreme degree of sclerosis, or when the thoracic aorta is sclerotic. In cases of pure contracted kidney all the chambers of the heart are hypertrophied; but when extreme arterio-sclerosis is present also the left ventricle is disproportionately enlarged. In all of these cases, whether the blood-pressure is raised in larger or smaller vessels, increased cardiac action is essential to meet the demands of the system-circulation.

Attention should be called to the causes of the so-called "**primary idiopathic hypertrophy.**" The main causal conditions are—(1) Prolonged physical exertion, as in certain occupations (blacksmiths, locksmiths, draymen, and athletes). (2) Constant over-distention of blood-vessels, as in the case of excessive beer-drinkers (*beer-heart*). Here the direct action of the alcohol upon the heart-muscle must also be taken into account. (3) Functional disturbances (neuroses), constant over-action of the heart, and even paroxysmal tachycardia, tea, coffee, and alcohol may give rise to primary and general hypertrophy. Idiopathic hypertrophy of the heart is undoubtedly due to increased activity, which may be due to a variety of irritating influences acting upon the heart-muscle (De Domenicis[2]). Excessive bicycling causes hypertrophy, particularly if arterio-sclerosis exists. *Primary congenital hypertrophy of the heart* is attributable either to circulatory disturbance (Simmonds[3]) or, as Virchow holds, to a diffuse myomatous neoplasia of congenital origin.

Hypertrophy of the right ventricle develops secondarily to any condition that offers obstruction to the pulmonary circulation or to the blood-current through the right ventricle. Among them may be mentioned—(1) mitral incompetency and stenosis; (2) emphysema, bronchitis, collapse of a portion of the lung, contraction of a lung from pleural adhesions, and cirrhosis of the lung; (3) right-sided valvular lesions, particularly obstruction at the pulmonary orifice; (4) it is doubtful whether, on account of the normal situation of the right ventricle, pericardial adhesions induce hypertrophy of this chamber.

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[1] *Deutsch. Arch. f. klin. Med.*, Dec. 9, 1897; *Phila. Med. Journ.*, Jan. 22, 1898.

[2] *Wien. klin. Woch.*, May 22, 1897. [3] *Munchener med. Woch.*, 1899, No. 4, S. 108.

Hypertrophy of the Auricles.—Hypertrophy with dominant dilatation of the left auricle occurs in mitral disease, and especially in mitral stenosis. The right auricle hypertrophies, though not invariably, when the blood-pressure in the pulmonary vessels is pronounced from any cause. Stenosis of the tricuspid orifice is occasionally the sole cause of thickening of the right auricular wall, which also becomes hypertrophied in tricuspid incompetency.

Symptoms.—There is usually an entire absence of subjective symptoms when compensation is efficient. When present, their intensity varies with the degree of the hypertrophy, which is then pronounced, as a rule, and often already attended by incipient dilatation. They may be *local* entirely, though frequently *general* as well. Of the former, *precordial discomfort* and *uneasiness* from the violence of the impulse occur. They are most annoying when the patient is in the recumbent posture on the left side and when the hypertrophy is dependent upon nervous causes. *Pain* and *palpitation* are seldom complained of except by neurasthenics and patients suffering from enlargement due to tobacco or excessive muscular exertion. Decided aggravations of the local manifestations may follow the operation of influences that create a demand for increased cardiac action, such as undue mental emotion or excitement, physical exhaustion, active bodily exercise, and gourmandizing.

The *general symptoms*, when present, may fluctuate or even intermit. Those most frequently observed are fulness in the head, often amounting to actual *headache*, *tinnitus aurium*, *carotid pulsations*, *flushing of the face*, *flashing of light before the eyes, and often prominent eyeballs.* These symptoms are attributable to the increased vigor of the cerebral circulation.

Remote Effects.—General or total hypertrophy promotes high tension throughout the arterial tree. Endarteritis and arterio-sclerosis are, as a consequence, frequent consentaneous developments in advanced cases, especially when the cause of the enlargement has been increased tension in the peripheral vessels, as in Bright's disease. With a circulation too forcibly carried on, as in hypertrophy, the sclerotic vessels are overstrained, and are apt to rupture. The break often occurs in the brain (*apoplexy*) or in the lung (*pulmomary apoplexy*), and hemorrhage from the lung (*hemoptysis*), due to left ventricular hypertrophy, is more common, I believe, than is supposed. Some of the symptoms are due to the cause or causes of the hypertrophy.

Physical Signs in Left-sided Hypertrophy.—*Inspection.*—In females and in children with soft, yielding ribs there is visible bulging. The intercostal spaces are much broadened and the apex-beat covers an increased area, the extension being downward and to the left. The whole body of the patient, and even the bed on which he may be lying, may share visibly in the cardiac impulse.

Palpation.—In pronounced grades the impulse may be felt as low down as the seventh interspace and as far to the left as the axilla. In simple hypertrophy it is carried downward to the sixth intercostal space and outward to a point near the anterior axillary line. The impulse is slow, forcible, and heaving, the "thrust" lifting the fingers of the examiner. In eccentric hypertrophy (hypertrophy with dilatation), though heaving and forcible, it is somewhat more abrupt, as in cardiac dilatation. Over the aortic orifice a short diastolic impulse may also be felt occasionally (double impulse). Pressing the fingers into the second and third right spaces will detect an impulse if the aorta be dilated. The pulse in pure hypertrophy is full, strong, regular, and of normal rate; it is also prolonged, owing to increased tension. In eccentric hypertrophy it is more abrupt, soft, full, and somewhat accelerated.

Percussion.—This defines only approximately the degree of enlargement, as the hypertrophy may take a backward direction or there may be more than the usual overlapping of the heart by the lung. Traced upward, dulness may terminate in the second interspace, whilst to the left it may extend 1 or 2 inches (2.5–5 cm.) beyond the mid-clavicular line. When hypertrophy is of moderate extent the left limit of dulness corresponds with the results of palpation and inspection; but when it is of immoderate extent the extension of dulness does not keep pace with the systolic impulse, which is diffused to points beyond the limits of contact of the heart with the thoracic wall. If concomitant hypertrophy of the right ventricle be present, dulness will also extend to the right (*vide infra*).

Auscultation.—The sounds vary with the grade of the morbid process and the variety. In simple hypertrophy of marked type a prolongation of the first sound is always appreciable, and usually it is duller than the normal. The second sound (aortic) is intensified, clear, and often ringing. The degree of accentuation depends partly upon the vigor of the left ventricle, though chiefly upon the condition of the blood-vessels. Reduplication of the second sound, due to high tension, is common (*e. g.* in Bright's disease). The first sound may also be duplicated. In dilated hypertrophy the first sound is clearer and more abrupt, while the second is less marked or even faint. Modification of these sounds occurs when hypertrophy is due to chronic valvular disease.

Hypertrophy of the Right Ventricle.—One or more of the causal factors that produce augmented tension in the pulmonary vessels are present, and, if properly appreciated, will throw light upon the condition. There may be an absence of all symptoms if the hypertrophy exactly balances the result of the obstructive forces, and this state may be maintained for a long period of time. Undue exertion, however, soon leads to *temporary dyspnea* in many cases. When secondary to emphysema or cirrhosis of the lung the symptoms occasioned by the latter diseases, such as *cough* and *dyspnea*, may completely veil any symptoms that may be due to the hypertrophy. *Discomfort* in the cardiac region should, however, arouse suspicions of the existence of the latter condition. When *dilatation* of the ventricle supervenes, as is usual, and the clinical evidences of tricuspid incompetency develop, then pulmonary symptoms, due to venous congestion, are prominent; these are bronchial catarrh, shortness of breath, and the like. Later, general cyanosis and edema appear. As pointed out in the discussion of Mitral

Stenosis with permanently heightened tension and overgrowth of the right ventricle, the lung-vessels become atheromatous and the lung-tissue the seat of brown induration. Owing to the fact that the sclerotic vessels are easily ruptured, *hemoptysis*—a not uncommon event after sudden great exertion—is to be expected; intense pulmonary congestion and apoplexy may also be met with in hypertrophy with dominant dilatation.

Physical Signs.—These have been in the main detailed in speaking of affections of the mitral valve. *Inspection* discloses bulging of the sixth and seventh left costal cartilages and of the lower sternum. In the angle between the ensiform cartilage and the seventh rib an epigastric impulse may be visible, but more commonly the impulse is in the sixth interspace, close to the left edge of the sternum. It is also very generally seen to the right of the sternum, in the third and fourth interspaces, and particularly is this the case in eccentric hypertrophy, forming a highly characteristic sign. The apex-beat is therefore diffuse, the radial *pulse* is small, and in dilated hypertrophy it is increased in frequency, and is small, unsustained, and irregular.

Percussion shows the extension of cardiac dulness to a point an inch (2.5 cm.) or more beyond the right sternal border. When there is great increase transversely, dilatation is most probably associated and may predominate over hypertrophy. The *auscultatory* signs are not distinctive unless dilatation also exists, when the first sounds are clear and sharp. In simple hypertrophy the first sound is slightly prolonged and lower than in health. Owing to the high vascular tension throughout the lungs the second sound at the pulmonary valve is accentuated, and reduplication of the second sound may occur for the same reason.

It must be kept in remembrance that when advanced emphysema is present all the physical signs will be greatly modified, and may even be entirely negative, though the heart be of large size. Under these circumstances venous pulsation in the neck would be diagnostic of dilated hypertrophy of the right ventricle.

Hypertrophy of the Left Auricle.—This may be assumed to occur in mitral stenosis and incompetency in order to compensate for these lesions: it cannot, however, be recognized positively by physical signs. When the chamber is at the same time extensively dilated, the dulness may be extended upward to the left of the sternum, passing over the third and even second interspaces. At this point—the second interspace—a presystolic wave may now be noticeable.

Hypertrophy of the right auricle, associated with dilatation, is perhaps more common than its counterpart on the left side. It is secondary to tricuspid incompetency (rarely stenosis) and enlargement of the right ventricle, and hence has the same etiology as the latter conditions. The *physical signs* are—systolic jugular-pulsation, sometimes a presystolic wavy pulsation over the third and fourth interspaces to the right of the sternum, extension of cardiac dulness to the same interspaces, and other signs of tricuspid regurgitation.

Diagnosis.—The recognition of cardiac hypertrophy is possible only by attention to the physical signs. Next to these, in point of diagnostic value, come the causes, which should therefore be diligently searched for; the rational symptoms are least in value, though usually corroborative. It is difficult to establish a diagnosis, even approximately, when extensive emphysema coexists. As before pointed out, venous pulsation in the neck would point indisputably to right ventricule enlargement.

Differential Diagnosis.—Conditions that cause an increase in the precordial area of dulness, except hypertrophy, must be eliminated. (1) *Pericardial Effusion.*—A careful analysis of the physical signs and the history will suffice. (2) *Aneurysm.*—In this affection the enlargement is altogether upward and to the left or right. This fact, joined with the other evidences of aneurysm, should obviate error. (3) *Mediastinal growths* also enlarge the dull space mainly upward and to the right or left, though the point of cardiac contact may be increased and the heart carried forward. (4) *Displacement of the heart* does not give a heaving impulse nor an increased area of dulness; moreover, it usually furnishes its special cause (pleural effusion). (5) Abnormally narrow-chested persons present a considerably increased superficial zone of dulness, partly owing to the position assumed by the lungs and partly (perhaps chiefly) to their imperfect development. Since there is usually an entire absence of all other physical signs of hypertrophy, ordinary caution will exclude the latter complaint. (6) *Affections of the Lungs and Pleuræ.*—Left-sided pleurisy with retraction may, by exposing a large part of the anterior surface of the heart, give rise to signs of moderate hypertrophy. The presence of the former condition, the lack of lung-expansion on deep inspiration, the displacement of the heart to the left and upward, and an absence of the causes of hypertrophy should lead to a correct conclusion. (7) *Phthisis and cirrhosis of the lung*, with or without pleurisy, may in like manner produce apparent enlargement of the heart. It must also be remembered that cirrhosis of the lung is one of the causes of right-sided hypertrophy, and that the latter condition may therefore be present.

Prognosis and Course.—The course that will be pursued depends largely upon the stage at which the case has arrived and the character of its special cause. I have repeatedly found *postmortem* evidence of a moderate grade of hypertrophy in persons who died of other affections, and with especial relative frequency in those who had constantly followed manual pursuits. Simple cardiac hypertrophy, being compensatory as a rule, exerts in nearly all instances a salutary influence, and if the processes that constitute the causal factors are not steadily progressive, life may not only not be curtailed, but be greatly lengthened by its existence. Even in organic valvular disease of the heart hypertrophy prolongs life by overcoming the ill effects of the valve-lesion and by maintaining the normal circulatory equilibrium. But since in this class of cases the lesion is progressive despite treatment, a limit is reached sooner or later beyond which the increased vigor on the part of the heart cannot be maintained. The nutritive functions become inadequate in obedience to a natural law, and muscular degenerations then occur, followed by disturbances of the circulation due to cardiac weakness and secondary dilatation. It must, however, be recollected that the heart may at no time, in the course of certain cases, fully compensate for the causal condition—*e. g.* as when a valve ruptures with startling suddenness. Failure of the cardiac nutrition at once renders the prognosis unfavorable. The cardiac sounds now give notice that the hypertrophy no longer meets the requirements of the case. The systolic pause grows longer (with abbreviation of the first sound), and the diastolic shorter. Occasionally, as the result of undue muscular exercise, acute dilatation, followed by a speedy termination of life, is observed. I believe that hypertrophy of the left ventricle warrants a more favorable prediction than can be made in hypertrophy of the right, and this for two reasons: first, the increased capacity for work of the left ventricle; second, the milder character of the many factors that are productive of left ventricular hypertrophy, as compared with those of the right. In special instances, however, the reverse may obtain, as when left-sided hypertrophy is associated with or caused by general arterial degeneration. It may be of advantage to the student and junior physician to recapitulate here a few of the chief points that are prognostically favorable as well as those that are unfavorable: *Favorable Conditions.*—(1) When the hypertrophic development fully compensates the causal lesion; (2) when the causes are removable or more or less amenable to treatment; (3) when the external conditions under which the patient lives, his habits, and general nutrition are good. *Unfavorable.*—(1) When signs of imperfect nutrition of the heart arise; (2) when evidences of advancing cardiac dilatation (dyspnea, rapid, irregular pulse, edema) show themselves; (3) when poverty, poor food, intemperate habits, and an unhygienic environment are all combined; (4) when apparent cardiac vigor suddenly gives place to dilatation and great cardiac weakness.

The **treatment** has for its prime objects the establishment of full, and the prevention of failure of, compensation (*vide* Chronic Valvular Disease).

Over-hypertrophy, as indicated by certain cerebral and thoracic symptoms, may require the employment of measures to reduce the contractile energy of the left ventricle, although direct cardiac depressants (aconite, and the like) are rarely needed. It requires careful dietetic and hygienic management. Briefly, the *diet* should be nutritious, but the more concentrated forms of food should be used very sparingly, and the daily quantity should be slightly less than that required in health. It must be non-stimulating, and tea, coffee, alcohol in all forms, and smoking must be prohibited. The physical exercise should be moderate in amount and of the gentlest sort; and if the patient's occupation tends to stimulate the heart, it must be immediately abandoned. A mild saline purge (ʒij to ℥ss—8.0 to 16.0—of Rochelle salts once daily) is quite beneficial.

For relief of the cerebral symptoms (tinnitus aurium, vertigo, fulness) and the precordial discomfort the physiologic relaxants of the capillaries and the arterioles are of great service, particularly when arterio-sclerosis is a traceable cause. Among them nitroglycerin in full doses and veratrum viride are most useful; the efficacy of both may often be enhanced by the addition of the bromids. In cases of nervous origin the bromids, with preparations of valerian, are the most valuable agents. Nothing, however, is of higher importance than the determination and removal of the cause when possible. After compensation has failed the further treatment is identical with that of cardiac dilatation.

DILATATION OF THE HEART.

Definition.—By dilatation of the heart is meant an enlargement of its various cavities. The walls of the chambers may in consequence be thinner than in health, but much more commonly they are thicker, as in *dilatation with hypertrophy.* Both hypertrophy and dilatation are relative terms, but the latter has reference to that condition in which the cavities are distended out of proportion to the diameter of their walls.

Varieties.—(1) *Dilatation with Hypertrophy.*—Here there is a progressive increase in the capacity of the chambers until they attain to large dimensions. The cardiac walls continue of abnormal thickness, yet the vigor of the divisions affected may be relatively diminished to a remarkable degree, owing to the weakening influence of the degenerative processes that attack the hypertrophied muscles. In eccentric hypertrophy the heart-cavities are dilated, but the hypertrophied cardiac walls are sufficiently vigorous to meet the demands of the circulation. This condition should not be regarded as identical with *dilatation with hypertrophy*, but frequently merges into the latter, the size of the cavities now being proportionately greater than is the thickness or the functional power of their walls.

(2) *Dilatation with Thinning of the Heart-walls.*—The diminution in the diameter of the cardiac muscles may be slight if the capacity of the chambers involved be only moderately increased. Instances of this sort are sometimes seen to follow prolonged fever (typhoid). On the other hand, the process of attenuation may reach a high grade, the greatly thinned cardiac wall being scarcely capable of holding the weight of the contained blood.

(3) *Dilatation with little or no variation from the normal cardiac wall* has also been described by some authors. It is to be observed, however, that stretching of a cavity whose walls are of normal thickness must be attended with thinning of those walls.

Pathology.—Dilatation with hypertrophy is generally secondary to valve-lesions, and affects more than one cavity as a rule. It may happen, as in advanced aortic regurgitation, that all the divisions are dilated. The right ventricle is somewhat more frequently dilated than the left, however, for reasons previously adduced. The auricles (especially the left) are more frequently expanded than the ventricles; hence of all the chambers the left ventricle is least apt to dilate. The extent of the relative increase in the capacity of the cavities is variable, and often remarkable. As an example of extreme dilatation of a chamber, the left auricle in cases of mitral stenosis may be singled out; I have seen an instance in which this auricle was capable of containing twenty-two ounces of blood. The septum may be seen to bulge when one ventricle only is stretched. Extensive dilatation of the chambers produces a dilated condition of the auriculo-ventricular rings, which in turn gives rise to relative incompetency. Other cardiac orifices are found to be similarly dilated. Dombrowski[1] has drawn attention to the fact, first pointed out by Wolf, that the surface of the mitral leaflets greatly exceeds the orifice, and Kirschner and Garcin contend that the anterior flap alone suffices to close the mitral orifice, "even when the left heart is considerably dilated." Dombrowski believes that functional incompetency is due, in many cases, "to muscular dilatation, producing a separation of the insertions of the papillary muscles, which in systole cannot approach each other near enough to allow the valves to close, the contraction of the papillary muscles only increasing the difficulty." Great dilatation of the left auriculo-ventricular ring is, however, probably an important factor in the causation of relative mitral incompetency. The tricuspid valves, being scarcely competent, normally, are unquestionably incompetent when that orifice is considerably dilated.

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[1] "Functional Insufficiency of the Valves of the Left Heart," *Revue de Médecine*, Sept. 10, 1893.

The *shape* of the heart is altered according to the seat and extent of the dilatation. When all the cavities are dilated the organ assumes a globular form, while dilatation of the ventricles only produces broadening of the apical region.

Condition of the Endocardium and Cardiac Muscle.—The muscular tissue generally exhibits degenerations (fibroid, fatty, or parenchymatous). Important as is the part played by the ganglia in maintaining the nutritive integrity of the heart by supplying nervous force, our knowledge of the alterations that may occur in them in this condition is as yet very imperfect. Ott and others have, however, found them to be degenerated. Opacity and patchy roughening of the endocardium are common. The parietes and endocardium may, however, have a normal color and structure.

Etiology.—Entering into the causation of cardiac dilatation, there are two essential factors: (1) increased endocardial tension; (2) diminished resistance. These often act together. Broadbent contends that the special feature of dilatation is the imperfect emptying of the ventricles.

(1) **Increased Endocardial Tension.**—It is to be premised that a primary and a secondary form occur, the latter being of greater importance clinically than the former. Primary dilatation occurs from a recent obstruction to the circulation of considerable magnitude and at any point throughout the blood-vessel system. A good example is afforded by aortic constriction, in which condition the obstruction of the aortic ring engenders dilatation of the left ventricle by raising the intraventricular pressure; this is quickly overcome by compensatory hypertrophy. In the vast majority of these instances the nutrition of the muscular fibers eventually suffers, with consequent dilatation. *Angiospastic dilatation* is a condition due to acute transitory spasm of the vessels (Jacob).[1]

Other causes of augmented endocardial pressure have been considered in the discussion of Hypertrophy and Chronic Valvular Lesions. In eccentric hypertrophy dilatation is a compensatory arrangement, until finally the cardiac nutritive functions fail and dilatation at once predominates (dilatation with hypertrophy). Compensation has now been ruptured. Among the exciting factors that may precipitate this accident are—recurrent endocarditis, intercurrent febrile affections which overstimulate the heart and impair its muscular tissue, general disturbances of nutrition, and physical and mental overstrain.

Acute primary dilatation may be brought about by sudden, great exertion, as in ascending mountainous elevations, excessive bicycling, and the like. Under these circumstances the heart palpitates violently, and there are epigastric pulsation and often pain in the cardiac region—evidences of dilatation of the right ventricle. Although the heart's reserve capacity for work has been exceeded, rest followed by quite moderate exercise often restores the conditions to the normal. I have seen acute primary dilatation produced by strong emotion; in such cases sudden contraction of the peripheral vessels occurs, attended with arrest of the heart's action; this soon gives place to violent palpitation, and rarely to dilatation (angiospastic dilatation). Sudden fright may act similarly.

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[1] *Zeitschr. f. klin. Med.*, Feb. 4, 1899.

The remarkable endurance of the athlete and the gymnast is in part owing to the abnormal amount of physiologic cardiac reserve force which they naturally possess, but it is mainly due to the invigorating effect of training. If, however, the training be not so conducted as symmetrically to develop the entire muscular system, or if the exertion be in excess of the reserve functional power of the heart, then acute dilatation may suddenly arise. From this accident (cardiac fatigue) recovery may take place; sometimes, however, it initiates organic valvular disease, and thus prohibits the further undertaking of unusual feats. Acute dilatation has been made conspicuous by recent contributions, in which bicycling is assigned as the cause.

Apparently idiopathic cases of cardiac dilatation of indeterminate etiology rarely occur.

(2) **Diminished Resistance owing to Weakened Cardiac Walls.**—The occurrences that weaken the cardiac wall are numerous, and not a few lead to acute primary dilatation, such as *myocarditis* due to acute specific fevers (scarlatina, typhoid, malaria, typhus). It is especially prone to occur in *rheumatic endocarditis* and *pericarditis.* B. Robinson [1] calls forcible attention to serious dilatation due to the toxic action upon the heart muscle of the rheumatic poison. The *chronic degenerations* (fatty, fibroid) impair the contractile power of the heart. *Nutritional disturbances* of varied origin, such as digestive disorders, ill-ventilation, lack of open-air exercise, and improper or defective food-supply, may induce enfeeblement of the cardiac muscle. Dilatation is met with also in *diseases of the blood* (chlorosis, anemia, leukemia).

Clinical History.—In *acute* dilatation the *onset* is sudden. It is accompanied by rapidly augmenting dyspnea and cardiac palpitation, a feeling of coldness, and frequently by pain in the precordial region.

The **physical signs** may be incontestable. They are *venous pulsation* in the neck, a *rapid, feeble apex-beat*, and a *systolic murmur* at the tricuspid valves, all of which declare the presence of tricuspid regurgitation. In *angiospastic dilatation* the pain may begin in the extremities, and the second heart-sound may be louder at the apex than the first. Among signs of subsidiary value are a venous turgescence, a marked epigastric pulsation, and a sudden extension of *dulness* to the right; the *pulse* is small, irregular, and exceedingly rapid.

In the more *chronic form* which arises from slowly-acting causes, or in that which accompanies eccentric hypertrophy or follows simple hypertrophy due to left-sided heart- or lung-trouble, the manifestations that characterize the earlier stages are not at all striking. They indicate weak heart-walls, and such chambers expel their contents imperfectly during systole. Hence with each subsequent diastole the abnormal amount of blood contained in them is increased. This blood-stasis, as previously pointed out, often extends from the left heart to the pulmonary vessels, from the latter to the right heart, and finally to the general venous system. Both in the acute and chronic forms, however, *failure of the right ventricle* more often determines rupture of compensation. Obviously, the symptoms must be those of organic diseases of the heart (tricuspid incompetency in particular). Dilatation of the right heart, without tricuspid insufficiency, is a frequent complication of pulmonary tuberculosis (Maisonneuve[1]).

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[1] *American Journal of the Medical Sciences*, Dec., 1899.

Physical Signs.—*Inspection* in dilatation of the left ventricle shows the apex-beat to be displaced outward and downward, and a diffuse, weak, fluttering, and often distinctly undulating impulse. The apex-beat will show a greatly diminished vigor in its normal area; or there may be no recognizable point of strongest impulse as in health. Distinct pulsation in the second left interspace is not rare. Its feebleness and diffuse character are confirmed by *palpation*. It may be quick and sharp, though always lacking in power. Walsh first made the capital observation—since abundantly corroborated—that the impulse may be visible, yet not palpable. There may be a mere vibration or an utter absence of the apex-beat in advanced cases. The pulse is small (rarely large), short, often rapid, and irregular. *Percussion* shows a lateral increase in dulness to the left, to or even beyond the mid-clavicular line, upward to the second rib, and downward as far as, though rarely below, the sixth interspace, except perhaps, in rare instances, in dilatation with hypertrophy. In emphysema the lungs unduly overlap the heart.

Dilatation of the right ventricle demands separate consideration so far as the impulse and percussion-dulness are concerned. The normal impulse is largely replaced by the abnormal apex-beat of the right ventricle, which advances to the anterior chest-wall. The chief impulse is now seen and feebly felt, as a rule, below the xiphoid cartilage, or, less commonly, to the right or left of the latter. A wavy pulsation is seen to the left of the sternum, over the fourth, fifth, and sixth interspaces and close to its right edge. If dilatation of the right auricle be associated, as is often the case, a distinct pulsation also occurs in the third right interspace. Dulness reaches to a point 1 inch (2.5 cm.) or more beyond the right sternal border on a level with the fourth interspace.

On *auscultation* variable results are obtained according to the state and diameter of the cardiac walls. When thin and not much disorganized, the first sound is much shorter, sharper, and louder than in health. In advanced cases the systolic sounds may be feeble, though almost always audible in the aortic area (unlike the first sound in hypertrophy). The first closely resembles the second sound, the long pause being shortened, resembling the systolic pause (*fetal heart-sounds*). This form of arrhythmia is a serious indication of failure of the ventricles. The *canter rhythm* is equally common. Irregular and intermittent cardiac action are usual phenomena. Reduplication may occur, but is not frequent.

Pre-existing *organic murmurs* obscure the sounds due to dilatation, and, on the other hand, the dilatation may also alter the murmurs (previously audible), and even cause them to disappear, as, for example, in mitral stenosis. Again, dilatation may induce relative incompetency or superadd a murmur, as in cases of chronic valvular disease at the auriculo-ventricular orifices. It is interesting to recall here that proper treatment may remove a murmur due to relative insufficiency, and that this treatment may, in turn, reproduce an organic murmur.

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[1] *Gaz. hebdom. de Méd. et de Chir.*, Oct. 30, 1898, No. 45; Année, No. 87.

Diagnosis.—This is made readily when there is obtainable a clear history, together with the following characteristic features: a weak, irregular heart-action; an extended, wavy impulse; a small, vigorless, irregular, and intermittent pulse; often an indistinct apex-beat; an outward, upward increase in the percussion-dulness on one or both sides, causing the outline to resemble a square; and a brief, sharp, yet feeble first sound that strikingly resembles the second, which is itself enfeebled.

Differential Diagnosis.—*Hypertrophy*, like dilatation, gives rise to an extended area of impulse and of percussion-dulness; hence by the careless observer these conditions are sometimes sadly confounded. From dilatations, in which the diagnosis rests upon the points above enumerated, hypertrophy is to be distinguished by symptoms of an opposite nature, such as indicate increased energy on the part of the heart. The latter are—a slow, heaving impulse; a full, sustained, regular pulse; an increase in the area of dulness, chiefly outward and downward; abnormal position of the apex-beat; and the prolonged, dull first and accentuated second sounds. To determine the point at which eccentric hypertrophy ends and dilatation (with hypertrophy) begins is often difficult; but a careful discrimination must be attempted, and I have already discussed the ushering-in symptoms of dilatation following hypertrophy (chiefly of the right ventricle) in connection with Chronic Valvular Disease. Occurring in left ventricle hypertrophy, dilatation first betrays itself by a change in the position of the visible apex-beat and the palpable impulse. Thus, the maximum point of the apex-beat of hypertrophy very early becomes rounded and indefinite, and later is diffuse and wavy. The strong, heaving thrust of the impulse gives place to the shorter, more sudden shock of commencing dilatation, indicating weakness. These signs, together with a reduction in the strength and an increased frequency or irregularity of the pulse, show the condition to be dilatation with hypertrophy.

The **prognosis** is bad, as a rule, and may be said to be that of the causative factors.

Treatment.—This in all essential particulars is identical with the treatment of organic heart-affections after rupture of compensation. The etiology in many cases differs from that of the organic valvular affections of the heart; and next to rest and the use of cardiac stimulants, the removal of the remote and near causes of the dilatation is the most important part of the treatment. Individual cases frequently present special indications; but in all the work of the heart is increased and the propulsive power of the organ diminished. In cases of non-valvular origin digitalis and other heart-stimulants may be omitted early, as a rule; though they should be resumed if there be a recurrence of serious indications of dilatation. When the dilatation has been overcome careful attention is to be bestowed upon all the details of the patient's life and sanitary surroundings in order to force his bodily nutrition to the utmost. Every precautionary measure having for its aim the prevention of a recurrence of the dilatation must also be advised and enjoined.

MYOCARDITIS.

(*Carditis.*)

Definition.—An inflammation of the muscle-substance of the heart. It may be acute or chronic.

ACUTE MYOCARDITIS.

Pathology and Varieties.—(1) **Acute Parenchymatous Myocarditis.**—This is characterized by a granular degeneration of the muscular fibers of the parenchyma of the organ, with a numerical increase in their nuclei. The muscle-structure throughout looks pale, is turbid, and very soft. Many cases of a severe type terminate in fatty degeneration.

(2) **Acute Diffuse Interstitial Myocarditis.**—Here the primary alterations affect the connective tissue of the myocardium; the histologic changes consist in round-cell infiltration.

(3) **Acute Circumscribed Myocarditis.**—In this variety the degenerative processes result in necrosis of the tissues over large or small areas, with abscess-formation. Though usually multiple, these abscesses vary considerably in number, and may rupture either into the various cardiac chambers or into the pericardium. Thus, the purulent contents of the abscess, when there is established a fistulous communication with an endocardial chamber, find their way into the blood-stream and are conveyed to all parts of the arterial system, frequently setting up, here and there, embolic processes of an infectious nature. The blood in turn enters the abscess-cavity, exerting pressure on the walls, and may either produce an acute aneurysmal dilatation of the heart-wall or occasion fatal rupture into the pericardium. More frequently, perhaps, the connective-tissue wall of the abscess yields gradually during the ventricular diastole, when the cardiac aneurysm is formed with corresponding slowness. Occurring in the vicinity of one of the auriculo-ventricular valves, abscesses may cause mitral or tricuspid incompetency. Owing to their tendency to burrow, they may perforate the interventricular septum, thus creating a fistulous connection between the two sides of the heart, and resulting in an intermingling of venous and arterial blood. The abscess may become encysted, then caseous, and finally undergoes a calcareous process. Multiple abscesses usually affect the left ventricle.

Etiology.—*The causes of myocarditis* are—(*a*) endo- and pericarditis in the course of rheumatism: it is probable that rheumatic myocarditis may also exist without involvement of the endo- or pericardium; (*b*) the infectious processes in acute specific fevers; (*c*) infectious emboli, lodging in the branches of the coronary arteries in connection with septicemia, pyemia, and acute ulcerative endocarditis, and commonly terminating in abscesses (circumscribed myocarditis). The first two of these causes give rise to acute diffuse interstitial and acute parenchymatous myocarditis as a rule, although Freund calls attention to the frequency with which circumscribed myocarditis is associated with rheumatism and diseases of the joints. As compared with the female ***sex***, the male suffers much more frequently.

Symptoms and Diagnosis.—The symptoms are practically negative. They point merely to great cardiac enfeeblement. When cardiac weakness, as shown by a rapid, small, compressible, and irregular *pulse*, and by attacks of *cardiac palpitation* and *syncope*, comes on suddenly in the course of rheumatism, septicemia, or other causal affections, myocarditis may be suspected. Later, signs of venous stasis appear. The *mental symptoms* may suggest meningitis or salicylic-acid poisoning. Koplik[1] calls attention to certain symptoms (pallor, faintness, vomiting, irregular, feeble heart-action, disturbance of the respiration and pulse-ratio) that should arouse suspicion of myocarditis in the course of an infectious disease in childhood.

The **physical signs** simulate those of dilatation, and may, indeed, be largely dependent upon the presence of the latter condition. Early the action of the heart is tumultuous; the sounds on auscultation are short, sharp, and finally very feeble. *Murmurs* in myocarditis are not rare, and are not necessarily dependent upon dilatation. Kiehl's work shows the dependence of the valves for their complete closure upon a normal state of different portions of the heart-muscles, and thus explains these murmurs. The special conditions rendering the murmurs audible are great dilatation, softening of the papillary muscle, and abscesses near the valves.

For the recognition of *cardiac aneurysm*, see p. 660. The symptoms of visceral or cutaneous *embolic processes*, combined with a murmur and a septic type of fever, are suspicious of the existence of circumscribed myocarditis The great variability as to the intensity of these murmurs is an important point, especially in attempts to discriminate from murmurs due to *endocardial changes*. The latter usually coexist with a more marked accentuation of the pulmonary second sound.

Prognosis.—The diffuse forms are fatal; the circumscribed form may, however, end in recovery. Myocarditis may end life suddenly.

The **treatment** is identical with that indicated for endocarditis and pericarditis—diseases of which myocarditis is often a complication. The effects of digitalis, particularly when myocarditis supervenes upon old heart-lesions, are quite unsatisfactory. When myocarditis is suspected as an independent condition absolute rest must be enjoined, the general nutrition maintained, and the more urgent symptoms relieved.

CHRONIC MYOCARDITIS.

(*Fibrous Myocarditis.*)

Definition.—A gradually developing inflammation of the cardiac interstitial connective tissue, resulting in induration.

Pathology.—The characteristic changes may be diffuse, though most frequently they are confined to certain portions of the muscular structure, the left ventricular wall, the septum, and the papillary muscles being the three favorite seats of the process. This is sometimes of antenatal development, and then its usual seat is near the apex of the right ventricle. The hardened spots take the form of more or less rounded patches or broad lines. In color they are gray, grayish-white, or grayish-yellow, the latter tint being due to the intermingling of fibers that have undergone fatty degeneration. Their size is exceedingly variable, some being so minute as to elude detection by the unaided eye, while others measure 1 or 2 inches (2.5–5 cm.) in diameter. Inflammatory induration (contraction, of the *conus arteriosus* of either ventricle causes narrowing of the pulmonary and aortic orifices, with the usual signs and symptoms. Similar changes, by disturbing the functions of the papillary muscles, produce valvular incompetency. Compensatory hypertrophy of the uninvolved portion of the heart is also observed, both the size and weight of the organ thus being increased; the hypertrophic enlargement may frequently be accounted for in part by an associated chronic endocarditis. Sometimes, however, the hypertrophy is occasioned mainly by general arterial sclerosis. Dilatation of the ventricles follows, with fresh and grave disturbances of the circulation.

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[1] *Medical News*, March 31, 1900.

Chronic inflammation usually attacks early the intima of the coronary arteries, and leads to thrombosis, with the formation of anemic infarcts that subsequently undergo sclerotic changes in the muscle-structure. It is probable that most cases of localized fibrous myocarditis have their origin in an obliterating endarteritis. Pasquier offers proof that fibroid myocarditis results from chronic congestion due to stopping of the vessels. The calloused zone may yield to the endocardial blood-tension, and thus slowly produce saccular dilatation (aneurysm). *Microscopically*, the affection is characterized by hyperplasia of the interfibrillar connective tissue with subsequent development of new fibrous tissue. Fatty degeneration and atrophy of the muscle-fibers (the latter in consequence of compression) are also observed. Fragmentation of the muscle-fibers has also been observed. This occurs as a *postmortem* change, and is due to a softening of the interfibrillar substance (the *état ségmentaire* of Renant).

Etiology.—The disease is most commonly traceable to the action of one or more of the following factors: an *excess in the use of alcohol* or *tobacco*, *lead-poisoning*, *gout*, *rheumatism*, *diabetes*, *chronic nephritis*, *malaria*, and *syphilis*. Thus, it may be produced by many infections and chemical irritants, the latter, in most cases, first causing a sclerosis of the coronary arteries, to which the patchy fibroid degeneration is secondary. Some of the causes of acute diffuse interstitial myocarditis may by their more slightly irritant effect (owing to the minuteness of the dose of the specific poison) lead to the subsequent development of the general chronic form (*e. g.* rheumatism). Certain irritants that usually engender localized lesions of chronic myocarditis may also affect the entire myocardium (syphilis, alcohol, gout). Chronic myocarditis may arise in consequence of a *direct extension* of the infective inflammatory processes in chronic endo- and pericarditis; it may also follow *injuries* of the antero-lateral thoracic region. *Sex* and *age* possess a predisposing effect, the disease being more common in males than in females, and after middle life than before that period. The right ventricle is apt to be the seat of chronic myocarditis during fetal life, if at all.

Symptoms.—Extensive indurated myocarditis has been met with *post mortem* in numerous instances that have been unattended by perceptible symptoms during life. In many of these cases the presence of compensatory hypertrophy accounts for the absence of any symptoms, and it may, therefore, be inferred that mild grades that fail to manifest themselves must frequently exist. The symptoms when present are, almost without exception, untrustworthy for diagnostic purposes, since they bear a striking resemblance to those of the organic valvular diseases, minus their more characteristic physical signs. Among the earliest phenomena that point merely to failing heart-power are *dys-*

pnea, and sometimes also, on exertion, *palpitation* and a *sense of heaviness or constriction* in the precordia. The patient suffers from marked general debility, and becomes *fatigued* in consequence of the slightest physical exertion. *Mental inertia* is the rule, and *chronic mania* may come on and last to the close. Later, more positive disturbances of the circulation gradually arise, and when the breathing becomes more difficult (*cardiac asthma*) signs of *venous stasis* affecting the liver, gastro-intestinal tract, and kidneys, and edema finally appear.

Two symptoms that are frequently manifested, and not without some diagnostic import, remain to be mentioned: (1) *Angina pectoris*, which is attributable to the sclerosed condition of the coronary arteries. (*Vide* Symptoms of Angina Pectoris, p. 676.) It is often followed by some form of arrhythmia. Cases occasionally occur in which recurring paroxysms of angina pectoris, with or without arrhythmia, are the only phenomena of the disease.

(2) *Cardiac Arrhythmia.*—Brachycardia is associated as a rule, there being a reduction in the pulse-rate to 50 or even 40 beats per minute. With this decreased rate intermittency is often combined, and various other forms of disturbed rhythm are also observed, though they are less frequent and less significant. Slowing of the pulse does not, however, prohibit the cardiac palpitation that is especially apt to arise during anginal attacks. Disturbance of the rhythm may, on the other hand, be entirely absent.

The *pulse* is slow, irregular, and of low tension if cardiac *atrophy* be present. Should *fatty degeneration* be conjoined, the pulse may be quickened and irregular, and this effect likewise obtains when the patient escapes sudden death and the usual dilatation supervenes.

Chronic myocarditis may be the sole cause of the *pseudo-apoplectic seizures* that often terminate life abruptly. Preceding the unexpected attack the patient, usually advanced in life, may have experienced from time to time slight vertigo, syncope, and oppression. These seizures may also be caused by a heavy meal or intense mental or physical exertion, and may consist in a momentary loss of consciousness, paralytic symptoms then being usually absent. At other times they last a number of hours, and are accompanied by paralysis which outlasts the coma, as a rule, by a few hours only. Convulsive twitchings may be present. During the attack cerebral hemorrhage occurs, and may leave the patient hemiplegic. It is highly characteristic of these pseudo-apoplectic seizures that they tend to recur, sometimes at intervals of a few hours for a day or two, but more commonly at longer intervals during many weeks or months.

Physical Signs.—The impulse may be feebly heaving (sometimes absent); the apex-beat is displaced downward and to the left, while the dull area is enlarged correspondingly in the same direction. Quite early the heart-sounds may be clear and strong, but subsequently they become weak and muffled. A contraction of the papillary muscles and of the chordæ tendineæ may cause mitral incompetency with its customary murmur.

With the occurrence of dilatation also comes an apical, systolic murmur (due to relative incompetency), with a gallop rhythm of the heart.

Differential Diagnosis.—(1) *Chronic valvular disease* can, as a rule, be eliminated prior to the occurrence of secondary dilatation. During this period murmurs do not occur unless the valvular adnexa (the chordæ and papillary muscles) are affected. In the latter event the secondary alterations in the heart, the symptoms, and whole course of the complaint are the same as in certain chronic valvular lesions.

(2) *Hypertrophy and Dilatation.*—In chronic myocarditis *hypertrophy* does not usually reach as high a grade of development as in the majority of the organic valvular complaints and other causal conditions. But after the occurrence of *dilatation*, following indurated myocarditis, the differential diagnosis between the latter and eccentric hypertrophy is purely conjectural.

(3) *Fatty overgrowth* must be distinguished from fibrous myocarditis, and is met with chiefly in brewers, publicans, and butlers. The disease is also found to be specially related to obesity, and sometimes to overeating and drinking, combined with indolent habits. These subjects suffer more frequently from bronchitis, emphysema, and nocturnal asthma than patients having chronic myocarditis alone. Slight vertigo is common, but true syncopal attacks are rare, according to my observation. In fatty overgrowth the heart-sounds are weak and decidedly muffled throughout; the pulse is weak, though regular as a rule. Marked obesity, however, often obscures the local signs.

Prognosis.—Chronic myocarditis is a fatal disease. Its course and duration, however, are subject to great variations. Among unfavorable surroundings are certain causal and associated conditions, particularly arterio-sclerosis, chronic interstitial nephritis, and diabetes mellitus. On the other hand, if syphilis has been the cause, hope for temporary improvement, if not for actual cure, may be reasonably entertained. Sudden death may result from a blocking of a vessel that is the seat of sclerosis.

Treatment.—The treatment should be managed according to the considerations pointed out in the treatment of Organic Valvular Disease. Rest of body and mind is imperative. Next to this come the dietetic and hygienic details. Residence in a mild climate in winter and a change to the country or to a moderate elevation in summer are matters of the greatest moment to the welfare of the patient. Those rather frequent cases that present, among other complications, such closely united conditions as arterio-sclerosis, gout, and chronic nephritis sometimes do well while sojourning at certain mineral springs, such as Marienbad, Carlsbad, Kissengen abroad, and Bedford or Saratoga at home. These waters must, however, be cautiously used.

When dilatation arises cardiac stimulants are called for, but must be used with an unusual degree of caution. Strychnin has proved itself to be valuable if perseveringly exhibited, and here, as elsewhere, digitalis deserves a trial; its careless administration, however, may give bad results if the pulse be much retarded or arterio-sclerosis coexist. For the angina pectoris morphin, administered hypodermically, is to be preferred. Recurrences of this distressing symptom may be averted by the cautious use of nitroglycerin, the use of which should, however, be limited to cases that seem to be dependent upon arterial degeneration with high tension. Attacks of syncope are most successfully met by the hypodermic use of the diffusible stimulants (ammonia, ether), and at the same time by putting the patient at rest with the head lowered.

DISEASES OF THE CORONARY ARTERIES.

It has previously been noted that in pyemia and allied disorders septic emboli may block the branches of the coronary arteries, causing suppurative infarcts (acute circumscribed myocarditis).

It has also been shown that one of the chief effects of sclerosis affecting the coronary arteries is the production of *chronic myocarditis.* Sudden blocking of one coronary artery by an embolus causes instant death. In numerous instances in which death has occurred suddenly either thrombotic or embolic obstruction has been the only discoverable *post-mortem* lesion. In others the pathologic evidences of local or general atheroma have coexisted. Ligation or plugging of the coronary vessels in the lower animals causes arrhythmia or even an abrupt arrest of cardiac action; a partial or even slight reduction in the lumen of the coronary vessels by diminishing the supply of blood to the heart-muscle induces degenerations in the latter. Kronecker found that occlusion of the coronary arteries by injecting paraffin caused the heart to become irregular, even when it solidified in only the smaller branches, and stopped almost at once. The anatomic peculiarity of the coronary arteries in that they are end-arteries is to be noted, since it affords a ready interpretation of the usual effects following total or partial occlusion. According to F. H. Pratt, however, the vessels of Thebesius, which extend from the auricles and ventricles to the myocardial capillaries and coronary veins, may rarely maintain the nutrition of the heart-muscles even after occlusion of the coronary arteries.

The blocking of the terminal branches by emboli or by the more gradual formation of thrombi usually produces the so-called *anemic necrosis* or *white infarct*—a condition that richly deserves brief description:

Anemic necrosis (*anemic infarct*) is met with most frequently in the left ventricle and septum, which receive their blood from the anterior coronary artery. The involved areas are small and circumscribed, and present irregular margins that project slightly above the surface. Rarely the infarct is wedge-shaped. Its color is grayish-white or grayish-red, while the central portion is often distinctly white and firm; less frequently it breaks down into a soft detrital mass (*myomalacia cordis*). When softening does not occur the fibers in the affected area lose their nuclei, becoming first hyaline and subsequently sclerotic. The chief histologic changes are of two sorts: (*a*) the striæ of the muscle-fibers are lost, the latter becoming granular and breaking down; and (*b*) the fibers assume a homogeneous hyaline appearance, the nuclei having disappeared.

The *symptomatic* consequences of the lesions are often obscure and unreliable. Sudden death may take place, and rarely this accident may be due to rupture of the heart. Weak and irregular action of the heart, evidences of embarrassed circulation (especially in the cardiopulmonary circuit, as shown by cough and dyspnea), and finally angina pectoris, are among the principal features observed. Death may ensue in the first attack. The paroxysms are presumed to be due to sudden occlusion of a branch of the coronary artery; but it should be stated that occasionally in fatal instances of true angina pectoris a total absence of lesions, including emboli, has been noted. I desire to lay stress upon the medico-legal importance of coronary disease; it may be the only lesion found in cases of quick death.

DEGENERATIONS OF THE HEART.

(*a*) **Fatty.**—The term "fatty heart" includes two pathologically distinct affections: (1) Fatty degeneration, in which the cardiac muscle-fibers have been converted into fat; and (2) Fatty overgrowth, in which an abnormal quantity of fat is deposited in and about the heart. According to Leyden, the cases of "fat-heart" (fatty overgrowth) are divisible into two subclasses: (*a*) fatty overgrowth, and (*b*) fatty infiltration.

FATTY DEGENERATION.

Pathology.—The condition may be either general or localized. Its most frequent seat is in the left ventricle, the papillary muscles and trabeculæ, first appearing as yellowish spots or stripes beneath the endocardium. The affected portions are light yellow or yellowish-brown (faded leaf) in color, due to an associated brown atrophy; they are also soft and friable, and are easily lacerated. The heart is enlarged, and often decidedly so if the process be general, and its walls lack firmness. The microscope reveals characteristic changes: the striæ and nuclei begin to fade, oil-drops and granules appear in the fibers, and finally the latter are occupied throughout by minute globules.

Etiology.—Fatty degeneration has already been mentioned as occurring in both the *primary* and *secondary* forms of *cardiac hypertrophy*. It is found also in association with fatty change in other organs in severe forms of *primary* and *secondary anemias*. It is most commonly encountered, however, in the *cachectic states* produced by such chronic diseases as carcinoma and phthisis, and in the course of *acute infectious diseases* of intense type, all of which may produce the condition. In poisoning by arsenic and phosphorus and in pernicious anemia it advances to a high grade. The various lesions of the *coronary arteries* previously considered bear the most significant causal relation.

Predisposing causes are—(*a*) *age*—it being most common after forty years of age; (*b*) *sex*—it occurs somewhat more frequently in men than in women, notwithstanding the fact that there are predisposing influences at work in the latter that do not obtain in the male sex, such as childbirth and amenorrhea; and, lastly, (*c*) whatever may be its apparent etiology, it is invariably preceded by a defective nutritive supply to the muscle-cells: this may be dependent upon a narrowing of the lumen of the coronary vessels, or upon impairment of the oxygen-carrying power of the blood, as in the anemias. An excessive supply of glucose, glycogen, and nuclein may be a factor.

Symptoms.—The disease may exist in an advanced form without noticeable symptoms, though the conditions under which it is most liable to occur afford secure ground for suspicion. The evidences of *cardiac* *enfeeblement* are usually present, but in pernicious anemia and chlorosis the pulse may even be full and regular.

Dilatation is apt to supervene early, owing to the weakened state of the heart; and hence it is probable that many of the symptoms that have been ascribed to the fatty changes are in reality due to secondary dilatation. Among these are *palpitation*, *dyspnea*, a *small*, *irregular*, and somewhat *quickened pulse*, and *cool* and *clammy extremities*. The heart-sounds are weak, as a rule, and the action of the heart often irregular; later the physical signs of dilatation are almost invariably present. *Dropsy*, however, is rare in uncomplicated cases. Sometimes sudden, great physical exertion produces equally sudden dilatation, whereupon a canter rhythm and an apical systolic murmur speedily develop. In most instances, however, the symptoms are more gradually brought to light. *Breathlessness* on exertion is often a striking feature, and syncopal attacks are sometimes troublesome. The *pulse*, in consequence of irritation of the inhibitory center in the medulla, often becomes greatly retarded, dropping from the normal rate to 30 or 40 beats per minute, and, in rare cases, to 10 or 12 beats. The fatty *arcus senilis* is devoid of diagnostic value. There are frequent attacks of *cardiac asthma* in the mornings, and these are apt to be accompanied at intervals by *angina pectoris*. *Disturbance of the intellect*, sometimes taking the form of maniacal delusions, may come on and persist. Syncopal attacks occur. *Pseudo-apoplectic attacks*, such as have been described (*vide* Chronic Myocarditis), may occur. *Cheyne-Stokes breathing* is among the later manifestations. It happens that this symptom and pseudo-apoplectic seizures are often found in association; they are more apt to be due to uremic toxemia, perhaps, than to fatty degeneration of the heart. Epileptiform attacks resembling *petit mal* may arise.

The **diagnosis** is sadly obscure. The history, the age of the patient, and the symptoms of cardiac weakness and subsequent dilatation, together with retardation of the pulse, apoplectic attacks, and Cheyne-Stokes breathing, in the absence of precedent hypertrophy merely justify a probable diagnosis. With a clear history and the presence of the more significant symptoms, including the signs of dilatation following hypertrophy, fatty changes may be inferred with some degree of assurance, although a positive opinion should be withheld.

The **prognosis** is as varied as the etiology. Death may come quickly, the process being commonly associated with sclerosis of the coronaries, though oftener the end is reached in a gradual manner, the signs and symptoms of advanced dilatation dominating the closing scene.

Treatment.—The cause in each individual case should be determined with as much precision as possible, and when ascertained a bold attempt should be made to remove it. This course often places the patient in the most favorable position for the successful treatment of the cardiac condition; and the method embraces many hygienic and dietetic considerations that assist in improving the nutrition of the cardiac tissue—one of the cardinal aims of a proper system of treatment. Anemia in one form or other plays an important rôle in the majority of the cases, and the particular variety present in each instance must determine the character of the remedies to be employed. In that large category of cases occurring in certain cachexias (cancerous, tuberculous) hematinics, arsenic and strychnin, are the remedies of choice.

A frequent, irregular pulse and other signs of cardiac failure indicate commencing dilatation, and under these circumstances digitalis should be employed in small doses. When used with perseverance it is of the greatest service, and in the form of the powder or the aqueous extract it may be conveniently combined with other cardiants.

I believe that gentle indulgence in physical exercise and light gymnastics is beneficial, since it tends to invigorate the heart-muscle; it is to be increased in proportion to the manifest improvement in the patient's condition. It sometimes happens, however, that even gentle exercise is badly borne, and it should then be discontinued. Kinesitherapy, particularly the milder Swedish method of gymnastic exercises (alternating movements of resistance), increases the contractile power of the heart and at the same time lessens the peripheral resistance, and should be accorded a careful trial. I have been in the habit of advising daily inhalations of oxygen gas in this class of cases with good results. Recourse to massage is also in the line of sound practice, but the sittings should not exceed half an hour in duration at the start. The more *prominent symptoms* may require special measures. The syncopal and anginal attacks are to be handled in the manner indicated for the same symptoms in chronic myocarditis. For the pseudo-apoplectic attacks rest in the recumbent posture, with the head slightly elevated, is useful. Therapeutic agents, as digitalis, ammonia, and ether, may be used hypodermically to stimulate the heart; it is also good practice to withdraw from 12 to 24 ounces (355.0–710.0) of blood directly from a vein. If the arteries be hard and tense, nitroglycerin is of distinct service.

A strictly horizontal posture and the application of ice to the precordial region often quickly terminate the attacks of cardiac asthma, and spartein sulphate, with nitroglycerin, is worthy of a trial. Hot toddy and other diffusible stimulants are valuable adjuvants. Should these remedies fail, hypodermic treatment by morphin is to be adopted.

FATTY OVERGROWTH.

Pathology.—The normal fat, particularly in the auriculo-ventricular furrows, is increased. I have elsewhere suggested the term "subpericardial over-fatness,"[1] to indicate the condition when unaccompanied by fatty infiltration. This over-production of fat may become so excessive as to form a complete enveloping mantle measuring an inch or more in thickness. In these extreme grades the muscular fibers may, from too great pressure, undergo atrophy and thus become weakened.

Etiology.—The principal cause is general corpulency. (For a consideration of the factors predisposing to fat production see OBESITY, p. 1229.) In the cachexias of carcinoma and phthisis, and the general atrophy of old age, fatty overgrowth and fatty degeneration coexist.

Symptoms.—The condition may be unaccompanied by any symptoms. The muscle-fiber is weakened (not degenerated, as a rule), hence extra labor suddenly thrown upon the organ excites the clinical indications of a weak (dilated) heart, as urgent dyspnea, vertigo, syncope, palpitation, and cyanosis. Later recurrences arise on every provocation. Distressing attacks of asthma may develop after a full meal or without an apparent exciting cause. A passive form of bronchitis may supervene. The cardiac impulse is feeble and may even be missing. The pulse is, as a rule, regular and moderately tense. Slight intermittence and, in marked heart-weakness, decided arrythmia may be noted. In moderate grades the heart-sounds may be clear; in marked cases with ensuing dilatation a systolic, apical murmur may be audible.

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[1] *Amer. Jour. Med. Sci.*, April, 1901.

The **diagnosis** rests upon the combined presence of marked obesity and cardiac enfeeblement. (For the **differential diagnosis,** see p. 658).

Treatment.—I wish to advocate warmly the system of treatment introduced by Oertel, as I have seen excellent results from its employment. It should not be resorted to in chronic valvular disease, in the stage of broken compensation, nor in marked atheroma.

Oertel's method comprises three parts: (1) The reduction of the amount of liquid taken with the meals and during the intervals, the total for each day being 36 ounces (1064.0). Frequent bathing (including the Turkish bath in suitable instances) and pilocarpin are employed to promote free diaphoresis.

(2) The diet is composed largely of proteids, as follows: *Morning.*—A cup of coffee or tea, with a little milk—about 6 ounces (178.0) altogether; bread, 3 ounces (93.0).

Noon.—Three to 4 ounces (90.0–120.0) of soup; 7 to 8 ounces (218.0–248.0) of roast beef, veal, game, or poultry, salad or a light vegetable, a little fish; 1 ounce (32.0) of bread or farinaceous pudding; 3 to 6 ounces (93.0–186.0) of fruit for dessert. No liquids at this meal, as a rule, but in hot weather 6 ounces (178.0) of light wine may be taken.

Afternoon.—Six ounces (178.0) of coffee or tea, with as much water. An ounce of bread as an indulgence.

Evening.—One or two soft-boiled eggs, 1 ounce (32.0) of bread, perhaps a small slice of cheese, salad, and fruit; 6 to 8 ounces (178.0–236.0) of wine, with 4 or 5 ounces (120.0–148.0) of water (Yeo).

(3) Graduated exercise up inclines of various grades. The distance to be undertaken each day is to be carefully specified and frequently, though gradually, increased. A like plan is to be pursued with reference to the degree of inclination. This is the most important part of the system, since it directly invigorates the heart-muscles.

Fatty Infiltration.—This condition may be associated with grave forms of myocardial degeneration, principally fibroid and fatty. In this place the term is limited in application to an infiltration or a dipping of fat between the muscle-fibers even to the endocardium, that is *secondary* to extreme obesity (*e. g.*, the anemic variety). It is clearly a rare condition, if we except the not uncommon instances in which the morbid process is limited to a thin layer of muscle-fibers situated directly beneath the epicardium. I have reported 5, and collected 7 additional cases from the literature.[1]

The *symptoms* may develop abruptly, after some unusual muscular exercise or after a profound systemic shock. More commonly, however, the clinical indications, which are not sharply defined as a rule, manifest themselves in a gradual manner. The principal features are *urgent dyspnea* (often an asthmatic form of breathing), and *utter exhaustion* upon muscular exercise, *precordial discomfort*, *pain under the sternum*, *cardiac palpitation*, *arrhythmia*, *syncope*, *vertigo*, *cyanosis*, and *angina pectoris*. Marked and constant disturbance of the cardiac rhythm is symptomatic of fatty infiltration. Hydrostatic *bronchitis*, with cough and expectoration, is commonly present. The angina pectoris may be dependent largely upon associated sclerosis of the arterial system. Emotional disturbance and mental apprehension were the chief nervous phenomena in my cases. The *physical signs* are neither constant nor characteristic; they are, in the main, those of cardiac dilatation. The *pulse* may be regular and of good tension, but after dilatation comes on it becomes irregular, frequent, and easily compressible. Moderate hypertrophy probably exists in the majority of cases, but cannot always be demonstrated owing to the extreme subpericardial over-fatness. A basic systolic murmur may be heard; it is not due to valvulitis as a rule. The *prognosis* as to cure is almost hopeless, although marked improvement may follow appropriate treatment. A fatal termination is often due to spontaneous rupture of the heart. The *treatment* must be directed especially to the over-fatness and the cardiac dilatation.

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[1] *Loc. cit.*

(*b*) **Brown Atrophy.**—A form of degeneration in which accumulations of yellowish-brown pigment-granules occur in the muscular fibers. The color exhibited by the heart-muscle is a reddish-brown, and in pronounced cases a dark-red brown. Brown atrophy is most commonly seen in the hearts of the aged, though also quite often in cases of chronic valvular disease that have reached an advanced stage.

(*c*) **Calcareous Degeneration** (*Calcification*).—Calcareous infiltration of the muscular fibers of the myocardium has been noted, though very rarely. Somewhat more common are the bony callosities that result from myocardial abscesses (*vide* Circumscribed Myocarditis).

(*d*) **Amyloid Degeneration.**—This form of degeneration is rare. It is limited to the blood-vessels and interstitial connective tissue; its causes are the same as those of amyloid degeneration of other viscera.

(*e*) **Hyaline Degeneration.**—This is sometimes seen in association with amyloid change. It also occurs independently in prolonged fevers (*hyaline transformation of Zenker*). The fibers are swollen, translucent, and homogeneous, and their striæ almost entirely disappear.

CARDIAC ANEURYSM.

(*Aneurysm of the Heart.*)

A CARDIAC aneurysm may involve either the whole diameter of the myocardium (aneurysm of the walls),[1] or merely the valves, together with a few myocardial fibers (valvular aneurysm).

Aneurysm of the Walls.—This is not of frequent occurrence. Its most common seat is the wall of the left ventricle near the apex; it is quite generally a sequel to chronic myocarditis, which occurs oftenest at this point. Anything that produces a decided localized weakness of the ventricular parietes (other forms of degeneration and endocardial and pericardial inflammations) may lead to its development. In size cardiac aneurysms are exceedingly variable, and may either be very small or as large as the average-sized head of an adult. As to form, two types should be recognized: (*a*) an equable dilatation of a part of the ventricular wall, and (*b*) the sacculated form, which communicates with the chamber by a comparatively small orifice. Layers of fibrin are often found in these aneurysmal dilatations—an indication of Nature's attempt at a cure, and occasionally she is successful. In most aneurysms non-laminated blood-clots are also found. It must not be forgotten that, once an aneurysmal distention has begun, a straining effort may cause a sudden great increase of its dimensions or even rupture it. The structures adjacent to the gradually formed aneurysm exhibit fibroid overgrowth—a conservative process.

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[1] Of 87 cases collected by Pelvet, 57 were in this situation, and of 90 collected by Legg, 59.

Diagnosis.—Aneurysm of the myocardium has no characteristic features. Usually the *symptoms* and *local signs* of chronic myocarditis or *dilatation* are more or less conspicuous, but the presence of the aneurysm is not even suspected unless certain physical signs develop in the course of the former complaints. These are—a *pulsating prominence* in the apex-region that may even perforate the chest-wall, and a coextensive dulness. The abnormal area of dulness is best appreciated early by stethoscopic percussion, but unless peculiarly circumscribed the condition cannot be distinguished from hypertrophy or dilatation. The *course* of these cases is unfavorable, death ensuing (rarely) from rupture of the sac or (more frequently) from gradual cardiac exhaustion.

Valvular aneurysms sometimes arise in acute ulcerative endocarditis, which destroys the segmented endocardium and permits of dilatation as the result of the intracardial blood-pressure. They occur with much greater frequency on the aortic than on the mitral valves. They are spheroid in shape, and project into the left ventricle when found at the aortic segments, and into the left auricle when at the mitral. Rupture of these aneurysms is common, with the subsequent development of valvular incompetency. They cannot be *diagnosticated* during life.

RUPTURE OF THE HEART.

THIS rare and serious accident may either be *complete* or *partial*. The term partial rupture implies laceration of the trabeculæ ventriculi, whereby the chordæ tendineæ are liberated. Rarely, the papillary muscles are torn. Valvular incompetency is the consequence of partial rupture. Complete rupture consists in a solution of continuity of the total diameter of the myocardium.

Pathology.—The chief seat of rupture is the anterior wall of the left ventricle, though it may also occur in the right ventricle and in the auricles. The rent runs parallel with the muscular fibers, and is to a certain extent the result of laceration, although chiefly of a separation, of the fibers. The fissural communication presents irregular edges, and at autopsy is seen to contain blood-clots; the pericardial sac is also occupied by coagula. If pericardial adhesions have previously obliterated the cavity, the escaped blood-clots may occupy the pleural cavity. Histologic examination of the adjacent muscle-structure shows the characteristic changes of fatty and other forms of degeneration.

Etiology.—Both *predisposing* and *exciting* causes may be at work.

The former are the more important and always obtain. and named in the order of their frequency of occurrence, the predisposing factors are disease of the coronary arteries (thrombotic and infectious embolic processes which produce anemic necrosis and abscesses), fatty degeneration,[1] chronic myocarditis, parietal tumors, and parasites in the heart-wall.

The influence of *age* is notable; rupture of the heart usually occurs after the sixtieth year has been passed, for the reason that the myocardiac changes that cause rupture belong to that period of life. Males suffer somewhat more frequently than females. The exciting cause is, as a rule, some form of muscular exertion, though it may occur during sleep.

Symptoms.—In the majority of instances rupture of the heart results in *sudden death.* Sometimes, however, the patient survives the accident for several hours or even for as many days. The symptoms are those of *internal bleeding*, in addition to *pain* that may be agonizing and is referred to the heart. The body-temperature falls, the skin surface becomes pale and cool, and it may be covered with cold perspiration, while the *pulse* grows small, very frequent, and finally almost vanishes. Occasionally gastro-intestinal symptoms and syncope tending to convulsions appear in consequence of the irritation of the vagus centers due to cerebral anemia. The *physical signs* of cardiac failure rapidly develop, and, if the leak be not too large, those of pericardial effusion more gradually.

Diagnosis.—Heart-anguish, rapidly progressive cardiac failure, the evidence of internal hemorrhage, and the speedy development of the signs of pericardial effusion should always excite suspicion of rupture, and in many cases suffice for a correct inference.

The **prognosis** is hopeless. When immediately fatal, death is the result of heart-shock; it may result from anemia of the brain or compression of the heart by the effused blood.

Treatment.—*Prophylaxis* is of the utmost importance. In all conditions of the cardiac parietes in which this accident is liable to occur the physician should not fail to give ample warning of the dangers connected with muscular strain of whatever sort. If rupture has either occurred or is suspected, the patient must be put at complete rest in the horizontal position. Full doses of morphin should be given hypodermically, and the ice-bag locally applied. Warmth to the extremities may be useful, but applied to the heart-region is probably harmful. The use of cardiac stimulants will be attended with increased bleeding from the rent, but agents that relax the peripheral arterioles, such as nitroglycerin, may be employed with a view to diminishing the heart's labor without diminishing its power. Should the rupture be partial and the hemorrhage slight, the patient's life may be prolonged, or even saved, by keeping him at absolute rest for a long period or until Nature effects recovery.

MINOR AFFECTIONS OF THE HEART.

(*a*) **New Growths.**—Primary carcinoma or sarcoma is rare indeed. Metastatic growths occur, but are very rarely sufficiently large (except perhaps the colloid variety) to be detected by *physical examination*, or to give rise to symptoms. Very large tumors may weaken the heart-muscle, but this must be an exceedingly rare occurrence. The separation of portions of the tumor may, if of considerable size, block one of the valvular orifices and cause sudden death, or more minute portions, becoming released, may give rise to embolism in distant parts. Tuberculosis and syphilis have been considered elsewhere.

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[1] According to Quain's statistics, about 75 per cent. of the cases are due to this cause.

(*b*) **Parasites.**—Four forms may invade the heart-muscle—the tænia echinococcus, actinomyces, cysticercus cellulosæ, and the pentastomum denticulatum. The former two are alone productive of mischievous results. The echinococcus growths may attain to considerable dimensions and are often multiple; they are secondary to echinococcus-cysts in other organs. Their effects are produced in a purely mechanical manner unless fragments become detached, when they may excite embolic lesions at different points in remote organs. Embolic abscesses have occasionally been observed, appearing like degenerations.

(*c*) **Misplacement** (*Transposition of the Heart*).—During intra-uterine life the heart (and rarely all the other thoracic and abdominal viscera) may either be transposed to the right side of the thorax, or the fetal position—in the median line—may be retained. The sternum may be missing in whole or in part, and the heart, which now lies immediately beneath the skin, can be seen and felt as a throbbing tumor. Recently a man of about forty years applied at the Medico-Chirurgical Hospital in whom the lower half of the sternum was absent; his heart occupied a position in the median line directly underneath the skin, where its strong pulsations could be felt. It had given the patient no inconvenience.

Very exceptionally other anomalous positions are acquired during ante-natal development, and the heart may become displaced upward in the chest-cavity even to the neck or downward into the abdominal cavity.

(*d*) **Floating Heart.**—The structures that serve to maintain the heart in its normal anatomic relations may become weakened or unduly lax, in consequence of which the organ may exhibit increased motility.

III. NEUROSES OF THE HEART.

PALPITATION.

Definition.—A more or less rapid action of the heart that is perceptible to the patient, and usually accompanied by an increased force of the cardiac contractions or a disturbance of the rhythm, and often also by precordial distress, anxiety, and dyspnea.

Etiology.—Chronic valve-disease and other organic affections of the heart seldom produce palpitation, numerous conditions outside of the organ being more frequently related causatively. Among these are—(1) Mental excitement, depression or emotion; (2) Anemia (from the local irritant action of the altered blood-state); (3) The acute infectious diseases, in which the toxins in the blood irritate the cardiac accelerating nerves; (4) Dyspepsia, even in robust-appearing persons (as in the gouty) who willingly or unwillingly commit dietetic errors. Special articles of diet may excite over-action (*e. g.* strawberries, shell-fish), the palpitation thus arising from reflex irritation being dependent upon gastric catarrh. (5) The use, and more especially the abuse, of tea, coffee, alcohol, and tobacco. These agents are injurious largely through their effects upon the nerves. (6) The female sex manifests a greater disposition to the complaint than the male, especially about the period of puberty and the menopause. In the male it is most common at or after the middle period of life, a time when the effects of the work and worry of life show themselves. (7) Disturbances of the ovaries and other pelvic organs may induce palpitation reflexly.

Symptomatology.—Cardiac over-action may, though rarely, be constant, but, as a rule, it displays a definitely *paroxysmal* character. The *onset* is sudden, and immediately preceding the attack there are often a blanching of the face and a slowing of the cardiac action, symptoms due to the momentary inhibitory effect of the nerve-affections that cause the "palpitation." The patient's *perception* of increased force and rapidity of the heart's action is the essential symptom. The patient may complain of *throbbing sensations* and *palpitation*, with a normally acting heart, the symptoms being wholly subjective in character. *Mental anxiety* is common, and dyspnea, the latter symptom assuming curious phases. In a recent case of my own the patient would attempt at intervals of three to five minutes a forcible, long-drawn inspiration, which would sometimes successfully relieve his respiratory difficulties for a while.

Physical Signs.—*Inspection* shows the impulse to be somewhat diffuse and forcible. Visible throbbing of the superficial vessels is also common. The *finger-tips* easily appreciate the increased strength of the impulse. At the wrist the pulse, though strong and full, as a rule is rapid, the rate varying from 120 to 160 per minute. *Percussion* may show the area of cardiac dulness to be enlarged, while *auscultation* reveals louder sounds than the normal. The attack is usually of brief duration—but a few minutes—though sometimes it may last for hours or days.

Attention should here be called to the *irritable heart* described by DaCosta—a form of palpitation common among young soldiers during the late Civil War. It was caused partly by mental excitement and partly by inordinate muscular exertion. A minor part in its production was also played by the diarrhea that was so often present. The leading symptoms were palpitation, a very frequent pulse, dyspnea, and cardiac pains of varying intensity.

Differential Diagnosis.—Nervous palpitation must be distinguished from the comparatively rare cases in which the heart contracts rapidly and irregularly, but does not excite subjective sensations. Some of the latter instances are to be looked upon as physiologic, while others are due to exhaustion and other causes. They do not constitute cases of palpitation, since they are unperceived by the patient, but are in reality cases of either tachycardia or arrhythmia.

Palpitation due to *chronic valve-disease* should also be differentiated from the purely nervous form. Here chief reliance is to be placed upon the presence of a murmur and other physical signs during the intervals between the attacks. *Anemic murmurs* are sometimes present, and must not be confounded with those of *organic nature*.

Prognosis.—The condition is free from real danger to life. Most authors, however, are agreed that cardiac hypertrophy may be a sequel.

Treatment.—The chief indications for treatment are—(1) *The arrest* *of the paroxysm.* The patient must be put at absolute rest in bed in a large, well-ventilated, darkened chamber, and his clothing loosened so that the respiration is unimpeded. Pressure upon the vagus in the neck or upon special points on the abdominal parietes (the ovarian region in particular) sometimes arrests the attack promptly. In my own hands the best results have been obtained from the application of the ice-bag to the precordial region. If this does not succeed in cutting short the paroxysm in the course of a couple of hours, the ice-bag should be removed every third hour. In conjunction with this measure the patient should be told to take large draughts of cold water or to swallow bits of ice. On the other hand, I have observed a few instances which were speedily relieved by the ingestion of hot and somewhat stimulating drinks. It is, however, not possible to formulate general rules that will be applicable to all cases. Kinnear[1] treats cardiac palpitation by applying cold over the sympathetic ganglia of the spinal cord.

Among the many therapeutic measures that have been employed, morphin alone has given quite constantly good results, and particularly when administered hypodermically. However, before employing morphin, other sedatives and narcotics should be tried, such as the bromids (in large doses), hyoscyamus, hyoscin, and camphor monobromate. In neurasthenic and hysteric subjects the bromids and the preparations of valerian are highly serviceable. The tincture of valerian or the elixir of valerian ammoniate may be used, and I have found the following capsule of great utility:

℞. Zinci valerianat.,	gr. x (0.648);
Strychninæ sulph.,	gr. $\frac{1}{3}$ (0.0216);
Ext. sumbul.,	gr. x (0.648);
Ext. hyoscyami,	gr. v (0.324);

M. et ft. capsulæ No. x.
Sig. One after meal-time.

If a special article of diet or an overloaded state of the stomach is the cause, an emetic may be given and the attack thus speedily controlled. Oxygen-inhalations have been warmly advocated.

(2) *To prevent a recurrence of the paroxysms,* the causal conditions, some of which may long antedate the occurrence of palpitation, must be removed, if this be possible. All exciting factors must also be avoided. The use of tea, coffee, and tobacco must be discontinued, and alcohol should be allowed only in small amounts. The general health must be considered, and anemia, chlorosis, neurasthenia, or hysteria must each receive appropriate treatment when present. When cardiac palpitation occurs in neurasthenia and hysteria the Weir Mitchell rest-cure should be advised, its results often being strikingly good if rigidly practised. Galvanism of the pneumogastric is sometimes useful, the positive pole being placed under the angle of the jaw, and the negative lower down, over each side of the neck. The removal of certain local conditions that sustain a causal relation, as gastric catarrh or intestinal parasitic diseases, is a question that must not be overlooked. If the heart be weak, digitalis may be exhibited for a long time in small dose. I have observed good effects from the use of baths (carbonated).

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[1] *Med. Rec.*, July 18, 1898.

TACHYCARDIA.

(*Tachycardia Paroxysmalis ; Synchopexia ; Rapid Heart.*)

Definition.—A rapid movement of the heart occurring in paroxysms of variable duration, and directly dependent upon either paralysis of the pneumogastric or stimulation of the sympathetic nerves. It is not dependent upon chronic valvular disease, nor upon other gross organic lesions, nor is it generally accompanied by notable subjective sensations. Martius believes that the condition is attributable to sudden dilatation.

Pathology and Etiology.—It occurs as a physiologic condition in a certain proportion of the human family ; in such cases the pulse may range from 90 to 100 beats per minute or over. Certain individuals can increase the pulse-rate by their own volition. The pathologic forms are divisible into—(1) Essential or neurotic tachycardia, and (2) Symptomatic tachycardia.

(1) **Neurotic Tachycardia.**—The causes of this variety are identical with many of those that excite palpitation ; hence these cardiac neuroses are often associated. Thus, among disposing factors are hysteria, anemia, neurasthenia, chlorosis, and toxic agencies (tea, coffee, tobacco, the poisons of fevers). Violent exercise, intense mental agitation, fright, grief, and other forms of shock are determining influences. Not a few cases are met at or about the menopause.

(2) **Symptomatic Tachycardia.**—The lesions that induce this form are —(*a*) *central* and (*b*) *peripheral.* In the former group are especially to be placed tumors, clots (due to hemorrhage), and softening of the medulla and cord ; and in the latter, tumors, aneurysms, enlarged lymph-glands (which paralyze the vagus by exerting pressure upon it either in the neck or thorax), and neuritis, affecting the pneumogastric nerve. The latter lesion may be associated with polyneuritis (alcoholic or infectious). Rapid heart may be due to *reflex irritation* (gastric, intestinal, arterial, uterine, ovarian), or gastro-intestinal intoxication.

Symptoms.—The clinical picture in most instances of the complaint is made up of recurring paroxysms of heart hurry (*paroxysmal tachycardia*). These attacks come on with great suddenness, and, as a rule, without prodromes or forebodings. If the latter occur, they consist of vertigo, tinnitus, and a sense of impending danger, and sometimes persist to the end of the attack. With the onset of the paroxysms the *cardiac movements* leap to 150, 175, 200, and 250, or even to 300 beats per minute. The *pulse* is feeble, small, readily compressible as a rule, and sometimes irregular (Fig. 53). Rarely it is full, strong, and of good tension. The *respiration* may or may not be increased in frequency, but dyspnea is not common. Respiratory oppression and smothering are seldom witnessed in genuine tachycardia.

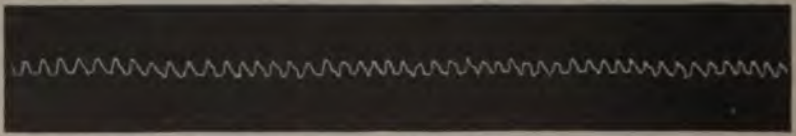

FIG. 53.—Radial pulse during an attack of paroxysmal tachycardia.

At first pale, the *skin* soon becomes flushed, and the *countenance* may wear an anxious expression; but unless "palpitation" is associated there are no symptoms present that denote an intense degree of suffering. In many cases the patient is not conscious of palpitation, or there may be a sense of slowing of the heart, when in reality the cardiac contractions may be increased to 200 or more; this is *typical tachycardia.* In a chlorotic girl I found that the pulse-rate increased to 200 beats, and lasted for a few minutes at each visit to my office. During the intervals between the visits the pulse was apparently normal in frequency. H. C. Wood reports a truly remarkable case occurring in a physician in his eighty-seventh year, who has had attacks at intervals since his thirty-seventh year. Following a sudden onset, the pulse rises quickly to 200 beats per minute. The attacks can be averted by the taking of ice-water or strong coffee.

Physical Signs.—A diffuse, rapid, and sometimes irregular impulse may be observed on *inspection* and *palpation*, but seldom is there an enlargement of the heart. The *sounds* are slightly modified, the first being accentuated and the second aortic greatly diminished in intensity, owing to the lessened amount of blood thrown into the aorta with each ventricular systole; the intensity of the second pulmonic, however, may be increased. An apical *systolic murmur* is occasionally audible. The carotids pulsate, and on auscultating over them a murmur is sometimes heard. The *duration* of paroxysmal tachycardia varies from one to two or more decades.

Diagnosis.—I would restate the fact that a high pulse-rate (200 or over a minute) and an absence or only a slight sense of palpitation or rapid heart-action are the distinctive features of true tachycardia. In *palpitation* (previously considered) the pulse-rate is not usually so high as in tachycardia, while the associated phenomena of dyspnea, precordial constrictions, smothering, and painful anxiety are correspondingly more pronounced.

Prognosis.—In the majority of cases no serious impairment of the general health follows, though the course is exceedingly chronic and recoveries are comparatively rare. When symptomatic tachycardia is due to lesions that are removable, it is often curable, though not invariably so. In sufferers who are advanced in years, however, the cerebral vessels may rupture. Boveret analyzed a number of cases, 4 of which proved suddenly fatal in consequence of heart-failure. In forecasting the issue during the paroxysm these serious accidents must be considered.

The **treatment** is to be conducted on precisely the same lines as those advanced for "Palpitation" (*vide* p. 669).

BRACHYCARDIA.

(*Bradycardia.*)

Definition.—Slowness of the pulse. The condition may be physiologic, the rate of the pulse being sometimes 60 or less, and very rarely as low as 40 per minute during perfect health.

All cases of pathologic brachycardia fall naturally and conveniently into two groups: (1) those that are secondary to other complaints (*symp-*

tomatic brachycardia); and (2) those that are due to, or associated with, a neurosis.

Pathology and Etiology.—**Symptomatic Brachycardia.**—(*a*) Arising during convalescence from acute infectious diseases, especially *pneumonia, typhoid, diphtheria, influenza*, and *acute rheumatism*. According to Riegel, who analyzed 1047 cases in which the pulse-rate was less than 60, the *acute fevers* must be awarded the first place among the causal factors. I have met with 3 cases of diphtheria in which the pulse in convalescence fell to 30 a minute. That such instances are, as Traube contends, due to exhaustion is true of some cases, but not of all, and doubtless there are other changes in a certain proportion. The slowing of the pulse that is observed after premature or full-time delivery is similarly produced. (*b*) The second place belongs easily to gastro-intestinal and hepatic disorders (*chronic gastro-intestinal catarrh, ulcer*, or *carcinoma of the stomach*). (*c*) Brachycardia occurs in diseases of the circulatory system—in *coronary disease*, fibroid and fatty myocardial change, most frequently; and chronic valvular disease much less frequently, if we except aortic stenosis. (*d*) Pulmonary complaints (emphysema and asthma). (*e*) Toxic agencies, as in jaundice, blood-poisoning, alcoholism, the unwonted use of tea, coffee, tobacco, and a few drugs (*e. g.* digitalis, strophanthus). (*f*) Constitutional affections (anemia, chlorosis, gout, diabetes). (*g*) Rarely skin-diseases and affections of the sexual organs, and commonly myxedema, are associated with brachycardia. In various organic nerve-affections (apoplexy, meningitis, epilepsy, tumors of the cerebrum, and the medulla in particular, injuries and diseases of the cervical portion of the cord). In such cases the brachycardia is due chiefly to direct or reflex irritation of the center or of the peripheral portion of the vagus system. Indeed, brachycardia is produced in one or other of these ways, except in those cases in which it is brought about by exhaustion of the automatic motor apparatus of the heart.

(2) **Brachycardia associated with a neurosis** may be found to be marked in *epilepsy*; less so in *hysteria, melancholia, mania*, and *general paresis* of the *insane*. It precedes palpitation.

Symptoms.—The sole characteristic symptom is the *slow action* of the heart, and this may either be temporary or permanent. If *paroxysmal*, both the onset and termination are apt to be sudden. A slow emergence is, however, more common than a slow beginning, though a small group of prodromes appears, comprising vertigo, tinnitus, and a sense of impending danger. During the paroxysm the patient may repeatedly suffer from *syncopal attacks* or become *unconscious* for hours at a time; *physical prostration* may also be marked, and especially when secondary to chronic valve-disease. The *pulse* is weak and small, and the beats per minute vary from 50, 40, 30, 20, to 10, or even 8. When the condition arises in the course of organic valve-lesions the *cardiac contractions*, as a general rule, may be increased in power, though greatly reduced in frequency. Thus, I observed this occurrence in a patient under my care at the Philadelphia Hospital suffering from a double mitral lesion and aortic constriction. The pulse fell from 70 to 28 per minute, but the systole was more powerful than before brachycardia was developed. The pulse at the wrist does not show the rate of cardiac contractions (when the heart is weak), since the latter do not always emit a pulse-wave that can be detected at the wrist; hence the heart-action must be noted by auscultation, and the rate compared with that of the peripheral pulse. The *impulse* and the *heart-sounds* are feeble.

Diagnosis.—A pulse below 48 beats per minute, with corresponding slowness of the systole, suffices for a certain diagnosis.

The **prognosis** is governed by the cause, being very grave in cerebral and advanced cardiac diseases. When fatal, sudden death is the rule.

Treatment.—Rest in the lying posture, particularly if the condition has come on in organic heart-disease, and such remedies as atropin, strychnin, caffein, nitroglycerin (in small doses), and ammonia are to be given a trial. If the ventricular contractions are very feeble and not below 30, small doses of digitalis will be found useful, though the effect must be closely watched. In the intervals between the attacks the general health must be improved and the causal states eradicated.

ARRHYTHMIA.

(*Irregular Heart- and Pulse-beat.*)

(1) THE irregularity may affect only the *volume* and *force* of the pulse. Here the intervals between the beats are equal, but in regard to fulness and strength the beats are unequal. Instances of irregularity in the volume and strength of heart-beats may give rise to the condition known as *pulsus alternans* (Traube), in which fuller and stronger pulse-beats regularly alternate with those of lesser volume and strength (see Fig. 54). (2) **Irregularity in Time.**—(*a*) Intermittent heart-beat. This is but an exaggerated degree of the first variety, and signifies a missed or dropped beat. This occurs at irregular intervals in most of the cases, though sometimes a cyclical irregularity is observed—*i. e.* every second, fourth, sixth, or eighth beat being lost. (*b*) Twin-pulse (*coupled beats, allorrhythmia*). When two beats follow each other quickly (the diastole being shortened), and the next two not so quickly (the diastole being lengthened), we have produced the *pulsus bigeminus*. The first and second beats may be of equal strength, but often the second is relatively feeble. This is best determined by auscultation of the heart, since the second systolic contraction (of the ventricle) may indeed be so weak as not to give rise to a palpable beat at the wrist. I have fre-

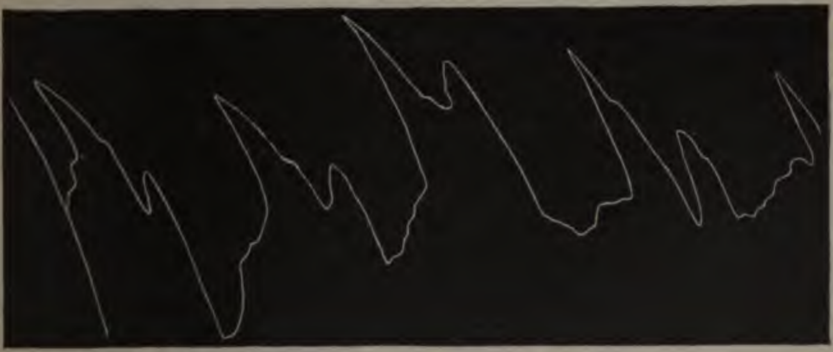

FIG. 54.—Pulsus bigeminus alternans (Eichhorst).

quently observed the *pulsus bigeminus* in mitral disease. With respect to the diastole, the approximated pulsations may be in blocks of three (*pulsus trigeminus*), or even of four (*pulsus quadrigeminus*). (3) **Combined irregularity** of time and volume. Whilst the forms of irregularity described above should be distinguished from one another, this is not always practicable, particularly in the last stages of valvular affections and in the acute infectious diseases—conditions in which the heart-muscle fails in consequence of degenerative changes. (4) The **paradoxical pulse** of Kussmaul also consists in irregularity of volume, strength, and time, though not indicative of so great peril as the preceding. It is dependent upon the act of inspiration—"normal as well as forced"—the beats during inspiration being more rapid, though weaker, than during expiration. This is met with in chronic adhesive pericarditis, in cases of pressure upon the root of the aorta by bands, in pleuro-pericarditis, and in a very weak heart. (5) **Delirium cordis** is a term very appropriately given to great irregularity and inequality of the pulse-beats. It is seen in extreme dilatation and advanced exophthalmic goiter. (6) **Embrocardia or Fetal Heart-rhythm.**—There is a shortening of the long pause with a striking similarity of the first and second sounds, as in the fetal heart. I have already pointed this out in connection with dilatation, though it also sometimes attends the advanced stages of grave fevers. (7) **Cantering Rhythm** (*bruit de galop*).—The sounds simulate the triple footfall of a horse at a canter. The interpolated sound is due to a reduplication of the second, though rarely it is the first that is doubled instead. It is developed in the hypertrophy of arterio-sclerosis and Bright's disease, in profound anemias, and in the myocarditis of certain acute infectious diseases. (8) **Tremor Cordis.**—By this is meant a ventricular systole so rapid as to be evidenced by mere vibrations.

Etiology.—Baumgarten's classification of the causes of arrhythmia (quoted by Osler) is the best, and is here given:

(1) Those due to central—cerebral—causes, either organic disease, as in hemorrhage or concussion, or more commonly psychical influences.

(2) Reflex influences, such as produce the cardiac irregularity in dyspepsia and diseases of the liver, lungs, and kidneys.

(3) Toxic influences. Tobacco, coffee, and tea are common causes of arrhythmia. Various drugs, as digitalis, belladonna, and aconite, may also induce it.

(4) Changes in the heart itself. (*a*) In the cardiac ganglia. Fatty, pigmentary, and sclerotic changes have been described in cases of this sort, and these may have an important influence in producing disturbances in the rhythm, but as yet we do not know their exact significance. They may be present in cases that have not presented arrhythmia. (*b*) Mural changes are common in conditions of this kind. Simple dilatation, fatty degeneration, and sclerosis are most commonly present, the two latter being usually associated with sclerosis of the coronary arteries.[1]

Symptoms.—Arrhythmia, particularly when functional or of reflex origin, may exist for years together, without associated symptoms referable to the heart, and hence is often discovered accidentally. When it is combined with palpitation or extreme weakness or dilatation of the organ, it is apt to arrest not only the attention of the observer in many instances, but also that of the patient.

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[1] *Transactions of the Association of American Physicians*, vol. iii.

Physical Signs.—In given cases the cause will be found to govern the character of the physical signs, which are often scanty or sometimes practically wanting. Those usually present have been indicated in speaking of the different varieties.

Diagnosis.—*Palpation* and *auscultation* of the heart while examining the pulse are matters that should never be neglected if reliable results are to be obtained. It is especially in this class of cases that the sphygmograph renders invaluable aid. Sphygmograms will often show the kind and degree of arrhythmia when all other means of examination have failed, and also distinguish marked dicrotism from irregularity.

It is important to *differentiate* functional arrhythmia or that of reflex origin from arrhythmia due to more or less grave myocardial disease. Important information is supplied by carefully reviewing the varied etiologic factors that produce the functional form, and by close observation of the cardiac symptoms.

The **prognosis** is variable. A gentleman with whom I am acquainted was rejected by a life-insurance company twenty years ago on account of occasional slight arrhythmia, though he is still in active business life and apparently in vigorous health. When the myocardium becomes involved, as occurs in chronic valvular or coronary disease or in the acute infectious diseases, the prospect is gloomy; on the other hand, when it is functional or due to other causes outside of the heart itself, the course pursued is as a rule favorable. When the second sound follows closely the first (marked abbreviation of the systolic pause) it is a serious indication.

Treatment.—There are many cases of the more benign form in which no treatment is required apart from methodic, physical training to improve the strength of the heart-muscle and the general systemic development. Removal of the causal forces, as tea, coffee, alcohol, indigestible food-stuffs, conditions acting in a reflex manner, must be executed promptly. When the condition is due to changes in the heart-structures, cardiants in addition to the general tonics should be prescribed. I prefer strychnin, arsenic, and the dried sulphate of iron in combination. Nitroglycerin is of service if the arterial tension be high. If the arrhythmia be due to excessive cardiac dilatation, digitalis should be employed. In purely functional cases, in which there is a predominating neurotic element, the subjoined formula has been useful in my **hands**:

℞. Ferri valerianatis,
Zinci valerianatis, *āā*. gr. xxx (1.94);
Strych. sulph., gr. j (0.0648);
Pulv. digitalis, gr. viij (0.518).
Ft. capsulæ No. xxx.
Sig. Take one after meal-time.

ANGINA PECTORIS.

(*Stenocardia, Breast-pang.*)

Definition.—A paroxysm of violent precordial pain extending into the neck, back, and arms, and at times attended by a sense of impending death. It scarcely deserves to be classified as a separate disease, being merely symptomatic of several cardiac or aortic (?) lesions.

Pathology.—Concerning the pathogenesis of angina many theories have been advanced, but conclusive *post-mortem* evidence in support of each and all is wanted. Neither does any single hypothesis fit the grouping of symptoms so constantly observed in cases of angina. It is to be recollected, however, that it is a neurosis affecting the cardiac sensory filaments that are given off chiefly from the pneumogastric, and in many cases the vasomotor apparatus is also involved. Allbutt[1] believes that the symptoms owe their origin to disease in the aorta—acute and chronic aortitis. Nothnägel has described a form distinct from the above in which the vasomotor apparatus is principally involved (*angina pectoris vasomotoria*).

Etiology.—Cases of angina unassociated with *arterial sclerosis*, *hypertrophy*, *aortic regurgitation*, or adherent pericardium, are rarely encountered. It is quite probable that with few exceptions sclerosis of the aorta and coronaries is present, and predisposes to the disease under discussion. This view also receives some degree of color from the fact that angina usually occurs after the *fortieth year*, and principally in the *male* sex. It may be a sequel of influenza. The overuse of tobacco may rarely cause true anginiform symptoms. The *determining factors* of the attack are undue exertion and mental emotion.

Symptoms.—The *paroxysm* begins quite suddenly during the action of one or other exciting cause. There is excruciating pain of a griplike character, affecting the entire chest and rendering the body motionless. The pain *radiates* most frequently to the left shoulder, though also at times to the right, and thence to the back, neck, and down the arms to the fingers. *Coldness* and *numbness* of the fingers or in the precordial area may be present. Not less agonizing than the pain is the awful sense of *impending death*. The *countenance* is frequently pale, and may assume a leaden hue, and is usually bathed in cold perspiration. The *respirations* are exceedingly shallow or even temporarily arrested, and the patient's anxiety is extreme. The *heart's action* may be regular, and the arterial tension, as shown by the pulse, is generally increased. The *duration* of the paroxysm varies from a few seconds to a minute or two, and after the pain is over gaseous eructations, vomiting, or the discharge of a large amount of clear urine may occur. With the cessation of the attack comes instant relief from the cardiac symptoms. On making a careful examination of the heart subsequent to the seizure there may be an utter *absence of signs*, and, though there is weakness, this soon disappears. The attacks may *recur* at intervals varying from a few days to many years. I have usually found that cases associated with aortic regurgitation give the shortest intervening periods as a rule.

In *angina vasomotoria* the pain in the heart-region is preceded for a few minutes by pallor of the face, coldness and stiffness of the limbs, due to spasm of the peripheral vessels.

Pseudo-angina.—This is also a *paroxysmal* affection, that may be associated with hysteria, anemia, or neuropathic inheritance, and is occasionally produced by infectious diseases and poisons (see p. 676).

Diagnosis.—The characteristic events are a sudden, most intense pain in the substernal and left parasternal regions with marked constriction of the chest, the peculiar manner of radiation of the pain, and the fear of death. Less diagnostic, though of considerable value, are the brevity of the attack, the sudden onset and cessation of the pain, the age and sex, and the anxious, moistened features. There are also lighter forms, in which one or more of the diagnostic phenomena above described are absent. If they occur between the ages of forty and sixty years in persons in whom either arterial sclerosis or aortic regurgitation is present, this disease should be thought of; and after the exclusion of certain complaints in which paroxysmal pain is prominent, such as gastralgia, intercostal neuralgia, and locomotor ataxia, the diagnosis of angina becomes reasonably certain.

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[1] *Philadelphia Medical Journal*, June 30, 1900.

The distinction between true and pseudo-angina pectoris rarely presents formidable difficulties (see table below):

ANGINA PECTORIS.	PSEUDO-ANGINA.
Etiology indeterminate, though generally associated with arterio-sclerosis (including coronary disease) or aortic regurgitation.	The causes are—hysteria, neurasthenia, toxic agents, and reflex irritations.
Occurs after the fortieth year, usually in males.	Occurs at any age (over six years), and usually in females.
Paroxysms, provoked by undue exertion or mental emotion, are rarely periodic and nocturnal.	Paroxysms arise spontaneously, are periodic and often nocturnal.
Pain intensely severe, and constricting, its chief center being to the back of the mid-sternum and toward the left.	Less intense pain, more diffused over the thoracic region: sensation of cardiac distention.
Duration of attack from a few seconds to one or two minutes.	From a half to several hours.
Patient silent and body fixed.	Restlessness and emotional symptoms of causative conditions associated.
Arterial tension increased as a rule.	Usually not increased.
Prognosis unfavorable.	Entirely favorable.

The vasomotor form of angina must not be confounded with pseudo-angina, which is infinitely more common.

The **prognosis** is bad, yet uncertain. I recall two instances that occurred ten and twelve years ago respectively: the first attacks were survived with no recurrence. When the arteries are sclerosed (particularly the coronaries) life is often suddenly terminated during the course of the attack. Occasionally the sufferer dies of syncope. The nature of the causal and associated lesions must be considered in estimating the prospect of life. In the vasomotor angina of Nothnägel the outlook is less grave, while in pseudo-anginal attacks it is bright.

Treatment.—*Prevention* of the attacks in persons who are subject to them is of the most importance. In order to do this all known exciting factors are to be rigidly avoided. The patient should be instructed to carry constantly in a convenient pocket such agents as nitroglycerin and amyl nitrite, beads or *perles* (strength 3 to 5 drops), and also how to use them with judicious care immediately upon the first indication of an approaching paroxysm.

The *treatment of the attack* must be prompt and energetic, though carefully conducted, amyl nitrite being inhaled at once from a handkerchief in doses of 3 to 5 drops according to the severity of the attack. The patient should then be placed in a cool apartment free from disturbing [illegible]. *Locally*, the use of the ice-bag may prove efficacious and should be tried at first. Rarely, hot applications (hot cloths or sinapisms) give better results than cold. If the pain is not controlled promptly by this method, the nitrite should be reinforced by the hypodermic injection of morphin (gr. $\frac{1}{3}$—0.0216) combined with atropin (gr. $\frac{1}{120}$—0.0005). This usually brings speedy relief, and is best suited to those instances in which there is no increase of arterial tension. In cases exhibiting high arterial tension the tincture of nitroglycerin, hypodermically, should be employed (dose ♏j—0.066—to be repeated once in a minute if needful).

During the *intervals between the attacks* the aim should be not only to obviate the action of the exciting causes, but also to overcome any predisposing influences that may exist. Schott[1] prefers baths (effervescent) and passive movements to drugs or other methods. In true angina, gymnastic exercises, in the form of passive movements alone, should first be performed by an assistant, but later may be safely entrusted to the patient. In cases in which the arterial tension is habitually exalted, nitroglycerin in increasing doses is to be used perseveringly, beginning with ♏j (0.066) and increasing by ♏j (0.066) every five or six days until the physiologic effects are produced. Sodium nitrite may be employed similarly, the dose being gr. j–iij (0.0648–0.194) three or four times daily. Marked arterial sclerosis, particularly if there be a syphilitic history, is favorably influenced by a long course of potassium iodid. It may be prescribed in doses ranging from gr. v to xx (0.324–1.296) three times a day. When hypertrophy of the left ventricle is excessive, the use of the following is effective:

℞. Tr. aconiti rad. ♏xlviij (3.10);
Sodii bromidi ℥ss (16.0);
Elix. simplicis q. s. ad ℥iij (96.0).—M.
Sig. ʒ j t. i. d.

It may be omitted at the end of every two weeks for two or three days. The presence of a gouty diathesis would call for special treatment.

In the vaso-motor form amyl nitrite and nitroglycerin are most valuable. Occasionally, hot foot-baths, followed by friction of the extremities, are also of the greatest utility.

The treatment of pseudo-angina must be directed at the cause of the complaint—the neurotic condition.

IV. CONGENITAL AFFECTIONS OF THE HEART.

These result from two leading causes: (1) Arrested development, and (2) fetal endocarditis. Occasionally, both these factors are operative.

Arrested development may produce a great variety of anomalies, [illegible] may be briefly enumerated: (*a*) Acardia, absence of the [illegible], or *reptilian heart*, in which the septum between the auricles and ventricles is absent, thus reducing the number of cavities to two. This is an instance of reversion to a lower type. (*c*)

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[1] Med. Rec., March 11, 1899.

Absence of the interventricular septum, the heart consisting of three chambers (*cor triloculare*). More frequently there is a mere perforation in or an incomplete development of the septum, and this is usually situated in the upper portion. Obstruction of the pulmonary orifice or of the conus arteriosus of the right ventricle are frequently conjoined conditions. (*d*) *Patency, or incomplete closure of the foramen ovale*. Persistence of the foramen is, in the majority of cases, associated with obstruction of the pulmonary valve, though it may be solitary. (*e*) An anomaly known as *ectopia cordis* deserves mention. This is a condition in which the sternum is usually divided vertically, and the heart is either entirely exposed or beating just beneath the skin in the cardiac, thoracic, or abdominal region. In this connection another and the most common form of malposition may be added—namely, *dextrocardia*. Here the heart occupies the right side, with reversion of the arch and displacement of the descending aorta to the right of the spinal column. Transposition of other viscera is usually associated. (*f*) *Anomalies of the valves*.—There may be either a numerical increase or decrease of the cardiac valves, particularly the segments of the semilunar valves of the aortic and pulmonary orifices. Supernumerary segments are usually rudimentary, and at the pulmonary valve one, two, or more are most commonly seen. A decrease in the number of segments is also most frequently observed at the arterial orifices, the aortic and pulmonic semilunar valves then being composed of two segments (bicuspid). This condition may be due to defective development on the one hand, or to endocarditis with resulting cohesion of segments on the other.

(2) **Fetal endocarditis** leads to valvular deficiencies in a manner similar to what occurs during the whole post-natal period. The valve-lesions originating during fetal life are most frequently situated on the right side, probably for the reasons that the ante-natal circulation is more actively carried on in the right than in the left heart, and that the former receives the oxygenated blood from the placenta. They may occur at the pulmonic, the aortic, or the auriculo-ventricular orifices. The changes are of the slow sclerotic form as a rule, and their character is determined largely by the antecedent anomalies that predispose to them. The leaflets present smooth, thickened, and contracted borders. Union of the mitral segments is common, and the chordæ tendineæ are often thickened and contracted. The small rounded bodies that are normally present on the mitral and tricuspid segments (*nodules of Albini*) must not be confounded with pathologic verrucosities.

The most frequent congenital valvular lesion is **stenosis of the pulmonary orifice** as the result of chronic endocarditis. Rarely, it is due directly to defective development, and perhaps more rarely still to endocarditis verrucosa. **Pulmonic constriction** of ante-natal origin may be an associated lesion in other forms of valvular disease in the young adult. With stenosis at the pulmonary orifice, there usually coexist stenosis of the conus arteriosus of the right ventricle, an open foramen ovale, and a patent ductus arteriosus; according to Peacock, "in 86 per cent. of the patients with congenital heart-disease living beyond the twelfth year the lesion is at this orifice." **Atresia of the pulmonary orifice** occurs, though less frequently than stenosis.

At the **tricuspid orifice** there may be stenosis or contraction of the valves, producing either obstruction or regurgitation. Similar lesions of the aortic orifice are infrequent. **Congenital mitral disease** also occurs, but only exceptionally; it is then usually associated with tricuspid stenosis. Boys are somewhat more liable to congenital affections of the heart than girls.

Symptoms.—There is an almost constant and strikingly distinctive symptom in congenital heart-disease—*cyanosis*. The *tint* of skin observed is variable, being at one time a general duskiness, at another a deep violet, and rarely almost black. This coloration is most noticeable about the lips and mucous membrane of the mouth, the nostrils, conjunctivæ, the fingers, toes, and lobules of the ears, and as a rule is general, though it may be a local condition. The tint may grow less distinct, and even almost vanish, when the child is in perfect repose or sleeping; excitants or efforts at coughing, however, increase the intensity of the discoloration. The cyanotic hue comes on almost invariably during the first week of life. The *fingers* present a decidedly clubbed appearance, and the *nails* are thickened and curved like the claws of certain animals. The *temperature* is subnormal, while the extremities are cool to the feel. *Dyspnea* on exertion and *cough* are usual concomitants. Cyanosis may be due to various causes that may act either singly or more often concurrently. Variot reports two cases with similar cardiac conditions—namely, interventricular perforation, and narrowing of the pulmonary artery, with the aorta arising from both ventricles. Cyanosis was absent from one case, and "this disproves the two leading theories with regard to the origin of cyanosis—the mixture of the two bloods and the obstruction to the pulmonary circulation."

Physical Signs.—In the very young the impulse is feeble (with an absence of a palpable thrill), the *percussion-dulness* is increased, especially to the right, and a loud *systolic murmur* is audible at the pulmonary orifice. When the auriculo-ventricular valves are the seat of endocarditis, the murmur may be apical. In pure pulmonary stenosis the second sound is feeble.

In older children the area of *dulness* is only slightly extended, particularly to the left, while the *murmurs* heard are loud and often musical.

It is interesting to note that in rare instances *cerebral abscess* is an associated condition.

Differential Diagnosis.—The distinction between congenital and acquired lesions in children may be assisted by a reference to certain points tabulated below:

CONGENITAL LESIONS.	ACQUIRED LESIONS.
History of almost constant cyanosis, beginning in the first week after birth.	Not so; history of endocarditis or of rheumatism or other complaints in which endocarditis occurs as a complication.
Slight enlargement of the heart. It is of the right ventricle, chiefly non-progressive.	Enlargement marked, frequently involving the left ventricle, and progressive.
Loud and musical murmurs present, audible over upper third of sternum, with small area of transmission upward and to the left; second sound weak.	Audible over apex or base; definite large areas of transmission. Second sound frequently accentuated.
Deficient bodily development.	Bodily development good, as a rule.
Mental faculties in abeyance.	Mental faculties normal.

Prognosis.—The prognosis is exceedingly grave. Many succumb within a few days after birth, more than one-half before the expiration of one year, and not less than three-fourths before the end of the third year. Few survive the first decade of life, and fewer still reach full adolescence. Among the forms giving the most favorable prognosis are pulmonary stenosis, especially when of moderate grade, and defective auricular and ventricular septa. In those instances in which life is spared in the first weeks after birth there is a disposition to affections of the lungs (phthisis, hemoptysis), nerve-complaints (convulsions, cerebral hemorrhages).

Treatment.—The treatment is, in the main, hygienic. The body must be warmly clad, flannels being worn next the skin, and every source of cold should be carefully guarded against. The diet is to be judiciously arranged, yet liberal, preference being given to the carbohydrates. Gentle exercise when it can be taken is valuable, as are also daily spongings of the surface followed by friction. Special therapeutic indications may arise, and must be met in accordance with general principles, while tonics, such as iron, arsenic, quinin, and cod-liver oil, are frequently applicable.

V. DISEASES OF THE ARTERIES.

ACUTE AORTITIS.

Pathology.—The morbid changes coincide with those noted in acute endocarditis, including the ulcerative variety.

Etiology.—The causes are not clear, but the condition generally follows the acute infectious diseases (typhoid fever, pneumonia, miliary tuberculosis). Alcoholism and syphilis are among the rarer causes. Various microörganisms have been discovered to be causal irritants. Boinet and Romary have recently shown that in experimentally produced aortitis a point of lessened resistance (either from traumatism or other previous arterial lesion) is necessary.

Symptoms.—The symptoms are *local* and *general.* Of the former, diffuse thoracic *pain*, with substernal *tenderness* under pressure and cardiac *palpitation*, are the chief. The pain may assume the type of true angina pectoris. Among the *general* symptoms a moderate febrile movement is almost constant. In a certain percentage of cases embolism is betrayed by the usual signs, as rigors, accompanied by a steep temperature-curve. These forms are analogous to the malignant variety of endocarditis. A cardiac murmur may be heard over the base.

Diagnosis.—All that the best clinicians can do is to establish a probable diagnosis even in the presence of the most frankly expressed features of the affection. From *acute endocarditis*, aortitis is to be discriminated by its diffuse pain and by the higher seat of its murmur.

The **prognosis** is serious, owing to the liability to infectious emboli and aneurysmal dilatation and the possibility of aortic rupture.

The **treatment** is similar to that of acute endocarditis.

ARTERIAL SCLEROSIS.

(*Arterio-sclerosis; Arterio-capillary Fibrosis; Endarteritis Chronica Deformans; Atheroma.*)

Definition.—An overgrowth of the connective tissue of the arterial coats (chiefly and primarily of the intima), followed by calcareous depositions. The elastic tissue of the intima is primarily increased.

Pathology.—The most frequent seat of the sclerotic process is the aorta, and the next most common the coronary arteries. Other vessels implicated are the arteries of the brain, the temporals, radials, brachials, ulnars, femorals, and iliacs. On the other hand, certain arteries, as the gastric, hepatic, and mesenteric, are rarely affected. Two forms may be recognized: (*a*) the circumscribed, and (*b*) the diffuse.

(*a*) **Circumscribed Arterio-sclerosis.**—Naturally, the intima presents a smooth internal surface, but when atheromatous changes occur it shows localized areas of thickening. These patchy prominences are often hemispheric in outline, yellowish-white in color, and their favorite seats are the orifices of the branches. They increase in depth and superficial area, and on reaching an advanced stage their interior disintegrates into granular material (*atheromatous abscess*).

In circumscribed or nodular atheroma the microscope discloses the fact that the middle and external coats are the primary seat of the changes, which consist of localized infiltrations. These lesions weaken the media and adventitia, and then (as shown by Thoma) compensatory processes are set up in the intima which lead to the formation of the so-called *atheromatous button.* The latter consists in a hyperplasia of the intima with a deposit of round cells, which causes a gradual increase in thickness; in this way the growing weakness of the middle and outer coats is compensated. When the prominences in the intima undergo softening or liquefaction, rapid dilatation (*aneurysmal*) of the affected vessels is apt to occur. The same accident may arise early or before the intima has reinforced the other layers by its increased thickness and strength.

(*b*) **Diffuse Arterio-sclerosis.**—In this form the morbid process is distributed throughout the greater part of the arterial system, the nodular or circumscribed form being usually combined with it in the aorta. Dilatation of the aorta and of its branches commonly coexist. Apart from the yellowish, translucent, elevated areas, the intima may be smooth and the naked-eye appearances almost normal. The coats, however, and particularly the intima, are very much thickened. Microscopic examination brings to light an extensive proliferation of the subendothelial connective tissue and a hyaline transformation of the entire media, particularly in the larger vessels. The muscular fibers and elastic tissue have *in advanced cases* almost totally disappeared. Necrotic degeneration of the media, especially in the smaller arteries, is also observed, and calcareous deposits, causing rigidity of the walls, occur among the later changes. This is particularly true of the so-called *senile arterio-sclerosis.* Atheromatous abscesses that burst, forming atheromatous ulcers, are likewise common pathologic events in the aged. There may be associated atrophy of the heart, liver, and kidneys, due to a lack of nutritive supply in consequence of the narrowing of the vessels. More commonly, however, in this, and constantly in circumscribed and diffused sclerosis, the heart is moderately hypertrophied. When coronary disease is present, fibrous myocarditis and sclerosis of the aortic cusps may be found associated. The kidneys may also become sclerotic—a condition that will be described hereafter (*vide* Diseases of the Kidneys).

Sclerosis of the pulmonary artery, previously referred to in the discussion of the Diseases of the Heart, exhibits all the changes observed in connection with atheroma of the systemic arteries, including aneurysmal dilatation of the trunk and rarely of the main branches. From the terminal tributaries the process may extend to the capillaries, and even to the pulmonary veins (*angio-sclerosis*).

The effect of arterio-sclerosis upon the physiologic functions of the vessel-walls, and the pathologic and clinical results are of the utmost importance. The elastic coat is destroyed, and hence the walls cannot bear the blood-pressure as well as in health. This predisposes to dilatation of the vessels (aneurysm).

Another result of extensive atheromatous degeneration of the vessels is an increase in the resistance to the blood-current, and a consequent elevation of the arterial pressure. Furthermore, the loss of elasticity in the coats of the medium-sized and smaller arteries removes an important factor in the propulsion of the blood. The left ventricle, in consequence of this fact, almost invariably becomes hypertrophied in cases of extensive arterio-sclerosis, provided the general nutrition of the patient is still well maintained (Strümpell).

The reduction of the lumen of the vessel, owing to the thickening of the intima, must lessen the blood-supply to the various viscera, and thus in part are explained such secondary affections as fibrous myocarditis, cirrhosis of the kidneys, and cerebral softening.

Sclerosis of the veins (*phlebo-sclerosis*) may rarely accompany arterio-sclerosis. It is, however, not infrequently found in association with hepatic cirrhosis and mitral disease when the portal system and pulmonary veins are involved. It is occasioned by increased intravenous pressure. Arterio-sclerosis apart from sclerosis of the peripheral veins may be encountered, though rarely.

Microscopically, thickening of the intima and atrophic degenerative changes in the media are commonly observed. Calcification and hyaline degeneration of the layers also occur, and I have observed them in one of my own cases. Moderate dilatation is not exceptional.

Etiology.—The diffuse form has, in part, a special etiology. It may appear in the young, though rarely; I have met with a case in the Medico-Chirurgical Hospital in a man aged twenty-four years. It is, however, most frequent in the middle-aged (who are able-bodied) and in the aged. At an earlier period it occurs as a result of *alcoholism*, *syphilis* (the overshadowing factor), *lead-poisoning*, *gout*, and *chronic nephritis*—agencies that subject the vascular system to undue wear and tear. In old persons atheroma is often *physiologic* and characterizes the natural involution-period of life. *Heredity* may play no inconspicuous part in arterio-sclerosis dependent upon the age. This fact furnishes, to some extent at least, the reason why senile changes in the arteries occur at a much earlier period of life in some instances, and even throughout whole families, than in others. *Negroes* are more liable than *whites* to this form of atheroma, and *males* than *females*, though it is more frequent in the latter sex than the circumscribed variety.

quently observed the *pulsus bigeminus* in mitral disease. With respect to the diastole, the approximated pulsations may be in blocks of three (*pulsus trigeminus*), or even of four (*pulsus quadrigeminus*). (3) **Combined irregularity** of time and volume. Whilst the forms of irregularity described above should be distinguished from one another, this is not always practicable, particularly in the last stages of valvular affections and in the acute infectious diseases—conditions in which the heart-muscle fails in consequence of degenerative changes. (4) The **paradoxical pulse** of Kussmaul also consists in irregularity of volume, strength, and time, though not indicative of so great peril as the preceding. It is dependent upon the act of inspiration—"normal as well as forced"—the beats during inspiration being more rapid, though weaker, than during expiration. This is met with in chronic adhesive pericarditis, in cases of pressure upon the root of the aorta by bands, in pleuro-pericarditis, and in a very weak heart. (5) **Delirium cordis** is a term very appropriately given to great irregularity and inequality of the pulse-beats. It is seen in extreme dilatation and advanced exophthalmic goiter. (6) **Embrocardia or Fetal Heart-rhythm.**—There is a shortening of the long pause with a striking similarity of the first and second sounds, as in the fetal heart. I have already pointed this out in connection with dilatation, though it also sometimes attends the advanced stages of grave fevers. (7) **Cantering Rhythm** (*bruit de galop*).—The sounds simulate the triple footfall of a horse at a canter. The interpolated sound is due to a reduplication of the second, though rarely it is the first that is doubled instead. It is developed in the hypertrophy of arterio-sclerosis and Bright's disease, in profound anemias, and in the myocarditis of certain acute infectious diseases. (8) **Tremor Cordis.**—By this is meant a ventricular systole so rapid as to be evidenced by mere vibrations.

Etiology.—Baumgarten's classification of the causes of arrhythmia (quoted by Osler) is the best, and is here given:

(1) Those due to central—cerebral—causes, either organic disease, as in hemorrhage or concussion, or more commonly psychical influences.

(2) Reflex influences, such as produce the cardiac irregularity in dyspepsia and diseases of the liver, lungs, and kidneys.

(3) Toxic influences. Tobacco, coffee, and tea are common causes of arrhythmia. Various drugs, as digitalis, belladonna, and aconite, may also induce it.

(4) Changes in the heart itself. (*a*) In the cardiac ganglia. Fatty, pigmentary, and sclerotic changes have been described in cases of this sort, and these may have an important influence in producing disturbances in the rhythm, but as yet we do not know their exact significance. They may be present in cases that have not presented arrhythmia. (*b*) Mural changes are common in conditions of this kind. Simple dilatation, fatty degeneration, and sclerosis are most commonly present, the two latter being usually associated with sclerosis of the coronary arteries.[1]

Symptoms.—Arrhythmia, particularly when functional or of reflex origin, may exist for years together, without associated symptoms referable to the heart, and hence is often discovered accidentally. When it is combined with palpitation or extreme weakness or dilatation of the organ, it is apt to arrest not only the attention of the observer in many instances, but also that of the patient.

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[1] *Transactions of the Association of American Physicians*, vol. iii.

Physical Signs.—In given cases the cause will be found to govern the character of the physical signs, which are often scanty or sometimes practically wanting. Those usually present have been indicated in speaking of the different varieties.

Diagnosis.—*Palpation* and *auscultation* of the heart while examining the pulse are matters that should never be neglected if reliable results are to be obtained. It is especially in this class of cases that the sphygmograph renders invaluable aid. Sphygmograms will often show the kind and degree of arrhythmia when all other means of examination have failed, and also distinguish marked dicrotism from irregularity.

It is important to *differentiate* functional arrhythmia or that of reflex origin from arrhythmia due to more or less grave myocardial disease. Important information is supplied by carefully reviewing the varied etiologic factors that produce the functional form, and by close observation of the cardiac symptoms.

The **prognosis** is variable. A gentleman with whom I am acquainted was rejected by a life-insurance company twenty years ago on account of occasional slight arrhythmia, though he is still in active business life and apparently in vigorous health. When the myocardium becomes involved, as occurs in chronic valvular or coronary disease or in the acute infectious diseases, the prospect is gloomy; on the other hand, when it is functional or due to other causes outside of the heart itself, the course pursued is as a rule favorable. When the second sound follows closely the first (marked abbreviation of the systolic pause) it is a serious indication.

Treatment.—There are many cases of the more benign form in which no treatment is required apart from methodic, physical training to improve the strength of the heart-muscle and the general systemic development. Removal of the causal forces, as tea, coffee, alcohol, indigestible food-stuffs, conditions acting in a reflex manner, must be executed promptly. When the condition is due to changes in the heart-structures, cardiants in addition to the general tonics should be prescribed. I prefer strychnin, arsenic, and the dried sulphate of iron in combination. Nitroglycerin is of service if the arterial tension be high. If the arrhythmia be due to excessive cardiac dilatation, digitalis should be employed. In purely functional cases, in which there is a predominating neurotic element, the subjoined formula has been useful in my hands:

℞. Ferri valerianatis,
Zinci valerianatis, āā. gr. xxx (1.94);
Strych. sulph., gr. j (0.0648);
Pulv. digitalis, gr. viij (0.518).
Ft. capsulæ No. xxx.
Sig. Take one after meal-time.

ANGINA PECTORIS.

(*Stenocardia*, *Breast-pang*.)

Definition.—A paroxysm of violent precordial pain extending into the neck, back, and arms, and at times attended by a sense of impending death. It scarcely deserves to be classified as a separate disease, being merely symptomatic of several cardiac or aortic (?) lesions.

Pathology.—Concerning the pathogenesis of angina many theories have been advanced, but conclusive *post-mortem* evidence in support of each and all is wanted. Neither does any single hypothesis fit the grouping of symptoms so constantly observed in cases of angina. It is to be recollected, however, that it is a neurosis affecting the cardiac sensory filaments that are given off chiefly from the pneumogastric, and in many cases the vasomotor apparatus is also involved. Allbutt[1] believes that the symptoms owe their origin to disease in the aorta—acute and chronic aortitis. Nothnägel has described a form distinct from the above in which the vasomotor apparatus is principally involved (*angina pectoris vasomotoria*).

Etiology.—Cases of angina unassociated with *arterial sclerosis*, *hypertrophy*, *aortic regurgitation*, or adherent pericardium, are rarely encountered. It is quite probable that with few exceptions sclerosis of the aorta and coronaries is present, and predisposes to the disease under discussion. This view also receives some degree of color from the fact that angina usually occurs after the *fortieth year*, and principally in the *male* sex. It may be a sequel of influenza. The overuse of tobacco may rarely cause true anginiform symptoms. The *determining factors* of the attack are undue exertion and mental emotion.

Symptoms.—The *paroxysm* begins quite suddenly during the action of one or other exciting cause. There is excruciating pain of a grip-like character, affecting the entire chest and rendering the body motionless. The pain *radiates* most frequently to the left shoulder, though also at times to the right, and thence to the back, neck, and down the arms to the fingers. *Coldness* and *numbness* of the fingers or in the precordial area may be present. Not less agonizing than the pain is the awful sense of *impending death.* The *countenance* is frequently pale, and may assume a leaden hue, and is usually bathed in cold perspiration. The *respirations* are exceedingly shallow or even temporarily arrested, and the patient's anxiety is extreme. The *heart's action* may be regular, and the arterial tension, as shown by the pulse, is generally increased. The *duration* of the paroxysm varies from a few seconds to a minute or two, and after the pain is over gaseous eructations, vomiting, or the discharge of a large amount of clear urine may occur. With the cessation of the attack comes instant relief from the cardiac symptoms. On making a careful examination of the heart subsequent to the seizure there may be an utter *absence of signs,* and, though there is weakness, this soon disappears. The attacks may *recur* at intervals varying from a few days to many years. I have usually found that cases associated with aortic regurgitation give the shortest intervening periods as a rule.

In *angina vasomotoria* the pain in the heart-region is preceded for a few minutes by pallor of the face, coldness and stiffness of the limbs, due to spasm of the peripheral vessels.

Pseudo-angina.—This is also a *paroxysmal* affection, that may be associated with hysteria, anemia, or neuropathic inheritance, and is occasionally produced by infectious diseases and poisons (see p. 676).

Diagnosis.—The characteristic events are a sudden, most intense pain in the substernal and left parasternal regions with marked constriction of the chest, the peculiar manner of radiation of the pain, and the fear of death. Less diagnostic, though of considerable value, are the brevity of the attack, the sudden onset and cessation of the pain, the age and sex, and the anxious, moistened features. There are also lighter forms, in which one or more of the diagnostic phenomena above described are absent. If they occur between the ages of forty and sixty years in persons in whom either arterial sclerosis or aortic regurgitation is present, this disease should be thought of; and after the exclusion of certain complaints in which paroxysmal pain is prominent, such as gastralgia, intercostal neuralgia, and locomotor ataxia, the diagnosis of angina becomes reasonably certain.

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[1] *Philadelphia Medical Journal*, June 30, 1900.

The distinction between true and pseudo-angina pectoris rarely presents formidable difficulties (see table below):

ANGINA PECTORIS.	PSEUDO-ANGINA.
Etiology indeterminate, though generally associated with arterio-sclerosis (including coronary disease) or aortic regurgitation.	The causes are—hysteria, neurasthenia, toxic agents, and reflex irritations.
Occurs after the fortieth year, usually in males.	Occurs at any age (over six years), and usually in females.
Paroxysms, provoked by undue exertion or mental emotion, are rarely periodic and nocturnal.	Paroxysms arise spontaneously, are periodic and often nocturnal.
Pain intensely severe, and constricting, its chief center being to the back of the mid-sternum and toward the left.	Less intense pain, more diffused over the thoracic region: sensation of cardiac distention.
Duration of attack from a few seconds to one or two minutes.	From a half to several hours.
Patient silent and body fixed.	Restlessness and emotional symptoms of causative conditions associated.
Arterial tension increased as a rule.	Usually not increased.
Prognosis unfavorable.	Entirely favorable.

The vasomotor form of angina must not be confounded with pseudo-angina, which is infinitely more common.

The **prognosis** is bad, yet uncertain. I recall two instances that occurred ten and twelve years ago respectively: the first attacks were survived with no recurrence. When the arteries are sclerosed (particularly the coronaries) life is often suddenly terminated during the course of the attack. Occasionally the sufferer dies of syncope. The nature of the causal and associated lesions must be considered in estimating the prospect of life. In the vasomotor angina of Nothnägel the outlook is less grave, while in pseudo-anginal attacks it is bright.

Treatment.—*Prevention* of the attacks in persons who are subject to them is of the most importance. In order to do this all known exciting factors are to be rigidly avoided. The patient should be instructed to carry constantly in a convenient pocket such agents as nitroglycerin and amyl nitrite, beads or *perles* (strength 3 to 5 drops), and also how to use them with judicious care immediately upon the first indication of an approaching paroxysm.

The *treatment of the attack* must be prompt and energetic, though carefully conducted, amyl nitrite being inhaled at once from a handkerchief in doses of 3 to 5 drops according to the severity of the attack. The patient should then be placed in a cool apartment free from disturbing sounds. *Locally*, the use of the ice-bag may prove efficacious and should be tried at first. Rarely, hot applications (hot cloths or sinapisms) give better results than cold. If the pain is not controlled promptly by this method, the nitrite should be reinforced by the hypodermic injection of morphin (gr. $\frac{1}{3}$—0.0216) combined with atropin (gr. $\frac{1}{120}$—0.0005). This usually brings speedy relief, and is best suited to those instances in which there is no increase of arterial tension. In cases exhibiting high arterial tension the tincture of nitroglycerin, hypodermically, should be employed (dose ♏j—0.066—to be repeated once in a minute if needful).

During the ***intervals between the attacks*** the aim should be not only to obviate the action of the exciting causes, but also to overcome any predisposing influences that may exist. Schott[1] prefers baths (effervescent) and passive movements to drugs or other methods. In true angina, gymnastic exercises, in the form of passive movements alone, should first be performed by an assistant, but later may be safely entrusted to the patient. In cases in which the arterial tension is habitually exalted, nitroglycerin in increasing doses is to be used perseveringly, beginning with ♏j (0.066) and increasing by ♏j (0.066) every five or six days until the physiologic effects are produced. Sodium nitrite may be employed similarly, the dose being gr. j–iij (0.0648–0.184) three or four times daily. Marked arterial sclerosis, particularly if there be a syphilitic history, is favorably influenced by a long course of potassium iodid. It may be prescribed in doses ranging from gr. v to xx (0.324–1.296) three times a day. When hypertrophy of the left ventricle is excessive, the use of the following is effective:

℞. Tr. aconiti rad.,		♏xlviij	(3.10);
Sodii bromidi,		℥ss	(16.0);
Elix. simplicis,	q. s. ad	℥iij	(96.0).—M.
Sig. ʒj (4.0) t. i. d.			

It may be omitted at the end of every two weeks for two or three days. The presence of a gouty diathesis would call for special treatment.

In the *vasomotor* form amyl nitrite and nitroglycerin are most valuable. Additionally, hot foot-baths, followed by friction of the extremities, are also of the highest utility.

The treatment of *pseudo-angina* must be directed at the cause of the complaint—the neurotic condition.

IV. CONGENITAL AFFECTIONS OF THE HEART.

THESE result from two leading causes: (1) Arrested development, and (2) Fetal endocarditis. Occasionally, both these factors are operative.

(1) **Arrested development** may produce a great variety of anomalies, some of which may be briefly enumerated: (*a*) Acardia, absence of the organ. (*b*) *Cor biloculare*, or *reptilian heart*, in which the septum between the auricles and ventricles is absent, thus reducing the number of chambers to two. This is an instance of reversion to a lower type. (*c*)

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[1] *Med. Rec.*, March 11, 1899.

Absence of the interventricular septum, the heart consisting of three chambers (*cor triloculare*). More frequently there is a mere perforation in or an incomplete development of the septum, and this is usually situated in the upper portion. Obstruction of the pulmonary orifice or of the conus arteriosus of the right ventricle are frequently conjoined conditions. (*d*) *Patency, or incomplete closure of the foramen ovale.* Persistence of the foramen is, in the majority of cases, associated with obstruction of the pulmonary valve, though it may be solitary. (*e*) An anomaly known as *ectopia cordis* deserves mention. This is a condition in which the sternum is usually divided vertically, and the heart is either entirely exposed or beating just beneath the skin in the cardiac, thoracic, or abdominal region. In this connection another and the most common form of malposition may be added—namely, *dextrocardia.* Here the heart occupies the right side, with reversion of the arch and displacement of the descending aorta to the right of the spinal column. Transposition of other viscera is usually associated. (*f*) *Anomalies of the valves.*—There may be either a numerical increase or decrease of the cardiac valves, particularly the segments of the semilunar valves of the aortic and pulmonary orifices. Supernumerary segments are usually rudimentary, and at the pulmonary valve one, two, or more are most commonly seen. A decrease in the number of segments is also most frequently observed at the arterial orifices, the aortic and pulmonic semilunar valves then being composed of two segments (bicuspid). This condition may be due to defective development on the one hand, or to endocarditis with resulting cohesion of segments on the other.

(2) **Fetal endocarditis** leads to valvular deficiencies in a manner similar to what occurs during the whole post-natal period. The valve-lesions originating during fetal life are most frequently situated on the right side, probably for the reasons that the ante-natal circulation is more actively carried on in the right than in the left heart, and that the former receives the oxygenated blood from the placenta. They may occur at the pulmonic, the aortic, or the auriculo-ventricular orifices. The changes are of the slow sclerotic form as a rule, and their character is determined largely by the antecedent anomalies that predispose to them. The leaflets present smooth, thickened, and contracted borders. Union of the mitral segments is common, and the chordæ tendineæ are often thickened and contracted. The small rounded bodies that are normally present on the mitral and tricuspid segments (*nodules of Albini*) must not be confounded with pathologic verrucosities.

The most frequent congenital valvular lesion is **stenosis of the pulmonary orifice** as the result of chronic endocarditis. Rarely, it is due directly to defective development, and perhaps more rarely still to endocarditis verrucosa. **Pulmonic constriction** of ante-natal origin may be an associated lesion in other forms of valvular disease in the young adult. With stenosis at the pulmonary orifice, there usually coexist stenosis of the conus arteriosus of the right ventricle, an open foramen ovale, and a patent ductus arteriosus; according to Peacock, "in 86 per cent. of the patients with congenital heart-disease living beyond the twelfth year the lesion is at this orifice." **Atresia of the pulmonary orifice** occurs, though less frequently than stenosis.

At the **tricuspid orifice** there may be stenosis or contraction of the valves, producing either obstruction or regurgitation. Similar lesions of the aortic orifice are infrequent. **Congenital mitral disease** also occurs, but only exceptionally; it is then usually associated with tricuspid stenosis. Boys are somewhat more liable to congenital affections of the heart than girls.

Symptoms.—There is an almost constant and strikingly distinctive symptom in congenital heart-disease—*cyanosis*. The *tint* of skin observed is variable, being at one time a general duskiness, at another a deep violet, and rarely almost black. This coloration is most noticeable about the lips and mucous membrane of the mouth, the nostrils, conjunctivæ, the fingers, toes, and lobules of the ears, and as a rule is general, though it may be a local condition. The tint may grow less distinct, and even almost vanish, when the child is in perfect repose or sleeping; excitants or efforts at coughing, however, increase the intensity of the discoloration. The cyanotic hue comes on almost invariably during the first week of life. The *fingers* present a decidedly clubbed appearance, and the *nails* are thickened and curved like the claws of certain animals. The *temperature* is subnormal, while the extremities are cool to the feel. *Dyspnea* on exertion and *cough* are usual concomitants. Cyanosis may be due to various causes that may act either singly or more often concurrently. Variot reports two cases with similar cardiac conditions—namely, interventricular perforation, and narrowing of the pulmonary artery, with the aorta arising from both ventricles. Cyanosis was absent from one case, and "this disproves the two leading theories with regard to the origin of cyanosis—the mixture of the two bloods and the obstruction to the pulmonary circulation."

Physical Signs.—In the very young the impulse is feeble (with an absence of a palpable thrill), the *percussion-dulness* is increased, especially to the right, and a loud *systolic murmur* is audible at the pulmonary orifice. When the auriculo-ventricular valves are the seat of endocarditis, the murmur may be apical. In pure pulmonary stenosis the second sound is feeble.

In older children the area of *dulness* is only slightly extended. particularly to the left, while the *murmurs* heard are loud and often musical.

It is interesting to note that in rare instances *cerebral abscess* is an associated condition.

Differential Diagnosis.—The distinction between congenital and acquired lesions in children may be assisted by a reference to certain points tabulated below:

CONGENITAL LESIONS.	ACQUIRED LESIONS.
History of almost constant cyanosis, beginning in the first week after birth.	Not so; history of endocarditis or of rheumatism or other complaints in which endocarditis occurs as a complication.
Slight enlargement of the heart. It is of the right ventricle, chiefly non-progressive.	Enlargement marked, frequently involving the left ventricle, and progressive.
Loud and musical murmurs present, audible over upper third of sternum, with small area of transmission upward and to the left; second sound weak.	Audible over apex or base; definite large areas of transmission. Second sound frequently accentuated.
Deficient bodily development.	Bodily development good, as a rule.
Mental faculties in abeyance.	Mental faculties normal.

Prognosis.—The prognosis is exceedingly grave. Many succumb within a few days after birth, more than one-half before the expiration of one year, and not less than three-fourths before the end of the third year. Few survive the first decade of life, and fewer still reach full adolescence. Among the forms giving the most favorable prognosis are pulmonary stenosis, especially when of moderate grade, and defective auricular and ventricular septa. In those instances in which life is spared in the first weeks after birth there is a disposition to affections of the lungs (phthisis, hemoptysis), nerve-complaints (convulsions, cerebral hemorrhages).

Treatment.—The treatment is, in the main, hygienic. The body must be warmly clad, flannels being worn next the skin, and every source of cold should be carefully guarded against. The diet is to be judiciously arranged, yet liberal, preference being given to the carbohydrates. Gentle exercise when it can be taken is valuable, as are also daily spongings of the surface followed by friction. Special therapeutic indications may arise, and must be met in accordance with general principles, while tonics, such as iron, arsenic, quinin, and cod-liver oil, are frequently applicable.

V. DISEASES OF THE ARTERIES.

ACUTE AORTITIS.

Pathology.—The morbid changes coincide with those noted in acute endocarditis, including the ulcerative variety.

Etiology.—The causes are not clear, but the condition generally follows the acute infectious diseases (typhoid fever, pneumonia, miliary tuberculosis). Alcoholism and syphilis are among the rarer causes. Various microörganisms have been discovered to be causal irritants. Boinet and Romary have recently shown that in experimentally produced aortitis a point of lessened resistance (either from traumatism or other previous arterial lesion) is necessary.

Symptoms.—The symptoms are *local* and *general.* Of the former, diffuse thoracic *pain,* with substernal *tenderness* under pressure and cardiac *palpitation,* are the chief. The pain may assume the type of true angina pectoris. Among the *general* symptoms a moderate febrile movement is almost constant. In a certain percentage of cases embolism is betrayed by the usual signs, as rigors, accompanied by a steep temperature-curve. These forms are analogous to the malignant variety of endocarditis. A cardiac murmur may be heard over the base.

Diagnosis.—All that the best clinicians can do is to establish a probable diagnosis even in the presence of the most frankly expressed features of the affection. From *acute endocarditis,* aortitis is to be discriminated by its diffuse pain and by the higher seat of its murmur.

The **prognosis** is serious, owing to the liability to infectious emboli and aneurysmal dilatation and the possibility of aortic rupture.

The **treatment** is similar to that of acute endocarditis.

ARTERIAL SCLEROSIS.

(*Arterio-sclerosis; Arterio-capillary Fibrosis; Endarteritis Chronica Deformans; Atheroma.*)

Definition.—An overgrowth of the connective tissue of the arterial coats (chiefly and primarily of the intima), followed by calcareous depositions. The elastic tissue of the intima is primarily increased.

Pathology.—The most frequent seat of the sclerotic process is the aorta, and the next most common the coronary arteries. Other vessels implicated are the arteries of the brain, the temporals, radials, brachials, ulnars, femorals, and iliacs. On the other hand, certain arteries, as the gastric, hepatic, and mesenteric, are rarely affected. Two forms may be recognized: (*a*) the circumscribed, and (*b*) the diffuse.

(*a*) **Circumscribed Arterio-sclerosis.**—Naturally, the intima presents a smooth internal surface, but when atheromatous changes occur it shows localized areas of thickening. These patchy prominences are often hemispheric in outline, yellowish-white in color, and their favorite seats are the orifices of the branches. They increase in depth and superficial area, and on reaching an advanced stage their interior disintegrates into granular material (*atheromatous abscess*).

In circumscribed or nodular atheroma the microscope discloses the fact that the middle and external coats are the primary seat of the changes, which consist of localized infiltrations. These lesions weaken the media and adventitia, and then (as shown by Thoma) compensatory processes are set up in the intima which lead to the formation of the so-called *atheromatous button.*' The latter consists in a hyperplasia of the intima with a deposit of round cells, which causes a gradual increase in thickness; in this way the growing weakness of the middle and outer coats is compensated. When the prominences in the intima undergo softening or liquefaction, rapid dilatation (*aneurysmal*) of the affected vessels is apt to occur. The same accident may arise early or before the intima has reinforced the other layers by its increased thickness and strength.

(*b*) **Diffuse Arterio-sclerosis.**—In this form the morbid process is distributed throughout the greater part of the arterial system, the nodular or circumscribed form being usually combined with it in the aorta. Dilatation of the aorta and of its branches commonly coexist. Apart from the yellowish, translucent, elevated areas, the intima may be smooth and the naked-eye appearances almost normal. The coats, however, and particularly the intima, are very much thickened. Microscopic examination brings to light an extensive proliferation of the subendothelial connective tissue and a hyaline transformation of the entire media, particularly in the larger vessels. The muscular fibers and elastic tissue have *in advanced cases* almost totally disappeared. Necrotic degeneration of the media, especially in the smaller arteries, is also observed, and calcareous deposits, causing rigidity of the walls, occur among the later changes. This is particularly true of the so-called *senile arterio-sclerosis.* Atheromatous abscesses that burst, forming atheromatous ulcers, are likewise common pathologic events in the aged. There may be associated atrophy of the heart, liver, and kidneys, due to a lack of nutritive supply in consequence of the narrowing of the vessels. More commonly, however, in this, and constantly in circumscribed and diffused sclerosis, the heart is moderately hypertrophied. When coronary disease is present, fibrous myocarditis and sclerosis of the aortic cusps may be found associated. The kidneys may also become sclerotic—a condition that will be described hereafter (*vide* Diseases of the Kidneys).

Sclerosis of the pulmonary artery, previously referred to in the discussion of the Diseases of the Heart, exhibits all the changes observed in connection with atheroma of the systemic arteries, including aneurysmal dilatation of the trunk and rarely of the main branches. From the terminal tributaries the process may extend to the capillaries, and even to the pulmonary veins (*angio-sclerosis*).

The effect of arterio-sclerosis upon the physiologic functions of the vessel-walls, and the pathologic and clinical results are of the utmost importance. The elastic coat is destroyed, and hence the walls cannot bear the blood-pressure as well as in health. This predisposes to dilatation of the vessels (aneurysm).

Another result of extensive atheromatous degeneration of the vessels is an increase in the resistance to the blood-current, and a consequent elevation of the arterial pressure. Furthermore, the loss of elasticity in the coats of the medium-sized and smaller arteries removes an important factor in the propulsion of the blood. The left ventricle, in consequence of this fact, almost invariably becomes hypertrophied in cases of extensive arterio-sclerosis, provided the general nutrition of the patient is still well maintained (Strümpell).

The reduction of the lumen of the vessel, owing to the thickening of the intima, must lessen the blood-supply to the various viscera, and thus in part are explained such secondary affections as fibrous myocarditis, cirrhosis of the kidneys, and cerebral softening.

Sclerosis of the veins (*phlebo-sclerosis*) may rarely accompany arterio-sclerosis. It is, however, not infrequently found in association with hepatic cirrhosis and mitral disease when the portal system and pulmonary veins are involved. It is occasioned by increased intravenous pressure. Arterio-sclerosis apart from sclerosis of the peripheral veins may be encountered, though rarely.

Microscopically, thickening of the intima and atrophic degenerative changes in the media are commonly observed. Calcification and hyaline degeneration of the layers also occur, and I have observed them in one of my own cases. Moderate dilatation is not exceptional.

Etiology.—The diffuse form has, in part, a special etiology. It may appear in the young, though rarely; I have met with a case in the Medico-Chirurgical Hospital in a man aged twenty-four years. It is, however, most frequent in the middle-aged (who are able-bodied) and in the aged. At an earlier period it occurs as a result of *alcoholism*, *syphilis* (the overshadowing factor), *lead-poisoning*, *gout*, and *chronic nephritis*—agencies that subject the vascular system to undue wear and tear. In old persons atheroma is often *physiologic* and characterizes the natural involution-period of life. *Heredity* may play no inconspicuous part in arterio-sclerosis dependent upon the age. This fact furnishes, to some extent at least, the reason why senile changes in the arteries occur at a much earlier period of life in some instances, and even throughout whole families, than in others. *Negroes* are more liable than *whites* to this form of atheroma, and *males* than *females*, though it is more frequent in the latter sex than the circumscribed variety.

The *general causes* may be thus classified—(1) *Biologic irritants*, as the specific micro-organisms of malaria, rheumatism, and syphilis. (2) *Chemical irritants* (chronic alcoholism, lead-poisoning, uric acid in gout). The above toxic agents produce their effects partly by their direct irritant action, and partly by increasing the resistance in the peripheral vessels and thus raising the arterial pressure. (3) *Bright's Disease.*—It must be admitted that there is a class of cases in which arterio-sclerosis is secondary to Bright's disease, but when found in association the former is more frequently the primary disease than the latter. The chief causes of chronic nephritis are also capable of setting up atheroma, and hence it must not be forgotten that the two diseases may develop independently of one another, and yet simultaneously, in consequence of the action of a common cause. (4) *Constant overfilling of the blood-vessels*, resulting from excesses in eating and drinking, in the opinion of certain authors, also causes arterio-sclerosis. (5) *Muscular over-strain*, which augments the blood-pressure while at the same time obstructing the peripheral circulation, is a leading factor. (6) The main causes of sclerosis of the pulmonary artery are *mitral disease* and *emphysema*.

Clinical History.—The disease may exist for years without becoming apparent; or it may be discovered incidentally at *autopsy* while palpating the arteries during the course of an examination for some supposed local visceral affection. The accessible peripheral vessels (radial, temporal, femoral, and brachial) should be carefully felt when the presence of the disease is suspected. The *walls* of the affected artery feel *hard*, and the *pulse*, owing to increased tension, is incompressible; as a result of this rigidity of the arterial walls the degree of vascular tension is difficult of estimation. In marked cases the pulse-wave may not be detectable on palpation. Again, the *tension* may be high, and yet sclerosis of the vessel-wall be slight or absent. When doubt arises as to whether or not sclerosis exists, the pulse should be palpated by means of two fingers. If now, while compression of the pulse is made with the index-finger, the middle-finger detects a pulse-wave, arterio-sclerosis is present. Rarely, however, a recurrent pulse may be felt notwithstanding, but, as Ewart points out, pressure on the ulnar artery at once arrests it. On account of the loss of elasticity of the vascular walls the pulse is retarded, and the *sphygmogram* shows a short sloping ascent, a wide top, and a slow, gradual descent, with almost an effacement of the dicrotic notch.

The opposition of the increased resistance to the circulating medium (due to the rigid vessel-wall) in the outlying portions of the body calls forth a correspondingly increased cardiac action, and thus *hypertrophy of the left ventricle* is engendered, with its customary symptoms and physical signs, including the ringing, accentuated second sound. The balance of the cardio-vascular forces may thus be maintained for a long period of time, during which the health of the patient often remains unimpaired. It happens sometimes that hypertrophy preponderates and veils completely the symptoms of arterio-sclerosis. In elderly persons suffering from atheroma the first sound is often surprisingly feeble. *Myocardial degenerations* frequently come on in the later stages, when dilatation of the left ventricle, accompanied by a mitral systolic murmur and marked rapidity of the pulse, may supervene. The *aorta* may be so dilated as to give rise to an abnormal area of dulness in the upper sternal region.

Palpitation, *dyspnea on exertion*, a feeling of *precordial constriction*, and light *febrile attacks* are not uncommon. *Angina pectoris* is an infrequent symptom except in coronary atheroma.

It cannot be stated absolutely that involvement of the *external arteries* implies a serious involvement of the aorta and its main branches. On the other hand, the circumscribed variety is not attended with characteristic alteration of the pulse. The *pathologic*, and more particularly the *clinical*, events above described may be more pronounced at one portion of the body than at others, and this fact has given rise to several distinct or *special types* (apart from the general or cardio-vascular form first depicted) according to the seat of the most marked symptoms and lesions. Among the latter I would mention the (*a*) cerebral, (*b*) pulmonary, (*c*) renal, and (*d*) peripheral types.

(*a*) **Cerebral Type.**—In the milder grades of this type such symptoms as headache, tinnitus, vertigo, syncopal attacks, and local palsies are variously blended as a rule. I have had under my care for two years a case of marked arterio-sclerosis in a man aged eighty years in whom tinnitus aurium and vertigo, with mild melancholia, are the only constant symptoms; on two occasions temporary aphasia was superadded.

Especially in the aged, the condition is apt to lead to *thrombosis* or cerebral *embolism*, small emboli being detached from the aortic area and conveyed to the brain, with the development subsequently of the symptoms of anemic softening of the latter. The loss of elasticity of the vessel-walls in atheroma renders them more liable to rupture than normal arteries, while the tension is much increased. Under these circumstances the danger from apoplexy is quite obvious.

(*b*) **Pulmonary atheroma** is considered in its clinical relations in connection with the diseases of the heart and lungs.

(*c*) The **renal type** includes those instances of kidney-lesion that are associated with or follow general arterio-sclerosis. The condition is essentially an atrophic nephritis, due to the diminution of the blood-supply to the organs in consequence of the narrowed lumen of the renal arteries.

(*d*) In the **peripheral type** the arteries leading to the extremities become obliterated to such an extent as to cause starvation of the tissue, with resulting gangrene.

Diagnosis.—Hardened arteries, increased arterial tension, left ventricular hypertrophy, and marked accentuation of the aortic second sound form a grouping of clinical characters that leaves no doubt as to the diagnosis. Not infrequently it is the occurrence of apoplexy, acute cardiac dilatation, or of some other such accident that leads to the discovery of general arterial sclerosis.

Raw has successfully skiagraphed the arteries by a special method; and C. Beck[1] and others have found that the x-rays are useful in determining the extent of arterio-sclerosis (*e. g.* whether local or general).

To **differentiate** the murmurs of dilatation of the left ventricle following the hypertrophy of this disease from *organic valvular lesions* is only possible by the history or the results of treatment. The systolic murmur over the aortic area in atheroma may suggest *aortic stenosis*. In such cases, however, the second sound is loud, and the pulse more voluminous than in aortic constriction (*vide* Aortic Stenosis).

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[1] *N. Y. Med. Journ.*, Jan. 22, 1898.

Prognosis.—Arterio-capillary fibrosis is an exceedingly chronic, though usually a progressive disease, and frequently it terminates life. The axiom that a man is as old as his arteries has been borne out by the test of extensive clinical observation. The condition may prove fatal either with great suddenness, as when it occasions apoplexy, or with unwonted slowness. Very rarely the aorta ruptures at the seat of an atheromatous ulcer, causing instant death.

Treatment.—Though the progress of the disease cannot in most instances be successfully stayed, it can be retarded frequently by correcting aggravating habits and by removing the influence of ascertainable causes. The syphilitic taint, if present, requires the liberal use of the iodids.

The *diet* must be simple and free from stimulating properties; skimmed milk is excellent, particularly if renal symptoms be manifested. Perhaps no other agent is so generally serviceable in all cases, especially in the earlier stages, as potassium iodid, which should be administered for several years, combined with appropriate gymnastics to regulate the bodily functions.

For the increased arterial tension nitroglycerin or the other nitrites should be employed. The former should be given in increasing doses until an impression has been made upon the blood-pressure, after which this effect should merely be maintained.

For the local aortic symptoms (fever, pain) absolute rest, a liquid and unirritating diet, and a small blister are most efficacious, together with internal minute doses of calomel, quinin, and potassium iodid.

ANEURYSM.

Definition.—A true aneurysm is a circumscribed dilatation of an artery, formed of one or more of its coats.

Classified according to their form, aneurysms are—(1) sacculated, (2) cylindric, and (3) fusiform. They are termed *axial* when the complete circumference of the vessel participates in this dilatation, and *peripheral* when a single sac is confined to the side of the vascular duct.

Miliary aneurysms occur along the course of the cerebral vessels, and are so termed on account of their minute size. On the other hand, they may attain the size of the human skull.

By a *false* aneurysm is meant one in which the coats are ruptured and the blood is found in the adjacent tissues.

A *dissecting* aneurysm is one that, owing to laceration of the internal coat, dissects between the layers of the vessel-wall. For its *seat* it usually selects the aorta, and may traverse its entire length.

An *arterio-venous* aneurysm arises from a direct fistulous connection between an artery and a vein (*aneurysmal varix*), or an aneurysmal sac may intervene (*varicose aneurysm*).

Pathology and Pathogenesis.—The wall of the aneurysm is commonly the seat of arterio-sclerosis, which, according to Malkoff,[1] is a compensatory arrangement. The intima is thickened, and to a less extent the media in the early stages. The media probably weakens first in most cases, and extreme atrophy of both the intima and media is not uncommon in the later stages, so that the wall of the sac is often formed almost exclusively of the adventitia. The intima (as in Daland's case of aortic aneurysm, in which there were both an old and a new transverse rent) may become lacerated, and finally the media and adventitia tear; this results in rupture unless the adherent neighboring structures compensate for the natural wall.

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[1] Ziegler's *Beiträge*, 1899, xxv.

The blood in the aneurysmal sac is partly fluid and is composed of old and new thrombi. The latter when comparatively recent may be soft, and when old may be firm or even calcified, yellowish in color, and adherent to the wall. With the progressive enlargement of the aneurysm surrounding organs are apt to be compressed and their functions disturbed.

Etiology.—Among recognized *causes* are—(1) **Arterio-sclerosis.**—It follows that the same conditions that originate the latter must also tend to bring about aneurysms. According to Rasch, syphilis was present in 56 per cent. and probably in 82 per cent. of 25 aneurysms of the aorta discovered in the course of 3165 necropsies at Copenhagen. (2) **Sudden Great Strain.**—This may be productive of aneurysm, particularly in the early stage of arterio-sclerosis or before compensatory endarteritis occurs. In no other manner can the fact be satisfactorily accounted for that most instances of aneurysm occur during the period of greatest bodily activity in the male sex. (3) **Embolic plugging of a vessel,** if complete, may cause aneurysmal dilatation on the proximal side of the point of obstruction. The development of aneurysm may under these circumstances be facilitated by the mechanical effects of the embolus, which may be of calcareous hardness, as when it comes from diseased heart-valves. Infectious emboli set up inflammation and softening. (4) **Mycotic Aneurysms.**—That aneurysms sometimes owe their existence to mycotic origin was first pointed out by Osler, who found an abundant growth of micrococci in the aneurysmal sacs. They are met with in ulcerative endocarditis, and are often small and usually multiple. (5) **Traumatism.**—Aneurysms have been produced experimentally by traumatism (Malkoff); hence it is obvious that it may become one of the assignable causes. (6) **Age and Sex.**—Aneurysms are most frequent between the *thirtieth* and *fiftieth* years, this being the period of greatest physical exertion. The *male sex* is more frequently affected than the *female*, owing to differences in occupation.

ANEURYSM OF THE THORACIC AORTA.

(*Aneurysma Aortæ.*)

The *thoracic portion* of the aorta is involved in about 75 per cent. of the cases, and the *abdominal aorta* and its branches in 25 per cent. Within the thorax nearly 60 per cent. of the cases originate in the *ascending portion of the aorta* (Lyman). Hare and Holden[1] collected 570 cases of aneurysm of the ascending arch, of which 504 were of the saccular variety.

Symptoms.—Intrathoracic aneurysms may exist, particularly if they are small, without symptoms or noticeable physical signs. When they attain to any considerable dimensions, however, they usually excite characteristic signs and distressing symptoms, the latter being the results of direct pressure, and hence varying with the seat and direction of the progressive enlargement. In a few instances truly diagnostic symptoms are present in the absence of a detectable tumor or physical signs. Finally, the more characteristic features—the tumor inclusive—may be more or less nearly intermittent. It is important to note the condition of the neighboring organs upon which pressure is exerted by the growing aneurysm, as well as the symptoms and signs thus occasioned. Aneurysms of the **ascending portion** of the arch usually *compress* the vena cava, causing *distention of the veins* of the head and arms, though in a proportionately small number of cases the subclavian may be the only vein compressed, with resulting *enlargement* and *edema* of the right arm. The largest aneurysms may even compress the inferior vena cava, causing edema of the lower extremities. The *heart* is displaced outward toward the left pleura, usually forward and upward, and rarely causing erosion of the ribs and sternum. The right recurrent laryngeal nerve may be implicated, giving rise to *dyspnea* and *aphonia*. *Pain* is a constant feature.

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[1] *Amer. Jour. Med. Sci.*, Oct., 1899.

Aneurysms of the **transverse portion** of the aorta, when they attain any considerable size, cause the most intense symptoms, owing to the relatively shorter antero-posterior diameter of the chest at this point, in consequence of which greater *compression* of the neighboring tissues takes place. By protruding backward they may exert pressure upon the trachea, causing paroxysmal *cough* and *dyspnea*, or on the esophagus, causing *dysphagia*; these are common events. The pressure may fall also upon the bronchus, inducing *dyspnea*, *bronchorrhea*, and *dilatation*, the latter in turn sometimes leading to circumscribed abscess. The left recurrent laryngeal nerve may be implicated, with resulting *aphonia*.

Upward extension of the aneurysmal process, with involvement of the coats of the carotid and subclavian on the left side, or of the innominate and carotid on the right, may occur. The *sympathetic nerves* in the cervical region may be irritated, causing dilatation; or they may be paralyzed, causing contraction of the *pupils*. Compression of the *thoracic duct* may occur, with resulting rapid emaciation. A *tumor* may appear in the jugular fossa.

The aneurysm may grow *forward*, in which event it lies directly behind the manubrium, which from the pressure becomes eroded and may finally disappear in part. In aneurysms involving the transverse portion of the arch, lateral pressure, both toward the right and the left, is also made, causing recession and compression of the lungs.

When the **descending portion** of the arch is affected the pressure is exerted upon the spinal column to the right, and upon the tissues as far as the shoulder-blade to the left. As a consequence of destruction and absorption of the vertebræ, compression of the spinal cord may ensue, and is an intensely painful process. Pressure may be made upon the esophagus, causing *dysphagia*, or upon the left bronchus, causing *bronchiectasis*, with its usual *sequelæ* (bronchorrhea, fetid bronchitis, gangrene of the lung).

The sac may, in consequence of the slow ulcerative process that attends its progress, *rupture* eventually into the bronchus, the pericardium (when situated near the sinuses of Valsalva), the pleura, the superior vena cava, or the esophagus, with sudden death as the result. Frequently repeated small *hemorrhages*, due to weepings from the thinned walls, may precede the fatal rupture. I saw a case of aneurysm of the transverse portion in which rupture into the esophagus resulted, with instantaneous death.

When the tumor has reached the subcutaneous tissue and bulges externally, the skin covering it becomes tense and shining, and with increased pressure the surface becomes reddened and finally necrotic. The necrosed area is covered with a dry brown scab, which later is thrown off, leaving an oozing surface. Rupture soon follows.

Leading Symptoms in Detail.—Among these *pain* stands primarily, being the first and most constant. It is of two kinds: (*a*) due to direct pressure upon and stretching of the nerves. When aneurysm is developed suddenly, a sharp, excruciating pain is felt in the upper sternal region, accompanied by a feeling of "something giving way." In consequence of the stretching of the nerves a constant pain is experienced that is subject to exacerbations when the intra-aneurysmal pressure is raised. Pressure against the bony structures causes erosion, and usually produces a continuous boring pain. In a recent case of aneurysm shown in clinic at the Medico-Chirurgical College, however, a tumor that had eroded the right half of the sternum, and of the size of a goose's egg, had given rise to no suffering whatever. In latent aneurysm there is an absence of pain until the growth terminates life. Anginose attacks sometimes occur when the sac has its seat near to the heart. (*b*) Reflected pains of a neuralgic character may be excited by aneurysm. This is true, in particular, of aneurysms situated in the transverse portion of the aorta, in which instances pain is commonly felt in the region of the neck and occiput and down the left arm. When the growth is situated along the course of the descending aorta, intercostal neuralgia may be excited, and it is probable that pain of this sort is due to pressure upon the nerve-trunks.

Cough.—The cough is paroxysmal, and frequently has a peculiar brazen, ringing character that points to its laryngeal seat. Pressure upon the windpipe excites a paroxysmal dry cough. Compression of a bronchus may lead to bronchiectasis, and the cough then occurs only in long and severe paroxysms which recur at intervals of a day or even longer, and are attended with copious, thick, ropy expectoration (*vide* Bronchiectasis).

Dyspnea is a conspicuous symptom in aneurysm of the transverse portion of the aorta (the aneurysm of symptoms—Broadbent). It arises (*a*) most frequently in consequence of pressure upon the recurrent laryngeal nerve, (*b*) direct pressure on the trachea, and (*c*) from pressure on the left bronchus. Marked stridor may accompany the first variety.

Paralysis of the vocal bands is occasioned by compression of the recurrent laryngeals, particularly the left, while a slight degree of compression or irritation of the same nerve causes *spasm* of the vocal cords. The symptoms indicating the presence of these conditions are hoarseness, cough, and aphonia respectively. The laryngoscope should be employed, since paralysis of one of the abductors may be present without giving rise to appreciable symptoms.

Hemorrhage may occur as a slow oozing, either from the point of compression in the trachea or externally; in either case the amount of blood lost is small. Profuse bleedings (often producing sudden death) take place in consequence of rupture of the sac into the lung, the bronchus, or the trachea.

Deglutition may be difficult, owing to compression of the esophagus. When an aneurysm has been diagnosticated or even suspected, the esophageal sound should not be passed for purposes of exploration, on account of danger of rupture of the sac.

Compression and irritation of the sympathetic system of nerves cause pupillary changes that have already been mentioned. With dilatation of the pupil there may be observed pallor of one side of the face, due to stimulation of the vaso-dilator fibers; on the other hand, with contraction of the pupil (due to paralysis of the dilator fibers) there is hyperemia of one side of the face and unilateral sweating.

Clubbing of the fingers and *incurvation of the nails* are not rarely met with in thoracic aneurysm.

Physical Signs.—*Inspection.*—Visible pulsation is one of the earliest appreciable signs. It is most frequently observed at the right side of the sternum, above the level of the third rib (second interspace), and much less frequently on the left side over a corresponding area. In aneurysm of the transverse portion pulsation may be seen at the episternal notch, though an impulse here may also be due to nervous palpitation, and have no connection with aneurysmal growths. The pulsation may occur in the absence of the slightest bulging; when associated with swelling, however, its diagnostic value is infinitely greater.

Involvement of the innominate artery produces pulsation in the neck above the sterno-clavicular junction, or less commonly above the sternum. Corresponding to the site of visible impulse, there is, sooner or later, bulging in most instances. It may, however, be so slight as to elude detection unless the keenest observation be practised, and in not a few instances the tumor itself is invisible from the front of the body, but is recognizable looking from behind or from either side. Again, on allowing the light to fall obliquely upon the chest slight prominences may be brought to view that would otherwise be inappreciable.

When the aneurysm is situated in the ascending part of the arch, the most frequent seat of the bulging—which varies in size from a hen's egg to a cocoanut—is over the first and second right interspaces near to, and frequently involving, a portion of the sternum; when seated just beyond the aortic orifice, a pulsating prominence may occupy the third interspace along the left sternal border; situated in the transverse section of the aorta, bulging of the upper part of the sternum is a frequent event, or a projection into the cervical fossa may occur, though with comparative rarity. In the descending portion the swelling, when present, is in the second and third left interspaces near to the sternum, or very rarely in the left scapular zone. The skin over the tumors has been described. The apex-beat is displaced downward and outward, chiefly from pressure, though to a lesser extent also from hypertrophy (functional).

Palpation.—The protrusion presents a more or less yielding and elastic mass, and when superficially seated fluctuation may be obtainable. The degree, and the rhythmic expansile character of the pulsation are to be noted, and also the fact that there is an alternate contraction and dilatation of the sac in every direction—a distinctive feature.

If the aneurysm is largely concealed, bimanual palpation should be employed, the palm of one hand being placed over the spine and that of the other over the sternum. In an inconsiderable number of cases aneurysmal pulsation is only yielded when the finger-tips are used, and quite rarely only at the end of expiration. A diastolic shock is often perceived, and forms a physical sign of no little value. A distinct systolic shock, sometimes accompanied by a purring fremitus, can also be felt over the aneurysmal sac.

Percussion.—If the growth be deep-seated, percussion may give negative results; when, however, the tumor causes bulging or comes in contact with the chest-wall, a proportionate area of flatness is presented. The abnormal field of dulness may be the only symptom present, as in an instance of suspected aneurysm that recently came under my care. Aneurysms of the ascending arch give flatness to the right of the sternum; those of the transverse arch, over the upper part of the sternum and to the left; while those of the descending portion are revealed by a flat area between the spine and the left scapula. With flatness of the percussion-note there is a sense of increased resistance. There is generally a moderate increase in the area of cardiac dulness. Conversely the left ventricle has been found of diminished size at necropsy.

Auscultatory percussion (practised after the method of Sansom and Ewart) quite often gives valuable results.

Auscultation.—Since murmurs owe their origin, in great part, to the presence of fibrin in the sac, they may be absent, and this even in the case of large aneurysms. When, as is usual, a murmur is present, it is systolic in rhythm, heard with greatest intensity over the flat area or body of the tumor, and is transmitted in the direction of the blood-stream, being, therefore, distinctly audible in the vessels of the neck and along the course of the aorta. The murmur has a booming quality.

Aortic regurgitation may be considered as associated with aneurysm near the aortic ring when a double murmur is heard. In a few instances the diastolic bruit is alone detectable. A much intensified, ringing second sound is present (unless marked aortic regurgitation coexists), and is a sign of the utmost significance for diagnosis.

The Peripheral Arteries.—The pulse in the vessels beyond the aneurysm is slowed. Hence the two radial pulses may exhibit differences in *time.* The *volume* of the pulse beyond the aneurysm is also lessened, and in cases of aneurysm of the abdominal aorta or the femorals it may be obliterated. Such differences as these will not only point to the existence of thoracic aneurysm, but also may indicate its *seat.* Thus, if there be dilatation of the transverse arch with no implication of the innominate, the pulse at the right wrist is strong and almost simultaneous with the cardiac impulse, while that on the left side is small, weak, and more retarded. If the reverse be true, then the aneurysm may be near to or involve the innominate.

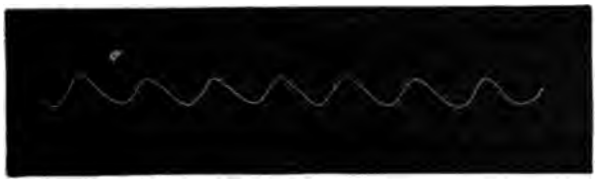

FIG. 55.—Sphygmogram of a case of aneurysm of the left subclavian artery (Foster).

The sphygmogram exhibits a slanting up-stroke with obliteration of the secondary wave (Fig. 55), though its characters are by no means constant.

Tracheal Tugging.—This sign may be practised while the patient is sitting or standing with the chin slightly elevated. The cricoid cartilage is then grasped between the thumb and forefinger and pushed gently upward so as to stretch the trachea. The patient must cease breathing momentarily, when, if this sign be present, there will be a downward dragging or tugging at each systole. The transmitted pulsations from the cervical vessels must not be confounded with the up-and-down movement of the trachea.

A new method of eliciting tracheal tugging, first suggested by Ewart, has been pretty widely adopted, and possesses the advantage of ensuring greater delicacy of touch than the old. He stands behind the patient, supporting the head of the latter against his body, and the cricoid is then grasped firmly between the tips of the forefingers. The method is in other respects similar to that previously described.

As shown by Toulmin, the tracheal tug may be present in health and in other diseases. The sign is probably one of less value than has been supposed.

Diagnosis.—In the presence of the following points the existence of thoracic aneurysm may be confidently inferred: (1) Antecedent arteriosclerosis (with the appropriate causes of the latter); (2) History of other etiologic factors, as age (between thirty and forty-five years) and occupation (such as entail unusual muscular strain); (3) Pressure-symptoms, as pain, dyspnea, aphonia, cough (either laryngeal or bronchial), bronchorrhea, dysphagia, edema, vasomotor disturbances; (4) Physical signs of a pulsating tumor (including the abnormal area of dulness, systolic murmurs, the systolic and diastolic shock, and tracheal tugging) somewhere along the course of the arch or its great branches, with or without differences in the volume and time of radial or carotid pulses. There are, however, several classes of cases which offer difficulties that are sometimes insurmountable: (*a*) Those in which the aneurysm is small and deep-seated. Here the symptoms and physical signs are indefinite. There may be thoracic oppression, in which pain may radiate to the left shoulder, and mild pressure-symptoms—a group of suspicious features merely—sometimes appear. I have under my observation now a patient suffering from aneurysm of the ascending aorta in which for a long time left-sided intercostal neuralgia was the only symptom. (*b*) Aneurysm of the transverse arch, in which the pressure-symptoms are more or less pronounced, but with no physical signs. In such, a clear history suffices to complete the diagnosis. Pressure-symptoms, on the other hand, without etiologic factors are just as likely to be due to other causes. (*c*) Those cases in which the more characteristic features are manifested intermittently. Fortunately, a proper diagnosis of aneurysm in obscure cases can be often made by the aid of the *x*-rays. The disease can also be excluded, in suspected cases in which it is not present, by fluoroscopic examination.

Extremely obscure are many of the cases in which the only symptoms manifested point to irritation of the trachea or bronchial tubes, with paroxysmal cough, bronchorrhea, and sometimes the signs of bronchiectasis. In a recent case of this sort tracheoscopic examination revealed compression of the windpipe, making clear the nature of the affection. In still another instance, in which laryngeal dyspnea and cough, with bronchorrhea, were the only symptoms, a laryngoscopic examination determined the diagnosis, in that it brought to view bilateral paralysis of the abductors of the vocal bands.

Differential Diagnosis.—The affections from which intrathoracic aneurysm must be distinguished are *pulsating empyema*, *pulmonary tuberculosis*, *abnormal pulsation of the aorta*, and *solid tumors*. Of the latter, those simulating aneurysm are carcinoma, sarcoma, and enlarged lymph-glands. These *mediastinal tumors* may duplicate all of the pressure-symptoms, though they are less apt to cause bulging, and less apt still to excite abnormal pulsation; when pulsation is noted it is observed to be quick, and not deliberate, heaving, and expansile, as in aneurysm. Solid growths also lack the characteristic shock—both systolic and diastolic—of aneurysm. The cardio-vascular symptoms are usually wanting in the case of solid tumors, especially the moderate hypertrophy, accentuation of the second sound, tracheal tugging, and the difference between the radial pulses.

Carcinoma of the mediastinum usually gives a history of the disease in other parts of the body, with enlargement of the axillary or other superficial lymphatic structures, and later the characteristic cachexia, this being particularly marked in carcinoma of the esophagus.

Abnormal pulsation in the aorta is noted in neurotic subjects, mostly females, and in aortic regurgitation; less frequently it is associated with retraction of the right lung, with spinal curvature, and with displacement of the aorta. In the case of the latter two conditions a careful consideration of the causal states and the absence of the characteristic physical signs would lead to a correct diagnosis. *Aortic regurgitation* is frequently associated with aneurysm of the arch, and in its course there is developed, not infrequently, a dilatation of the ascending portion of the aorta. The diagnosis of aneurysm of the arch of the aorta should not be made, even when all the characteristic physical signs of aortic regurgitation are present in any given case, unless the signs of the pulsating tumor above the heart be unmistakable. Dynamic pulsation of a neurotic origin is seen and felt in the episternal notch, as a rule, and a correct appreciation of the nervous element, whether inherited or acquired, will prevent the observer from committing an error.

Pulsating empyema can only be confounded with large aneurysmal growths, and, as pointed out by Wilson, it does not have the same definite relation to the central long axis of the body as do aneurysms. The abnormal area of dulness is situated at the base of the lung in empyema, and usually extends over a larger superficial area or is less circumscribed than in aneurysm. In empyema, moreover, the pulsation is not expansile, but is caused by pressure of the respiratory movements. Auscultation in empyema renders audible neither a bruit nor the double shock of aneurysm; the pressure-symptoms and pulse-characters are also entirely wanting.

Pulmonary tuberculosis may be mistaken for thoracic aneurysm. When an aneurysm compresses a bronchus, bronchiectasis, attended with cough, bronchorrhea, fever, and emaciation, may be the result; but in phthisis the fever and emaciation are more pronounced, tubercle bacilli are present, whilst the characteristic cardio-vascular signs of aneurysm are absent.

Prognosis.—The occurrence of perforation and consequent speedy death in unsuspected cases must be recollected. In other instances the end is approached in a very gradual manner, and cases in which rupture does not supervene sometimes pursue the general course of chronic valvular affections of the heart. The condition ends in death as a rule, and the *immediate causes* of the fatal issue are as follows: (1) Rupture of the aneurysm, followed by hemorrhage into any of the adjacent cavities or organs (pericardium, heart, large vessels, mediastinum, trachea, a bronchus, esophagus, lungs, pleura, spinal canal); it may, though rarely, rupture externally, in which case slight hemorrhages occur and life may last for weeks; (2) Gradual asthenia; (3) Direct pressure; (4) Independent diseases, either primary or secondary to, and induced by, the aneurysm. Among these *pulmonary complications*—fibrinous pneumonia, abscess, gangrene, tuberculosis—are of first importance.

Treatment.—There are two objects of treatment around which all others center in the management of this disease—first, the promotion of coagulation of the blood, and secondly, the contraction of the sac. The **clotting of the blood** within the growth may be greatly favored by retarding the blood-current. Nothing so well accomplishes this object as *absolute rest* in the recumbent posture. This cannot always be rigidly enforced, but muscular exertion must be minimized, mental application regulated, and emotional excitement avoided; stimulants, arterial and nervous, are to be eschewed for like reasons. Palpitation of the heart, when present, is to be allayed by the local use of the ice-bag. The coagulability of the blood is also increased by removing as far as possible the liquid portion of the diet.

The measures already indicated tend to lessen the volume of blood and the intra-aneurysmal pressure, thus inviting **contraction of the sac** as well as consolidation of its contents. Among medicinal agents, ergot and potassium iodid have been employed, the latter with good effects. The exact manner in which the iodid produces its favorable results in these cases is unknown, though most probably it acts upon the vascular walls, and hence would be most efficacious when the disease is of syphilitic origin; this view accords with my own personal experience. I would advise against the internal use of ergot, which can have little to recommend it, and the prolonged use of which may be attended with unpleasant effects. Langenbeck and others have obtained good results from the direct injection into the sac of the aqueous extract of ergotin dissolved in water or glycerin, every day or two. When employed in this manner ergotin induces contraction of the smooth muscles in the wall of the aneurysm. Numerous observers have resorted to the use of horse-hair, fine wire, fine catgut, slender watch-springs, with a view to coagulating the blood as it comes in contact with these foreign bodies. Electrolysis is a method that has been warmly advocated (Loreta).

Combined wiring and electrolysis (Corradi's method) has been successfully employed by Rosenstein, Kerr, D. D. Stewart, and Herchy. The details of the method are briefly as follows:

A piece of fine, slender wire, several feet in length, is passed directly from a spool through a hypodermic needle, so that the wire curls up within. This is now attached to the positive pole, while the negative is connected with a surface pad placed over the abdomen or with an insulated needle inserted into the sac, and the current is then passed through. It is important to test the strength of the current beforehand by inserting the needle attached to the positive pole into the white of an egg and observing its power to coagulate albumin. Each application of the current should last from one to two hours. It is not, however, without serious dangers (hemorrhage and embolism).

After the same method galvano-puncture has long been resorted to, and in the hands of some clinicians with encouraging results. The cases that receive most benefit from the above measures belong to the saccular variety; this is also true of the special plan first commended by Tufnell, which is especially applicable in the earlier stages. Tufnell's method is founded upon two main principles—absolute rest in the recumbent posture, and a much-restricted, dry diet. With physical rest a quiet mental state should be conjoined. The diet is as follows: Breakfast, 2 ounces (64.0) of bread and butter and 2 ounces (64.0) of milk; for dinner, 2 or 3 ounces (64.0–96.0) of meat and 3 or 4 ounces (96.0–128.0) of milk or claret; for supper, 2 ounces (64.0) of bread and 2 ounces (64.0) of milk.

The chief advantages growing out of this method are the lessened number and decreased force of the heart-beats in consequence of the posture and bodily rest, and the diminution of the blood-volume in consequence of the dietetic restrictions. It should be persevered in for several months. The bowels should be regulated, and the patient should be told not to strain while at stool.

A. E. Wright has particularly insisted upon the value of calcium salts in increasing the coagulability of the blood (gr. x to xv—0.648 to 0.972, t. i. d., may be given). A. E. Taylor's studies show that if it is desired to saturate the body with calcium salts, water should be given in abundance.

Injections of gelatin in aneurysm have a specially favorable effect according to certain observers. Moyer[1] reviews the literature, and in the main his conclusions are: Gelatin solutions are of some value in the treatment of saccular aneurysms, but not of the diffused forms. Solutions not stronger than 1 per cent. should be used; they should be kept in a brood-oven to determine bacterial growth, and great care should be taken in the technique. Absolute rest in bed should be enjoined. This method is worthy of extended trial, but great caution and watchfulness must be exercised in its administration.

Special Symptoms.—Pain is often relieved by potassium iodid. When marked arterial sclerosis is present I have seen relief from pain afforded by the internal use of nitroglycerin (♏j to ij—0.066 to 0.133, three or four times a day). In the later stages morphin should be given to allay suffering. When there is bulging the pain may be assuaged by the local use of the ice-bag or by a belladonna plaster.

Dyspnea and great venous congestion are to be met by free bleedings from a vein, and tracheotomy may be required if the dyspnea be shown to be due to bilateral paralysis of the abductors. In dyspnea arising from pressure on the trachea or bronchus, however, tracheotomy would be a valueless expedient. When the aneurysm forms a large external tumor the application of an elastic bandage to the chest may be both agreeable and advantageous, as in a case referred to by Osler.

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[1] *Medicine*, March, 1899.

ANEURYSM OF THE ABDOMINAL AORTA.

The vicinity of the celiac axis is the favorite seat of abdominal aneurysm, which is by no means so common a condition as intrathoracic aneurysm, though not rare. It may assume a fusiform or saccular nature.

Symptoms.—The tumor may grow backward; but more frequently its growth is in a forward direction. Projecting from the *posterior wall*, it usually erodes the vertebræ, and compression of the cord is apt to take place, producing *paraplegia*, preceded by *tingling* and *numbness* of the *legs*.

Pain is the leading symptom. It may be neuralgic or of a boring or gnawing character, due to destruction of the bone. Rarely, the aneurysm perforates the diaphragm, and finally *ruptures* into the lungs or pleura. Arising from the *anterior wall*, it may early form a well-defined tumor. It may, however, when situated high up or near the diaphragm, conceal itself until it has attained a comparatively large size, as in a case recently under my care at the Medico-Chirurgical Hospital. *Vomiting* and *gastralgic seizures* may be troublesome, and the fact that *embolism* of the superior mesenteric artery may occur and give rise to severe colicky pains must be recollected. *Jaundice* has been observed.

Physical Signs.—Epigastric pulsation may be *visible*, and occasionally an epigastric swelling. The *palpating* hand detects a heaving, expansile pulsation that may be accompanied by a thrill. When the tumor hugs the diaphragm the pulsation may be double. The femoral pulse is diminished in volume and delayed. An abnormal area of dulness may be present. In most instances a *soft bruit* is audible. The diastolic murmur and shock of *intrathoracic aneurysm* are quite usually absent.

Diagnosis.—A certain diagnosis demands the presence of a definite growth that is seizable and has a heaving, expansile pulsation. Mere pulsation attended with a thrill and a systolic murmur are insufficient.

Differential Diagnosis.—A *throbbing aorta*, as met with in neurotic females and in anemia (particularly in instances of the traumatic form), is not infrequently distinguished from aneurysm of the abdominal aorta with great difficulty. It does not, however, present a tumor that can be held in the grasp and possessing an expansile pulsation, as in aneurysm.

When *solid growths* lie upon the aorta the latter may manifest pulsation, a thrill, and a systolic murmur, but the very general absence of pulsation, owing to the fact that the tumor falls forward when the patient is placed in the knee-elbow position, suffices usually to differentiate the condition from genuine aneurysm. Again, expansile pulsation is not evinced by a solid growth.

The **prognosis** is very gloomy. Very rarely, however, nature effects a cure if the conditions be favorable. "Death may result from (*a*) the complete obliteration of the lumen by clots; (*b*) compression-paraplegia; (*c*) rupture either into the pleura, retroperitoneal tissues, peritoneum, the intestines, or, very commonly, into the duodenum; (*d*) embolism of the superior mesenteric artery, producing infarction of the intestines" (Osler).

Treatment.—Apart from the measures indicated for thoracic aneurysm, there is one means of cure that may be tried if the growth be low down—viz. *pressure.* This must be maintained for twenty-four hours at least under an anesthetic. It is best to make steady pressure on the proximal portion of the vessel, and unless practised with great care the sac will be damaged and death ensue.

ANEURYSM OF THE PULMONARY ARTERY.

Dilatation of the pulmonary artery is of frequent occurrence in affections that oppose obstruction to the lesser circulation (*e. g.* mitral disease, emphysema, phthisis). Very rarely extreme dilatation of the vessel is followed by semilunar incompetence, when a diastolic murmur at the pulmonary orifice (second left interspace) becomes audible.

Aneurysms involving the pulmonary artery are quite rare: such as occur are small and of the saccular and fusiform varieties.

The **symptoms** resemble those of *intrathoracic aneurysm,* though they are rarely well marked, owing to the fact that they remain of small size as a rule.

Physical Signs.—Pulsation (and, rarely, a small tumor) is detectable in the second left interspace. *Palpation* may also render appreciable a thrill and diastolic shock. Coextensive with the area of pulsation there may be *dulness* on percussion, and over the second interspace to the left of the sternum a loud *superficial systolic murmur* is heard on auscultation, together with a diastolic shock. Before attaining to a large size, these aneurysms usually *rupture* into the heart itself.

The **prognosis** is altogether unfavorable, the treatment having reference to the principles that are appropriate in thoracic aneurysm.

The **coronary arteries** may be the seat of aneurysm, though exceptionally. The condition arises in consequence of weak points (due to arteriosclerosis) in the course of the vessels, and is unrecognizable during life.

ANEURYSM OF THE CELIAC AXIS.

This condition is sometimes observed in combination with aneurysm of the upper portion of the abdominal aorta.

ANEURYSM OF THE SPLENIC ARTERY.

This branch of the celiac axis is occasionally the seat of aneurysmal dilatation. It may be single or multiple, and, whilst it is small as a rule, may in rare cases be quite large.

The **symptoms** are indefinite, but distressing. Deep-seated abdominal *pain,* which shows a tendency to radiation, forms, with *vomiting,* and rarely *hematemesis,* the main features. By *percussion* a tumor may be mapped out in the left hypochondriac region, the dulness merging with that of the spleen and the left lobe of the liver. Usually, pulsation, and, rarely, a tumor can be *felt,* and *systolic murmur* is often heard. The condition may be confounded with *gastric ulcer.*

ANEURYSM OF THE HEPATIC ARTERY.

This is exceedingly rare, the total number of cases on record being about 20. H. B. Schmidt has recently reported a case associated with symptoms of gall-stones, in which, as shown by the autopsy, death was caused by rupture of the sac into the bile-ducts. Schmidt found records of but 5 cases of this mode of termination. Osler and Ross have reported an instance associated with multiple hepatic abscesses.

The **symptoms** are, in the main, *colicky pains*, *vomiting*, *hematemesis*, and *obstructive jaundice*. A *tumor* is rarely discernible, though an abnormal area of pulsation is relatively more frequent. The recognition of the condition during life is entirely conjectural.

Aneurysm of the superior mesenteric artery is of rare occurrence. *Pain* in the epigastric and lumbar regions, and *demonstrable tumor* near to or directly over the median line of the abdomen, are the symptoms displayed. Detached fragments of the clot may produce *embolism* of the terminal branches of the mesenteric arteries. The condition terminates usually in rupture into the peritoneal cavity.

Aneurysm of the inferior mesenteric artery runs a course similar to the above. It is quite rare and possesses slight interest.

Aneurysm of the Renal Arteries.—Small multiple dilatations are occasionally seen, but large ones are of great rarity. They are prone to rupture into the retroperitoneal cavity.

ARTERIO-VENOUS ANEURYSM.

Definition.—An artificial communication between an artery and a vein. A sac may intervene between the two vessels (*varicose aneurysm*) or there may be a direct fistulous communication without an intervening sac (*aneurysmal varix*).

In varicose aneurysm the sac is developed from the structures that mark the boundaries of the communicating duct. The majority of cases are caused by the simultaneous wounding of an artery and a vein during venesection. Hence their most frequent seat is at the bend of the elbow. Pepper and Griffith have analyzed the records of 29 cases in which the ascending portion of the aortic arch had opened into the vena cava.

Symptoms.—The symptoms are largely *aneurysmal*, and in addition there appear in rapid sequence *great swelling of the veins*, *cyanosis*, and *edema* of the upper portion of the body. A continuous *thrill* and *buzzing murmur*, with systolic intensification, are the chief signs.

In the **treatment** of thoracic arterio-venous aneurysm the same general plan is to be pursued as advised in the purely arterial variety.

CONGENITAL ANEURYSM.

This condition arises because of a defective ante-natal development of the elastic coat. It is often multiple, and the tumors are, as a rule, small in size, ranging from that of a pea to a hazel-nut. The most common situations for these growths are the coronary and pulmonary arteries. To Eppinger belongs the credit for having pointed out the fact that the aneurysmal walls consist only of the adventitia and intima. "*Peri-arteritis nodosa*," a rare condition, which Eppinger holds to be a form of congenital aneurysm, presents the symptoms of general infection rapidly developed. "On examination after death the arteries are found beset with nodules of active inflammatory products, chiefly on the outer coat" (Allbutt). The condition may be met with in children and rarely in adults.

PART VI.

DISEASES OF THE DIGESTIVE SYSTEM.

I. DISEASES OF THE MOUTH.

STOMATITIS.

CATARRHAL STOMATITIS.

(*Stomatitis Erythematosa.*)

Definition.—A simple, acute inflammation of the buccal mucous membrane. It is more commonly met with in children than in adults.

Etiology.—As a *primary* affection its causes are mainly mechanical and chemical irritation, such as the presence in the mouth of hard and sharp bodies, dental caries, acids, hot or cold food, condiments, tobacco, certain drugs (as mercury), eruption of teeth, and bad feeding, particularly in illy-nourished children. It is the result often of a neglect of the mouth-toilet, leading to the decomposition of accumulated bits of food and mucus, and many cases probably owe their origin to infection. *Secondarily*, catarrhal stomatitis may be associated with certain of the eruptive fevers (scarlet fever, measles, typhoid), also with gastroenteric derangements, and may follow, by direct inflammatory extension, upon ulcerative tonsillitis, pharyngitis, and the like.

Symptoms.—The *local symptoms* of this affection are those usually seen in an inflammation of a mucous membrane—redness, heat, swelling, and dryness, soon followed by increased secretion and soreness. The lips and gums only, or the membrane of the whole mouth, may be inflamed, and the swollen lips, cheeks, and furred tongue may be indented by teeth-marks. Enlarged and reddened papillæ on the tongue and minute vesicles inside the cheeks and lips from projecting mucous follicles are sometimes seen. These, later, may terminate in simple small ulcers. A craving for cold drinks is nearly always noted, as well as distress and even pain on suckling, mastication, or touching with the inspecting finger, and there is a disagreeable taste due to the perverted buccal secretions. *Chemical examination* of the dribbling saliva shows an acid reaction, with the presence, microscopically, of an excess of desquamated pavement epithelium that has undergone partial fatty degeneration. Leukocytes, micrococci, the leptothrix buccalis, and the remains of food may also be seen (Strümpell). Aside from restlessness and the symptoms common to slight febrile disturbances, the *constitutional condition* is rarely disturbed, except when the stomatitis is secondary either to inflammations lower down in the digestive tract, or to the specific infectious fevers.

The *course* of the disease is usually acute, and the duration about one week.

The **differential diagnosis** of catarrhal stomatitis is easily made by inspection of the membrane.

The **prognosis,** though favorable, will vary as to time and severity according to the cause.

Treatment.—After proper attention to the hygienic surroundings of the patient and the removal of all irritant influences, the treatment is mainly local. The first indications are to cleanse the mouth and allay the pain, and these may be met by the use of cool solutions of boric acid, sodium bicarbonate, or potassium chlorate, 5 and 10 grains (0.324 and 0.648) respectively to the ounce (32.0) of glycerin and rose-water, as mouth-washes, or for swabbing in the case of infants. When iced drinks are ungrateful and the inflammation is more intense and protracted, the use of hot milk and lime-water, mucilaginous decoctions, and sedative and antiseptic sprays of 1 or 2 per cent. solutions of cocain or carbolic acid are often beneficial; or mild astringents may be needed, as ½ to 1 per cent. solutions of silver nitrate, alum (5 to 10 grains—0.324 to 0.648—to the ounce—32.0—of honey), and glycerite of tannin (2 drams to the ounce—8.0 to 32.0—of water), especially if there is a tendency to chronicity of the trouble, as in topers and inveterate smokers. Tender and spongy gums may be relieved by the application of equal parts of the tinctures of myrrh and rhatany on a camel's-hair brush (Strümpell). *General symptoms* as they arise must be met by the remedies rationally indicated. Small doses of aconite or potassium citrate for the pyrexia, with a minimum dose of bromid for irritability and sleeplessness, may be all that is required. The internal use of potassium chlorate in children is not to be recommended in this affection, both because of its deleterious action upon the kidneys, and also because it seems to be unnecessary (Forchheimer; Blackader). Sometimes an associated gastro-intestinal catarrh needs correction by the use of laxatives. The administration of bland foods and mild ferruginous tonics should be continued throughout convalescence.

APHTHOUS STOMATITIS.

(*Follicular Stomatitis; Stomatitis Aphthosa.*)

Definition.—A variety of catarrhal stomatitis that is characterized by the eruption of one or more vesicles upon the edges of the tongue, the cheek, or the lips, rapidly passing into small round, or oval discrete spots that are slightly raised and surrounded by yellowish-white bases with narrow red areolæ.

Etiology.—Though more common in children between the ages of two and six years, they are by no means rare in adults. Predisposing influences may be found in the seasons (spring and autumn), malnutrition, tuberculosis, dentition, persistent gastro-enteric disorders, anemia, and the acute exanthemata. The *exciting causes* are supposed to be certain deleterious substances, bacterial or toxic, though no special parasite has yet been isolated.

Symptoms.—The herpetic vesicles soon rupture, leaving the aphthous ulcers as described above. They are found singly, or at times as many as twenty in number, pin-head to split-pea in size, inside the lips, especially near the frenum, along the tongue-edges, and sometimes inside the cheeks near the edges of the back teeth. They are exquisitely *tender*, so that almost any motion of the affected parts causes sharp burning pain; nourishment is therefore difficult. Patches of *catarrhal stomatitis*, and even of gingivitis, are seen adjacent to the aphthous spots. There is an increased flow of the secretions of the mouth, and the breath is heavy, though not offensive. *General symptoms*, as slight fever, anorexia, and furred tongue, constipation or diarrhea, and irritability, are usually present, with the additional symptoms of any associated disease that may coexist. Gastro-intestinal affections, though often associated with aphthous stomatitis, are most probably due to the common cause, and are not necessarily the cause of the stomatitis in these instances. In some of the specific infectious fevers many aphthæ may appear and tend to run together; these form large irregular ulcers, and give rise to the *confluent form* of stomatitis aphthosa. The special form known as *Bednar's aphthæ*, occurring in young marantic babes, is a rare condition in America. Large white patches are seen on both halves of the posterior part of the hard palate near the alveolar processes, and these may cause large ulcers and involve the bone. Pressure of the tongue upon the thin mucous membrane during nursing, or other forms of traumatic irritation, appear to act as causes.

Aphtha Cachectica (Riga's disease).—Fede has described a form of aphtha, occurring principally in Southern Italy (a raised, gray swelling), situated on the frænum and under surface of the tongue. It affects children soon after the eruption of the lower incisors. A severe type is sometimes met, and this may terminate fatally.

The average *duration* of the ordinary discrete aphthous eruption is from four to seven days; in very ill-nourished and poorly cared-for cases the appearance of successive crops of aphthæ will prolong the distress.

Diagnosis.—This is based upon the characteristic appearance of the ulcers and the degree of soreness. Aphthæ must be differentiated sometimes from *thrush*, and the distinguishing features will be dwelt upon in the description of the latter affection. Herpes of the mouth, so called, and aphthous vesicles are probably identical in most cases.

Prognosis.—The discrete form is mild, and favorable in its course toward recovery; confluent aphthæ is more troublesome, and follows a prolonged course on account of the general debility induced by the associated disease (Starr). In certain cases the affection is apt to recur; relapses are also frequent in those having weak digestive and imperfect assimilative functions. Recovery from Bednar's aphthæ is rare.

Treatment.—It is first necessary to remove all irritating influences, and in order to minimize the intense pain of the aphthous spots the blandest liquids and the softest foods that are consistent with the sustenance of the patient are imperative. Absolute cleanliness of the mouth, the foods, and the vehicles of administration, especially in bottle-fed children, is important. Local applications are of obvious value. Demulcents, as mucilage of sumac, or of marshmallow, with boric acid (gr. v to ℥j—0.324 to 32.0), sodium bicarbonate (gr. v–x to ℥j—0.324-0.648 to 32.0), carbolic acid, or potassium permanganate (gr. iv to ℥j—0.259 to 32.0), are invariably useful. Swab-applications of wine of opium (♏v to ℥j—0.333 to 32.0) or of cocain (4 per cent. solution) may be necessary when the pain is intense, and prior to taking food.

To promote the healing of the ulcers a very light touch with the silver-nitrate stick or solution (gr. x–xxx to ℥j—0.648–1.94 to 32.0) is often beneficial. Much favor is deservedly given also to potassium chlorate in solution (gr. x to ℥j—0.648 to 32.0), or applied in the dry powdered form. In the confluent aphthous ulcer the use of sodium salicylate (ʒj to ℥j—4.0 to 32.0) has been recommended, while an ethereal solution of iodoform (ʒij to ℥j—8.0 to 32.0) has been advised by J. Lewis Smith. For bleeding and spongy gums the mild astringents mentioned in the treatment of catarrhal stomatitis are indicated. Stronger astringents may answer for application to sluggish ulcers; thus copper sulphate, either solid or in solution (gr. x to ℥j—0.648 to 32.0), and zinc sulphate (gr. xv to ℥j—0.972 to 32.0) are useful. Potassium chlorate acts as a specific in ulcers of the mouth, and is eliminated by the buccal secretions, which keep the ulcerated surfaces constantly bathed with the drug, so that its internal use is to be recommended, though in very small doses in children, being given well diluted, as in the following formula:

℞. Potassii chlorat.,	gr. xxiv	(1.55);
Tinct. myrrhæ,	gtt. x	(0.666);
Syr. acaciæ,	f℥ij	(64.0);
Aquæ dest.,	q. s. ad f℥iij	(96.0).—M.

Sig. Teaspoonful every three hours for a child three years of age.

Constitutional symptoms are to be combated on general principles and require careful attention. Remedies directed to the correction of digestive derangements and to the stimulation of assimilation are also often required. Good food and ferruginous tonics are necessary.

MEMBRANOUS STOMATITIS.

(*Stomatitis Crouposa.*)

Definition.—In this form of stomatitis the inflammation is more intense and more extended in area than in the aphthous form, being also attended with the formation of a false membrane.

The **pathology** of these membranous patches, which are very seldom confined to the mouth alone, is embraced in the article on Diphtheria. If in the latter disease the typical false (diphtheritic) membrane is removed, it leaves a deeper ulcer than does the removal of a croupous membrane, in which the coagulation-necrosis involves the more superficial layers.

The **etiology** of membranous stomatitis is usually specific (diphtheritic, streptococcic). Membranous stomatitis may also be due to gonorrhœal or syphilitic infection of the new-born.

Symptoms.—Some of these cases are, doubtless, true diphtheria of the oral cavity (usually secondary from extension), and an account of the symptoms presented may be found in the chapter on Diphtheria, p. 179. The writer has seen instances of extensive membranous stomatitis, in which bacteriologic examination showed the presence of streptococci (principally), and also staphylococci. The symptoms were analogous to streptococcic membranous pharyngitis with this difference, in my cases of membranous stomatitis, that salivation was marked and distressing. The *general features* may be quite pronounced.

ULCERATIVE OR FETID STOMATITIS.

(*Stomatitis Ulcerosa.*)

Definition.—A specific ulcerative inflammation of the buccal mucous membrane and gums, attended with marked fetor of the breath, and having a tendency to extend widely and deeply.

Etiology.—The predisposing causes of this malady are principally as follows: Childhood, after the commencement of the first dentition, and usually between the ages of three and eight years; damp weather, especially during spring and autumn; unhygienic surroundings, particularly the lack of pure air, of good and abundant food and clothing, and the added detriments to health for which neglect and filth, specific infectious diseases, uncleanliness of the mouth, caries and loosening of the teeth, and congenital heart-disease (Duckworth) are responsible. An endemic type of this affection has been observed among soldiers in camps and barracks, among children in crowded eleemosynary institutions, and in jails. Its epidemic and contagious character likewise points to a microbic origin. The specific *exciting cause*, it has been held, corresponds to the hoof-and-mouth disease of cattle, the poison being conveyed in milk. Payne suggests the identity of the virus with that of impetigo contagiosa. The careless administration of mercury may also be followed by this affection. Scurvy (*scorbutic stomatitis*) and the persistent use of lead and phosphorus are also excitants.

Clinical Symptoms.—*Locally*, the disease starts, as a rule, at the edges of the gums opposite the lower incisor teeth, gradually spreading backward and to the adjoining portions of the lips and cheeks. The gingival mucous membrane is deeply red and swollen; the gums soon become spongy, bleed easily, and break down into thick, soft, grayish sloughs, which leave deep and ragged ulcers surrounding the necks of the teeth. The latter even become loosened, and in protracted cases the alveolar periosteum may become inflamed and cause necrosis of the bone. Profuse salivation, a foul breath (that once earned for the condition the term of "putrid sore mouth"), occasional slight hemorrhages from the gums, and excessive discomfort, or even pain, on mastication are nearly always present. The tongue is coated, swollen, and tooth-marked; aphthæ are sometimes seen, and the submaxillary glands are generally swollen. The *general symptoms* attending this ailment are those of a lowered state of vitality, produced by an unhygienic environment, or cachexia, or severe illness primary to it, with, usually, moderate fever. Nausea and vomiting or an offensive diarrhea may supervene as the result of swallowing the putrid discharges.

Course and Duration.—Though acute in its course, the highly debilitating character of the disease may tend to make it chronic, especially when there is alveolar necrosis and a neglect of proper treatment. Ordinarily, with careful management, convalescence may be established in from four days to a week. Goodhart regards the occasional termination of the pyrehia by lysis, with an accompanying improvement of the local symptoms in such cases at least, as suggestive of the specific nature of ulcerative stomatitis.

Neurotic Ulceration.—Under the head of ***stomatitis neurotica chronica*** Jacobi describes an herpetic (rarely pemphigoid) eruption in neurotic subjects. Sibley has met three cases, all of which occurred in women beyond middle age. In all cases the condition had lasted for a number of years and produced great difficulty in talking and mastication.

Diagnosis.—Upon examining the mouth and noting the characteristic ulceration, the fetid breath and saliva, and the cachectic appearance, the disease is usually recognized, and should not be confounded with the dark, extensive, gangrenous sloughs of noma.

The **prognosis** is favorable in typical cases, though less so in marasmic and neglected cases. When cancrum oris or necrosis of the jaw occur, chronicity, deformity, and even death, may take place.

Treatment.—It is well in nearly all ill-nourished, uncleanly-kept, and sickly children, as well as in cases in which mercury is to be administered for any length of time, to prescribe mouth-washes of potassium chlorate (gr. xv to ℥j—0.972–32.0), in order to prevent the occurrence of mercurial or ulcerative stomatitis. The ***hygienic treatment*** of ulcerative stomatitis is important. On account of the contagiousness of the disease cases occurring in a family or in institutions should be isolated, and fresh air, light nourishment, and cleanliness are ***sine quâ non*** of recovery. The ***local*** treatment is virtually a specific one in the use of potassium-chlorate washes (gr. x–xx to ℥j—0.648–1.296 to 32.0—of rose-water or demulcent), aided by the internal administration of the same salt in small doses. For the disagreeable fetid odor the alternate use of antiseptic washes is indicated. Solutions of carbolic acid or potassium permanganate, in strength equal to or slightly over that indicated in the treatment of aphthæ, or hydrogen peroxid (ʒj–iij to ℥j; 4.0–12.0 to 32.0), or listerin and water (equal parts), are useful. Pencilling the spongy gums with such astringents as tincture of rhatany, silver nitrate (gr. x to ℥j—0.648–32.0), alum, and also with tannic-acid solutions, may be necessary. Loosened teeth should not be disturbed, as they may grow firm with convalescence, though surgical interference may be required in cases of necrosis of the alveolar process. Kissel's method in obstinate cases is to curette the ulcers and rub into them powdered iodoform once daily. Careful attention to the teeth is always requisite. During the height of the disease constitutional treatment may have to be directed toward stimulating the languid and lowered vitality. For this purpose either whiskey or brandy, in half- or one-teaspoonful doses in milk, is extremely useful; the elixir of cinchona, with some vegetable salt of iron, as the citrate or tartrate, also forms a useful combination. When there is pyrexia or a diminished urinary secretion the internal use of potassium chlorate must be cautiously employed. The following prescription is almost specific:

℞.	Potassii chloratis,	gr. xlviij	(3.11);
	Acidi hydrochlor. dil.,	fʒj	(4.0);
	Syrupi,	fʒvj	(24.0);
	Aquæ destillat.,	q. s. ad f℥iij	(96.0).—M.

Sig. Teaspoonful diluted, every two hours for a child three years old (Starr).

The prolonged use of tonics and cod-liver-oil emulsion with lime-salts in scrofulous, rachitic, and scorbutic subjects must be carried on in order to prevent relapses of ulcerative stomatitis.

PARASITIC STOMATITIS.

(*Thrush; Stomatitis Mycosa.*)

Definition.—A specific, contagious fungous disease, characterized by the rapid formation upon the oral mucous surfaces of small, whitish, soft, and lightly adherent spots or flakes, tending to coalesce and spread throughout the entire buccal cavity.

Etiology.—*Predisposing causes* are—infancy with its concomitant disorders of the gastro-intestinal tract (especially when unhygienic surroundings prevail), also congenital syphilis, tuberculosis, and the exanthemata. The disease may attack adults and complicate the typhoid and cachectic states, as in the final stages of low fevers, carcinoma, chronic tuberculosis, and diabetes. The growth of thrush-patches is due, specifically, to the *saccharomyces albicans*, though micrococci have also been found. It is a characteristic of this fungus to develop from round or oval spores in the formation of long-branching mycelium filaments, from the ends of which a multiplication of ovoid torulæ-cells takes place by the process of simple budding. These mycelia exhibit a tendency to penetrate the deeper layers of the mucosa of the mouth and also into the mucous glands (Starr). Since the growth of this organism requires both an altered condition of the mucous membrane and an acid medium, the primary or exciting cause of thrush is to be found in whatever produces such a favorable nidus. Most important in this connection is uncleanliness, particularly in the case of poorly-nourished and bottle-fed children. The development of catarrhal stomatitis and the acid fermentation of remnants of food (especially of saccharine substances), which prevent the nutrition of the mucosa and acidify the normally alkaline oral secretions, are common causes of thrush. The further growth of the fungous patches also contributes to the acid state of the already abnormal buccal fluids. The fact that the spores of thrush may be transferred to other cases by bottle-tips, spoons, and ill-kept feeding-bottles is well recognized as an explanation for the occasional endemic character of the malady.

Symptoms.—Any marked local symptoms are due rather to the coexisting stomatitis than to the thrush itself (Allchin). There will be some *soreness*, *heat*, *persistent dryness*, and *lividity* of the mucous membrane. Thrush-spots, slightly raised above the surface, begin to appear on the tongue, and grow into patches that may coalesce and spread to the cheeks, lips, and hard palate: they may even invade the tonsils, pharynx, and esophagus, and, rarely, the true vocal cords, the stomach, and cecum (Parrot). At first pearly-white in color, the curd-like flakes may become yellow and even brown, owing to slight hemorrhages caused by the irritation. Though early adherent, in a few days they become loose, and when brushed off leave a smooth surface; when complicating some serious gastro-intestinal disease or dyscrasia, however, their attachment is deeper, and the deposit may sometimes appear in successive crops. A *microscopic examination* of the thrush-patches shows interlacing, irregular, and branched mycelial threads, spores, occasional bacilli, and leptothrix-filaments imbedded in a mass of granular débris and fetid particles. The buccal fluids are acid in reaction. The *general symptoms* depend upon the associated disease, and are usually those of wasting, artificially-nourished children having digestive troubles or a constitutional taint.

Diagnosis.—This may be accurately made upon the discovery of the fungus by microscopic examination. Only very rarely are portions of the thrush-organism found in the false membrane of *diphtheritic stomatitis*. *Milk curds* may be readily removed, and are not necessarily associated with the stomatitis accompanying thrush or the grave systemic states. The important point of differential diagnosis arises in the case of *aphthæ*. The following table will express the main points:

Parasitic Stomatitis (Thrush).	Aphthous Stomatitis.
Dryness of the mouth.	Salivation.
Whitish, raised spots or patches with no red areola; these are easily removed, leaving no ulcer and causing no bleeding.	An ulcer with a yellowish-white, depressed base, surrounded by a red areola. The base is removed with difficulty by forceps, and bleeding results.
Spots are numerous.	Usually few in number and discrete.
Begins in the form of minute spots.	Not so; ulcers appear, preceded by the formation of herpetic vesicles.
Ulcers not painful. Discomfort depends on the associated stomatitis.	Ulcers exquisitely tender.
The characteristic thrush-fungus is always detectable with the microscope.	No specific micro-organism determined, though probably present.

Prognosis.—This is favorable as regards the thrush alone, but, occurring in marantic children and cachectic adults, its appearance is of grave significance, and may portend a speedy death.

Treatment.—Prophylaxis is of great moment, since it is much easier to keep the mouth clean and the secretions normal, and to attend to proper food, and thus avoid creating a soil for the growth of the vegetable parasite, than it is to prevent absolutely the entrance of thrush-spores. Efforts directed toward preventing acidity are especially indicated. This is to be done by the use of mild alkaline mouth-washes, as soda-water and lime-water. The dietary should be carefully looked after, and should exclude sugars and all starchy food; the addition of lime-water to the milk (about one part to four) is a desirable precaution to take, particularly with children. Cleansing the feeding-apparatus and the mouth after each feeding is essential, both in the prevention of the formation, and in decreasing the further growth, of thrush when present. The local treatment consists in the use of alkaline and antiseptic applications, preferably by means of the spray. Solutions of boric acid or sodium hyposulphite (ʒj—4.0—of either to ℥j—32.0—of water, with the addition of a little glycerin), potassium permanganate, or hydrogen peroxid, are useful. Syrupy excipients are to be excluded. Potassium chlorate may exert a beneficial effect in those cases in which stomatitis is associated, as may also pencilling with a solution of silver nitrate. Concetti[1] urges the use of a 3 to 5 per cent. solution of silver nitrate instead of the weaker strength usually employed. The use of the galvano-cautery is often serviceable.

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[1] *Rev. mens. des Mal. de l'Enfance*, July, 1899.

When esophageal obstruction exists it may be necessary to gently force a rubber tube through the mass of thrush-deposit in order to give nourishment (Forchheimer).

Medicinal treatment embraces the administration of nourishing and easily digestible food, occasional stimulation, and the correction of gastro-intestinal disorders. Attention must also be paid to the primary affections to which the thrush is superadded. Iron, cod-liver oil, and acid and bitter tonics in palatable form are usually indicated in debilitated subjects, along with general hygienic measures. The internal use of small doses, frequently repeated, of calomel or mercuric chlorid may also be tried for a possible specific effect in combating thrush.

LA PERLÈCHE.

This contagious disease is confined to the angles of the mouth. It was first described in 1886 as prevalent among the children of Limousin in France by Lemaistre. It was found that the drinking-water in that locality contained cocci similar to the spherobacteria that infested the epithelial thickenings, and that these were probably conveyed to human beings by drinking-vessels. Little elevations and fissures, said to resemble those of congenital syphilis, were seen around the oral angles. The latter were the seat of smarting pain, particularly on opening the mouth suddenly or too far, and caused the patient to lick (*perlicher*) them constantly. The disease seemed to be entirely local, and lasted from two to three weeks. Alum and copper-sulphate solutions were most useful.

GANGRENOUS STOMATITIS.

(*Noma; Cancrum Oris.*)

Definition.—A rapidly-spreading gangrenous affection of the cheek and gums, of rare occurrence, usually asymmetric, and ending fatally in most cases.

Pathology.—In addition to the necrotic changes in the cheeks, the process may extend to the jaws and lips. The blood-vessels contain thrombi, thus preventing hemorrhage from the sphacelus. The submaxillary and cervical glands may be slightly enlarged and soft. Blood-changes of an uncertain character have been noted. Hemorrhagic infarctions, aspiration broncho-pneumonia, or gangrene by inhalation of gangrenous particles or metastasis, may be met in the lungs. Wharton has described an associated membranous form of colitis, and a metastatic infiltration of the cardiac muscle and purulent pericarditis may also be seen *post-mortem*. Klementorosky met with a peculiar and fatal form of gangrene limited to the gums of babes and occurring a few days after birth.

Etiology.—*Predisposing Causes.*—This uncommon affection attacks girls more frequently than boys, usually between the ages of two and five years; it appears to be endemic in low, moist countries, as Holland, though apparently it is not contagious. Sickly and ill-nourished children suffering from the effects of overcrowding and previous disease are especially liable to noma. Most often, however, it is secondary to measles; it may also follow scarlet fever, typhoid, small-pox, or less frequently pertussis. The causative influence of mercurialization and ulcerative stomatitis has been overrated.

Bacteriology.—Freymuth holds that noma is an infectious process without, however, a specific microörganism in all cases. Guzzetti found pseudo-diphtheritic bacilli together with staphylococci and streptococci. Noma is probably due to a double infection (Foote).

Symptoms.—The mucous membrane of one cheek, near the corner of the mouth, is usually first affected, a *dark, ragged, sloughing ulcer* appearing and spreading insidiously for two or three days before the substance of the cheek is involved. A *hard* and *sensitive nodule* may then be felt by grasping the cheek between the thumb and finger on placing the one within the mouth over the ulcer and the other outside. Brawny induration of the skin over this nodule soon becomes manifest, and then there appear collateral edema and an unctuous-looking, deeply livid, gangrenous spot, soon becoming bullous and leaving a black eschar. Perforation of the cheek may occur on the third day, though usually not until a week has passed. There is an *ichorous discharge* of shreds of gangrenous tissue from the unhealthy wound. The *fetor* of the breath is almost intolerable and characteristically gangrenous. The necrosis may extend over one-half the face of the side affected, and may involve the gums and jaws, but seldom does it attack the opposite side of the face. The *general symptoms* of such a grave malady may be slight at a very early period, but with the formation of the eschar they become rapidly severe and typhoid in type. Great prostration, delirium, pyrexia (104° F.—40° C.), diarrhea, and edema of the feet are common. The course rarely extends beyond two weeks.

Complications.—Septic lobular pneumonia may occur from aspiration of gangrenous particles; colitis and gangrene of the genitalia in females (*noma pudendæ*) are also seen. In those very rare cases that recover granulations form, the gangrenous edges become clean, and cicatrization, with great disfigurement of the face and even restricted jaw-motion, is then apt to follow.

Diagnosis.—The disease when fully established is easily diagnosed by its characteristic origin, the gangrenous ulcer-nodule, the eschar-formation, and perforation, associated with a previous history of measles or other acute infectious fever of childhood. The offensive fetid odor and severe constitutional depression are also of great value.

Differential Diagnosis.—From *anthrax* it differs in that the latter affection is more common in adults, with a history of contagion, and in the fact that malignant pustule starts on the exterior of the cheek, and perhaps in a previous abrasion in the skin. The discovery of the bacillus anthracis in the blood and discharges is conclusive. *Ulcerative stomatitis* of a severe and neglected type may be confounded with cancrum oris, but in the former the destruction of tissue is mainly of the gums and alveoli, the cheeks being simply ulcerated and no extensive sloughing taking place; the breath, though fetid, is not gangrenous, and the oral discharge, though sometimes bloody, is not mixed with shreds of gangrenous tissue (Starr). Finally, the course of ulcerative stomatitis is less severe, a fatal termination being extremely rare.

Prognosis.—Noma is seldom recovered from, the mortality being about 80 to 90 per cent. (Bogel). When recovery does take place the development of ectropion, facial deformity, and local disability, with a protracted convalescence, render life burdensome.

Treatment.—This embraces the prevention of gangrenous stomatitis by means of a proper management of the diseases that are known to cause it; careful hygiene and the avoidance of mercurialization will also be of undoubted use. The primary indication in the *local treatment* is the arrest of the gangrenous process, thus causing, if possible, a healthy reaction on the part of the surrounding tissues. All dead sloughs should be cut away before using escharotics, and with this end in view some recommend the prompt application of strong caustics, as fuming nitric acid, the acid nitrate of mercury, solid zinc chlorid, silver nitrate, carbolic acid, a concentrated solution of perchlorid of iron, Vienna paste, and the actual cautery. For the protection of the healthy parts and for efficiency the Paquelin or the galvanic cautery is probably best. Anesthesia is requisite for such strong measures. Milder applications, however, seem to be quite adequate in some cases. Thus, bismuth subnitrate, potassium chlorate, and aristol, or the following formula by Dr. Coates, may be tried:

℞. Cupri sulph., ʒij (8.0);
Pulv. cinchonæ, ℥ss (16.0);
Aquæ, q. s. ad f℥iv (128.0).—M.

It is essential, for the prevention of septic infection to ensure cleanliness of the wound and of the mouth, and to promote the separation of the sloughs. To effect the former we employ mild antiseptic washes of carbolic acid, hydrogen peroxid, Labarraque's solution, potassium permanganate, etc.; for the latter and for the diminution of the fetor, antiseptic charcoal poultices containing boric or salicylic acid are useful. Mild antiseptic and astringent lotions of boric acid, zinc sulphate (gr. ij to ℥j—0.129 to 32.0), or balsamic ointments with vaselin, may aid in healing the granulating surfaces in favorable cases. The internal treatment must be directed toward sustaining the strength of the patient by the administration of the most nourishing food, stimulants, and tonics. Rectal feeding may be necessary. Plastic operations may be needful after recovery to mitigate oral disabilities or facial deformities resulting from cicatricial contractions. W. C. Cahall has successfully treated a case of noma with anti-streptococcus serum.

MERCURIAL STOMATITIS.

(*Mercurial Ptyalism.*)

Definition.—An inflammation of the mouth and salivary glands, caused by the excessive use of mercury; it is rarely seen as a result of the therapeutic use of other drugs.

Etiology.—Predisposing causes are dyscrasia and occupation, mainly. The peculiar individual susceptibility of these subjects to dyscrasia will not permit the use of even minimum doses of mercury without serious and almost immediate symptoms of ptyalism. This is also seen in barometer-makers, mirror-silverers, chemists, and others who handle mercury in their daily work. The exciting cause of ptyalism is the ingestion, inhalation, or cutaneous absorption of mercury.

Symptoms.—A *metallic taste* in the mouth is first noticed by the patient. Soon the *gums* become "touched"—*i. e.* red, swollen, tender to the touch, and sore during the act of mastication. A marked secretion and *flow of saliva*, with a *fetid breath* and swollen tongue, follow. Very rarely in this disease the affection passes into an *ulcerative stomatitis*, and causes loosening of the teeth and necrosis of the maxilla. *General symptoms*, as constitutional depression, anorexia, diarrhea, mental anxiety, and nervousness, may supervene.

The recognition of the foregoing causal factors—predisposing and exciting—renders the *diagnosis* easy. The *prognosis* is favorable, and, although the local symptoms may be harassing, recovery is attainable within a few weeks as a rule.

Treatment.—The toxic action of mercury in the production of ptyalism can be avoided by a knowledge of individual susceptibility and by the local and internal use of potassium chlorate. Upon the first appearance of the symptoms there must be a prompt withdrawal of the mercurial influence, and a change of occupation if that be the predisposing cause. Locally, soothing, alkaline, and mildly antiseptic mouthwashes, as in the treatment of catarrhal stomatitis, may be all that is necessary. For the fetid breath solutions of boric acid or potassium chlorate may be used. Ulcers may be brushed with silver-nitrate solution. The internal treatment should be directed toward keeping the bowels soluble; in addition, alkaline mineral waters may be used, and in severe cases potassium chlorate in 5- to 10-grain (0.324–0.648) doses. Atropin (gr. $\frac{1}{100}$—0.0006) and opium have been recommended to decrease the excessive salivary secretion and to allay pain, and hot baths will aid the treatment materially. In severe cases the resulting debility and anemia should be met by the use of highly nourishing liquid foods and by tonics.

Osler points out that the condition of the teeth known as *erosion*, which sometimes follows infantile stomatitis, and especially the mercurial form, is to be discriminated from the deformed teeth of congenital syphilis. In the former the first permanent molars, and then the incisors, are observed to have small pits or discolored and eroded spots, due to a morbid deficiency in enamel-formation. The notched and irregular teeth of hereditary syphilis in children (Hutchinson) are sufficiently distinctive.

II. DISEASES OF THE TONGUE.

GLOSSITIS.

ACUTE GLOSSITIS.

(*Glossitis Acuta.*)

Definition.—An acute parenchymatous inflammation of the tongue, sometimes ending in abscess.

Etiology.—Predisposing causes are supposed to be an impaired general health and exposure to cold, humid weather. The exciting causes are most frequently the stings and bites of insects, or burns, scalds, and the action of corrosives. I believe that many cases follow slight injuries to the tongue that allow of the introduction of inflammatory poisons or microbes. A. J. Hall describes a case of membranous glossitis complicating acute nephritis.

Symptoms.—These come on *rapidly* and with more or less local severity and danger. The *tongue* becomes much swollen, and may even protrude beyond the lips. It is very *tender* and *painful*, and coated with a thick, soft yellowish-white fur, and it may also be dry, cracked, and ulcerated. *Catarrhal stomatitis* is often associated, salivation is usually profuse, and talking, swallowing, and even breathing are rendered difficult and distressing. *Dyspnea*, even to suffocation, may be imminent. The cervical and sublingual glands may be swollen, moderate *fever* is always present, and the obstruction to breathing and administration of nutriment may assume a dangerous aspect.

The inflammation reaches its height in about three or four days, tending to subside almost entirely about the seventh day. Not rarely the inflammatory infiltration passes into suppuration with the formation of a circumscribed abscess of variable size in one-half of the tongue; fluctuation may not, however, be obtainable, spontaneous rupture being sometimes the first indication of abscess. The **prognosis** is favorable, except that serious obstruction is likely to remain.

Treatment.—When the case is seen quite early and during the congestive stage, the topical use of ice, allowed to slowly dissolve in the mouth, is serviceable. Mucilaginous mouth-washes, containing some mild antiseptic, as sodium borate with sodium bicarbonate (gr. v–xx to ℥j—0.324–1.296 to 32.0), should also be employed. A brisk saline purge, given early, will aid in reducing the inflammation, and should the tongue become alarmingly swollen, deep scarification and the use of half a dozen leeches between the hyoid bone and the jaw-angles may be of decided service. Steam-atomization, medicated with the compound tincture of benzoin or ammonium chlorid (ʒj to ℥j—4.0 to 32.0), favors resolution (Cohen). Abscesses must be incised and washed out with antiseptic solutions. Tracheotomy is rarely called for to relieve the dyspnea. Rectal alimentation with predigested foods may be necessary, and during convalescence ferruginous tonics in glycerin and bland foods should be continued for some time, in order to prevent chronic inflammation and thickening. Any local source of irritation, as from carious or sharp teeth, should be removed.

CHRONIC SUPERFICIAL GLOSSITIS.

Definition.—A chronic inflammation of the mucosa of the tongue.

Etiology.—This disease is often preceded by several acute attacks, the habitual use of tobacco, both in smoking and chewing, and of strong spirituous liquors being mainly productive of the original affection. The frequent use of irritating foods is also a prominent factor in some instances.

Symptoms.—The surface of the tongue is continually sensitive and more or less reddened. Often there are seen ovoid patches of various size, smooth and shiny, on account of the loss of papillæ, and separated by furrows that extend to the depth of the mucosa itself. The tongue may also be slightly furrowed in intervening spaces, especially at the base. The general health is somewhat deteriorated.

Diagnosis.—This rests upon the history of the case and upon the results of examination of the organ.

The **prognosis** is favorable as to alleviation, but guarded as to cure.

Treatment.—The blandest dietary must be insisted on, as well as absolute abstention from the causal irritants, exacerbations being prone to occur. The local use of demulcents and of mildly alkaline and antiseptic lotions, such as Seiler's tablets in solution, and of solutions of chromic acid or silver nitrate (gr. v–x to ℥j—0.324–0.648 to 32.0) in glycerin or honey, applied once or twice daily by gentle brushing, is to be recommended. General tonics and the avoidance of irritating drinks will be indicated.

GLOSSITIS DESICCANS.

A rare disease, chronic in nature and of unknown causation. It is characterized by "the gradual development upon the surface of the tongue of a number of deep fissures and indentations, giving the organ an uneven and ragged look. The pain is due to the frequent presence of excoriations and ulcers in these fissures" (Strümpell). The *prognosis* of the affection is favorable as regards any danger. The *treatment* is hygienic, consisting of cleanliness of the mouth and the use of disinfectant mouth-lotions, together with the topical use of alterative or astringent applications, as silver nitrate or chromic acid, to any ulceration.

LINGUAL PSORIASIS (TYLOSIS LINGUÆ).

In this disease there are small regular areas of hyperplasia of the glossal epithelium, eventually causing a map-like appearance of the surface of the tongue—"lingua geographica." The trouble is obscure in its etiology and persists for years. Seldom is there any discomfort associated, although mental anxiety or hypochondriasis may develop.

LEUKOPLAKIA ORIS (BUCCAL PSORIASIS).

In this affection the mucous membrane of the mouth and tongue may be involved. On the lateral borders of the tongue white or bluish-white scar-like spots or patches, often slightly notched, make their appearance. Some of these pass away to be replaced by others, and the affection progresses despite all attempts to cure it. The true *cause* is unknown, but it has been suggested that some irritant, as the use of a pipe, may account for the condition. The malady has, however, been seen in women. A syphilitic taint is said to especially predispose to the disease (Strümpell). The affection must be carefully diagnosed from the oral manifestations of syphilis, if for no other reason than to relieve the mind of a morbidly anxious patient. Excepting some pain connected with possible ulceration, there are no annoying *symptoms*, and the *treatment* suggested for glossitis desiccans is appropriate. Kyle touches the white patches daily with pure tincture of iodin.

In children a similar tongue-affection has been named "wandering rash." The patches are circinate and enlarge peripherally, forming rings of epithelial hyperplasia, within which is a red, glossy center "devoid of filiform papillæ, though the fungiform remain" (Allchin).

ANGINA LUDOVICI.

(*Ludwig's Angina.*)

Definition.—A rare acute phlegmonous inflammation of the floor of the mouth.

Etiology.—The condition may be idiopathic, but more often it is secondary to specific infectious diseases (scarlet fever, diphtheria). Undoubtedly it is directly caused by a streptococcus and by an extension of the infection from adjacent glands (Osler). It may result from trauma.

Symptoms.—These are *intense* at the *outset*, and begin with swelling in the region of the submaxillary gland, with a rapid involvement of the cellular tissue of the floor of the mouth as well as of the anterior portion of the neck. *Pain* is marked, and this, with the *acute swelling*, renders articulation, mastication, and deglutition extremely difficult. Compression or edema of the larynx may often cause dangerous dyspnea. The *constitutional disturbance* is usually febrile, and may either approach the typhoid type or may be septic. The condition generally terminates either in abscess or extensive sloughing (*cynanche gangrœnosa*), and only rarely does resolution take place.

The **diagnosis** is easily made when complicating a specific fever.

The **prognosis** is always guarded, since death sometimes occurs. Relapses are likewise apt to follow in weakly and strumous subjects.

Treatment.—The most that can be done is to sustain the strength of the patient and secure prompt surgical interference when the process has reached the point of beginning suppuration or gangrene. Tracheotomy may be demanded if asphyxia threatens life.

III. DISEASES OF THE SALIVARY GLANDS.

HYPERSECRETION.

(*Ptyalism.*)

Definition.—An abnormal increase in the secretion of saliva.

Etiology.—Salivation as an idiopathic affection is rare, and as such is considered to be a neurosis. Thus, it has been seen in emotional children of from two to eight years of age, though apparently in perfect health. According to Bohn, the secretion in these cases is mostly increased during active exercise, is reduced on lying down, and absent during sleep. Spontaneous recovery takes place in a few years. As a deuteropathic disease ptyalism may be the result of oral disease (*e. g.* noma, ulcerative stomatitis), and also of gastro-enteric, pancreatic, uterine (as gestation), centric (as diseases or tumors of the medulla or of the facial nerve), toxic, systemic (as small-pox, the use of mercury, iodids, pilocarpin, tobacco), and hydrophobic irritation and disease.

Diagnosis.—It should be pointed out that a failure in swallowing the normal quantity of saliva may cause dribbling from the mouth and simulate true hypersecretion.

The **prognosis** is favorable in itself, but dependent on the cause.

Treatment.—The causes are to be removed and the general health toned up. For stomatitic salivation potassium chlorate is first in rank as an internal and local remedy. Iron and arsenic are valuable in neurotic cases, and the bromids or hyoscin may be of supplemental use. Atropin (gr. $\frac{1}{200}$ to $\frac{1}{100}$—0.0003 to 0.0006) and belladonna are almost uniformly successful in idiopathic as well as in central ptyalism.

XEROSTOMA.

(*Aptyalism; "Dry Mouth."*)

Definition.—A morbid arrest of the salivary and buccal secretions.

Etiology.—The disease is probably due to an affection of the nerve-supply of all the glands of the mouth (Harris). It may follow sudden mental phenomena as a temporary condition. A. J. Hall collected 39 cases, of which 32 occurred in females. In most of the cases the causes were unknown. Not uncommonly xerostoma is an effect of the febrile state, of mouth-breathing (due to nasal obstruction), and of diabetes.

Symptoms.—Apart from the sensation of dryness, mastication, deglutition, and articulation are difficult. The *local appearances* show a glazed, shiny, red, and sometimes cracked condition of the tongue and labial and palatine mucous membrane. With dryness of the mouth the teeth may become diseased and crumble.

The **diagnosis** is made on inspection, the **prognosis** depending on the removability of the cause, and rightfully being guarded on account of the frequent obstinacy of the trouble.

Treatment.—Attention to the systemic condition is requisite. Small doses of potassium iodid and pilocarpin (gr. $\frac{1}{20}$—0.003) in gelatin lamellæ or in lozenge form, allowed to dissolve in the mouth with the aid of a sip of water, have been productive of relief. In cases of centric origin the galvanic current should be tried.

Glassblowers' Mouth.—This condition is found among glassblowers and also among musicians. It occurs in about 2.5 per cent. of all glassblowers. Scheele,[1] who reports two cases, describes the condition as a hernial-outpocketing of the muscles of the cheeks. The epithelium of the mucous membranes shows the so-called *plaques opalines.* It is often combined with a distention of Steno's duct. In addition to a ballooning out of the buccal mucosa, there is likely to be a disturbance of hearing and cramp-like contraction of the cheek. The parotids may be emphysematous and crepitate on palpation.

SYMPTOMATIC PAROTITIS.

(*Parotid Bubo.*)

Definition.—A secondary inflammation of the parotid gland, generally due to septic infection and tending to suppuration.

Etiology.—Not being a primary affection, the causes giving rise to it may be mentioned as follows: (*a*) Acute infectious fevers, as typhoid, typhus, pneumonitis, pyemia, erysipelas; (*b*) Injury or disease of the abdomen or pelvis (Stephen Paget), especially when associated with the genito-urinary tract, as mild traumatisms or derangement of the testes or ovaries, the use of a pessary, or even menstruation or pregnancy; gastric ulcer may be accompanied by it; (*c*) Peripheral neuritis with facial paralysis (Gowers).

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[1] *Berlin. klin. Wochenschrift,* Mar. 12, 1900.

Most of the cases are probably septic and indicative of an unfavorable course in the progress of the associated disease, and especially of the fevers mentioned. The *symptoms, diagnosis*, and *treatment* of the parotitis itself fall more properly under the scope of surgery.

Chronic Parotitis.—Mikulicz first described this condition and reported a case in which symmetric enlargement of the lachrymal, and subsequently of the salivary, glands occurred. Kümmel and Osler have also recorded cases. It may be caused by lead or mercury and may be secondary to mumps, inflammation of the throat, and chronic Bright's disease. The condition may be painless.

IV. DISEASES OF THE TONSILS.

ACUTE TONSILLITIS.

Definition.—An acute inflammation of the tonsil or tonsils, affecting either the mucous membrane, the follicles, or the parenchyma, and ending either in resolution, suppuration, or chronic enlargement.

Pathology.—In the *superficial variety* of acute tonsillitis the mucosa is simply red, swollen, and sometimes covered with a thin, soft exudate of muco-pus. The tonsil itself may also be swollen. In *follicular tonsillitis* the lacunæ become filled with a cheesy exudate which often protrudes from the tonsillar crypts; epithelial and pus-cells, cellular débris, and occasional cholesterin-crystals are found in these cheesy masses. In older, darker-hued masses an offensive odor is given off, and numerous micrococci and bacteria are found. In adults, calcareous infiltration of the cheesy little masses may be met with. *Parenchymatous tonsillitis* is shown by a greater enlargement of the tonsil, due to a marked infiltration of all the tissues. Suppuration in the tonsil is frequent, the follicles usually bursting and uniting in abscess-formation. Pus may burrow into the cellular tissue surrounding the tonsil, and find its way even down to the clavicle. The *herpetic* or *ulcero-membranous form* of tonsillitis described by Rilliet and Barthez, DaCosta, and others, in which an eruption of herpetic vesicles on the tonsils is followed by their rupture and the formation of a lightly adherent, membranous covering is rarely met with. In *necrotic tonsillitis* (Strümpell) a grayish-white adherent necrotic membrane is observed, that is limited by the inflamed membrane surrounding the mucosa covering the tonsils, which are moderately swollen. A dirty ulcer often remains after the slough separates.

Etiology.—*Predisposing causes* are age, sex, temperament, and atmospheric conditions. The disease is most common in youth and in early adult life. Boys and young men appear to be attacked more often than the opposite sex. Tonsillitis is most prevalent during the spring season. An individual susceptibility is most distinct in lymphatic and strumous constitutions. It is aggravated by, or tends to recur especially in, the rheumatic diathesis. The proportion of cases in which tonsillitis precedes rheumatism is probably over 30 per cent. It is certain also that one attack of acute tonsillitis predisposes to subsequent ones, particularly when the first attack has left some enlargement of the tonsils. Sudden and extreme climatic changes, and the special conditions seen in connection with outbreaks of scarlet fever, measles, and diphtheria, predispose to the disease.

The *exciting causes* of acute tonsillitis are most commonly the following: (*a*) exposure to cold and dampness, or talking in a cold, moist atmosphere; (*b*) exposure and talking in an overheated atmosphere vitiated with smoke or other irritating vapors or gases; (*c*) bad drainage, sewer-gases; (*d*) specific infectious fevers, as scarlatina, measles, and erysipelas; (*e*) irritation from hard and sharp foreign bodies or chemical irritants; (*f*) the presence of microbes (streptococci, staphylococci).

Clinical Symptoms.—Three principal varieties of acute tonsillitis occur clinically, the symptoms of which will be described separately.

(*a*) **Acute Catarrhal or Superficial Tonsillitis.**—This form is often associated with acute pharyngitis. The earliest *local symptoms* are pain and difficulty in swallowing, the former often becoming quite acute and radiating to the ear and lymphatics at the angle of the jaw, where tenderness on pressure may also be elicited. In speaking a nasal twang is often noticed. During the laborious act of swallowing the sensation of a lump in the throat, especially when the mouth is dry, is commonly complained of. Simple stomatitis may be associated, and rarely there is a slight cough with the painful expectoration of a sticky mucus which accumulates in the throat (Browne). There may be salivation, with fetor of the breath. *Inspection* shows the tonsil to be red and swollen. Though dry and glazed at first, the surfaces soon become covered with a thin exudate of muco-pus, which is easily detached by brushing, gargling, or "hawking" the throat. There is usually some accompanying redness, and also a *tumefaction* of the uvula and faucial pillars. The *constitutional symptoms* of simple erythematous tonsillitis at the outset are mildly febrile. The attacks usually come on rapidly, and last but a few days, subsidence taking place rapidly also. *Otitis media* may follow the extension of the tonsillar inflammation, and *acute pharyngitis* is a common complication.

(*b*) **Acute Lacunar or Follicular Tonsillitis.**—In this form, which is quite common in children, not only the mucous membrane lining the crypts is inflamed, but that covering the surface of the tonsils also, giving rise to more or less associated *catarrhal tonsillitis*. The *local subjective symptoms* in this disease are pain, tenderness, and difficult deglutition, the counterparts of those of the preceding form. The tonsils are seen to be covered with small, slightly prominent, whitish-yellow spots or patches of a characteristic creamy exudate corresponding to the position of the crypts and numbering from two to eight or ten or more. These little masses or plugs may be pressed out of the follicles with a spatula. A predominance of pus-cocci and cells may rarely forerun the further formation of little follicular abscesses, and even of slight erosions and ulceration of the mouths of the lacunæ. Unlike simple catarrhal tonsillitis—at least in so far as simultaneous involvement is concerned (Cohen)—both tonsils are usually affected in this trouble, though one to a greater degree than the other. The whole tonsil is considerably swollen, and in severe cases the cervical lymph-glands also. The *constitutional symptoms* of follicular tonsillitis may be quite severe. The disease may be ushered in with a pronounced chill, headache, aching of the back and limbs, marked anorexia, a heavy deposit of urates, and insomnia, along with a rapid rise in the temperature to 103° or 104° F. (39.4°-40° C.)—in children as high as 105° F. (40.5° C.). The general depression may be so great as to simulate adynamia. Though sudden in its onset and rapid and often intensely acute in its progress, the disease seldom lasts more than five or eight days. Follicular abscesses complicate the case, while chronic swelling of the tonsils, desiccation, and bacterial degeneration of the lacunar masses may be sequelæ. Packard has reported five cases of endocarditis following acute angina. Pericarditis and pleuritis also rarely occur as complications. Albuminuria with casts may appear in the course of tonsillitis. Skin-lesions, particularly erythema nodosum, may also accompany angina. The exudate may become calcified, and may be expectorated as concretions or chalk-plugs.

(*c*) **Acute Parenchymatous Tonsillitis** (*Tonsillar Abscess* or *Quinsy*).—In this form of tonsillitis, which occurs most often during adolescence and early adult life, the symptoms reach the most pronounced and severe types. The stroma is inflamed and the tendency is toward suppuration.

Local Symptoms.—Complaint is first made of dryness of the throat, with painful and difficult deglutition. The pain is a prominent subjective sign, and may be referred to one or both ears according as one or both tonsils are inflamed. The secretion of a viscid mucus soon takes place, and as the tonsillar swelling increases, the husky voice of sore-throat and difficult articulation supervene; in cases of aggravated swelling dyspnea may often appear later. On examining the tonsils they are found to be greatly enlarged, deeply reddened, and edematous. The surrounding soft parts, the faucial arches, pillars, and the uvula, also manifest a deep congestion. The swollen tonsils may cause a bulging forward of the anterior pillars of the fauces, and push the often elongated and edematous (jelly-like) uvula to one side; or if both tonsils are affected, they may grasp or push it forward. In severe cases the tonsils may meet in the median line. They are firm to the touch. Patches showing follicular tonsillitis are not infrequently seen associated with the trouble. The submaxillary glands may be engorged, and opening the mouth is often performed with difficulty; it is usually only partial, on account of the fixation of the jaw.

In a few days, perhaps, softening and fluctuation may be detected in the tonsils, and spontaneous rupture and discharge of the pus may occur, with almost instant relief to the patient. Suppuration and tonsillar abscess are not always the termination, however, of parenchymatous inflammation, resolution sometimes taking place in the milder cases. The abscess may open in one or more places, and rupture during sleep may rarely cause suffocation by the entrance of pus into the larynx. The tonsil may regain its original size in a few days after the discharge of pus, and all the symptoms subside. The *constitutional phenomena* of parenchymatous tonsillitis are usually severe from the start, even in children, and more so than in the follicular form (Mackenzie). The temperature rises to 104° or 105° F. (40° or 40.5° C.), and the pulse-beats may reach 130 per minute. The usual symptoms accompanying inflammatory fever are marked. There may be delirium, and the symptoms generally increase until the abscess bursts or is opened, when the constitutional as well as the local disturbance rapidly abates.

Course, Duration, and Terminations.—Though often severely acute in its course, quinsy seldom goes on to rupture in children, usually ending in resolution in from three to five days. If both tonsils are inflamed, only one suppurates as a rule, or but one at a time. The duration of an attack ending in tonsillar abscess is about eight or ten days in adults.

Complications and Sequelæ.—The tonsillar suppuration may invade the cellular tissue between the tonsil and the pterygoid muscles; a peritonsillar abscess may then result that may open even above the clavicle. Deep involvement of the tonsil may also cause ulceration into the internal carotid or internal maxillary arteries, and fatal hemorrhage occur, though these accidents are, fortunately, rare. Edema of the larynx is also an infrequent complication. French writers, as Guble, Germain Sée, and others, have reported cases of paralysis of the soft palate and pharynx following inflammatory throat-diseases. On subsidence of the tonsillar inflammation the trouble becomes evident in the difficult swallowing and partial regurgitation of liquids and solids into the nasal passages, and in the nasal intonation of the voice. A frequent sequel, especially in those predisposed by heredity, is chronic enlargement of the tonsils.

(*d*) **Necrotic Tonsillitis.**—This affection is considered by Strümpell to be in some instances entirely distinct from diphtheria in its etiology, although he admits that quite frequently it is simply a mild form of the latter disease, and that often it is impossible to distinguish between the local appearances of the two conditions: these have been referred to under the heading of Morbid Anatomy. The *constitutional disturbances* are severe, especially in children, though they seldom last longer than a week, and are followed by a rapid convalescence. The cervical glands are not swollen to the same extent as in diphtheria. The occurrence later of palatal and pharyngeal *paralysis* in a supposed case of necrotic tonsillitis would point to its true diphtheritic origin.

Diagnosis.—The appearance of the several forms of acute tonsillitis, associated with the clinical history of each case, should enable a ready diagnosis to be made in the majority of cases. A difficulty may, however, arise in discriminating follicular tonsillitis from diphtheria, and apparently transitional forms are not uncommon. The appended table gives the important points of differentiation between these diseases:

FOLLICULAR TONSILLITIS.	DIPHTHERIA.
A soft, pultaceous, yellowish-white deposit occurs in spots or patches situated over the mouth of the follicles, with areas of redness intervening.	A tough, ashy-gray, continuous, and uniform pseudo-membranous deposit covers the tonsils.
The exudate is easily removed, leaving a smooth surface.	Very adherent, and can be torn off in strips only, leaving a bleeding erosion.

Follicular Tonsillitis.	Diphtheria.
The deposit is limited to the tonsils (important).	The pillars of the fauces and uvula are involved as well.
If the creamy deposits unite to form a continuous layer, removal is either not followed by re-formation, or very late.	Removal of the membrane is followed by re-formation within twelve to twenty-four hours.
May have high temperature, but lasting only a day or two. Albuminuria extremely rare, if present at all.	Persistent elevation of the temperature; more or less albuminuria is common.
Cervical lymphatic glands seldom or slightly swollen.	Usually markedly swollen glands.
Complications rare and mild.	Complications frequent and grave.
Bacteriologic test shows no special organism; often, however, streptococci and staphylococci.	Bacteriologic examination shows presence of Klebs-Löffler bacillus.

Cases seen early, with severe constitutional symptoms and red and *swollen tonsils* having no deposit, may give rise to the question whether *simple angina* or *scarlet fever* is to follow. In such cases the latter disease may be excluded by a negative history of exposure to contagion, by the absence of a very high pulse-rate, and by the non-appearance of the scarlatinal eruption. Necrotic tonsillitis may be discriminated from the lacunar variety in the same manner as diphtheria—*i. e.* by its local manifestations, a full description of which has already been given under the heading of Morbid Anatomy.

The **prognosis** is good as regards life, and favorable as regards complete recovery. The occurrence of either fatal hemorrhage or asphyxia in *quinsy* is extremely rare. In debilitated and strumous individuals relapses are prone to occur, and successive acute attacks of tonsillitis tend to cause permanent hypertrophy of the tonsils. In cases of *necrotic tonsillitis*, especially during the earlier periods, the prognosis should always be guarded.

Treatment.—Particularly in the lacunar and necrotic forms of tonsillitis the patient should be kept apart from others as much as possible, since both types appear to be contagious to a certain degree; or, if other persons in the house are subject to a common source of infection —not human—their frequent nearness to a given case only serves to augment their own liability to similar attacks. Individual susceptibility to frequent attacks of sore throat may be lessened by systematic cold bathing of the neck. Constitutional and local rest is a first and constant requisite. Efforts at swallowing and talking should be reduced to a minimum, and in marked cases of follicular or suppurative tonsillitis rest in bed is often sought without direction. Bland nourishing liquids, as milk, broths, and the like, should constitute the only nutriment during the attack.

Medicinal Treatment.—Early in the case a free evacuation of the bowels should be obtained, and small doses of calomel (gr. $\frac{1}{8}$–$\frac{1}{6}$—0.008–0.010, repeated hourly until about gr. 1—0.0648—has been taken), followed by a Seidlitz powder or Rochelle salts in hot water, will be effective in most cases. In severe cases of quinsy relief from the pain is urgently called for, and either a Dover's powder or a hypodermic injection of morphin (gr. $\frac{1}{6}$–$\frac{1}{4}$—0.010–0.016) and atropin (gr. $\frac{1}{100}$—0.0006) will probably suffice for their relief. A high temperature must be combated by small doses of aconite, frequently repeated: this drug has been much used in the follicular tonsillitis of children. Quinin, in solution with dilute sulphuric acid, is also often given.

The administration of sodium salicylate or benzoate, of salol, or of the ammoniated tincture of guaiac in 1-dram (4.0) doses (Sajous), seems to lessen the duration and severity of tonsillitis, and even to cure some cases of the lacunar form within forty-eight hours and without local applications. The tincture of the chlorid of iron in glycerin (4 or 5 drops to the dram—4.0—given every two hours) is regarded by Bosworth as almost specific at the commencement of an attack of acute follicular tonsillitis. During convalescence semi-liquid and soft, light foods may be allowed gradually; and bitter tonics and iron are to be administered if there are depression and anemia. The following is a favorite prescription:

℞. Strychninæ sulph., gr. ss (0.032);
Syr. acaciæ, ℥ss (16.0);
Liq. ferri et ammon. acetat., q. s. ad ℥iij (96.0).—M.
Sig. ʒj (4.0) *t. i. d.*, in water, after meals.

Local Treatment.—If the case is seen early, the use of cold is of great value in giving local relief and in shortening the attack. Ice may be sucked, and flannel dipped in ice-water and wrung out may be applied around the neck, or an ice-bag used. Lozenges of guaiac (gr. ij—0.129) or the ammoniated tincture in 1-dram (4.0) doses in milk, and used as a gargle, are indicated early, and, according to Sajous, seldom fail to control or arrest the inflammation. Equal parts of the tincture of the chlorid of iron, glycerin, and water, applied gently with a camel's-hair brush, have long been used locally on the surfaces of the tonsils, and with marked benefit. Alkaline and mild antiseptic solutions, used as gargles or sprays (preferably the latter), are generally useful. Thus, Dobell's solution, or Seiler's tablets dissolved in water, or borax and thymol, or carbolic acid, or potassium permanganate in weak solution, may be serviceable. Mild counter-irritation at the angle of the jaw by means of iodin or slightly irritating embrocations is helpful.

Early scarification of the tonsils as a depletory measure, and painting with cocain (10 per cent.), I have found useful to bring about resolution.

Astringent sprays containing alum or silver nitrate are often efficacious after a day or two. When the case is first seen and fully developed, the atomization of a warm solution of cocain (4 to 8 per cent.) or lime-water, with the external application of heat by means of poultices, is indicated. Should gargling be possible, nothing is better than hot water or milk. If, in parenchymatous tonsillitis, fluctuation be detected or suppuration be even suspected of commencing, the prompt use of the bistoury (the blade being guarded by wrapping with cotton or adhesive plaster), with the production of free bleeding or the discharge of pus, will give great satisfaction and relief. The patient's head, especially if it be a child, should be tilted forward during the operation, so as to allow most of the blood and pus to pass into the mouth. When incision of the tonsil fails to bring pus, it has been advised to puncture through the anterior pillar, where pus may be formed in the cellular tissue in front of or behind the tonsil.

When the tonsillar enlargement threatens life through suffocation, excision of the tonsils, laryngotomy, tracheotomy, or intubation may have to be performed.

CHRONIC TONSILLITIS.

(*Hypertrophied Tonsils; Adenoid Vegetations.*)

Definition.—Enlargement of the tonsils (faucial and pharyngeal), due to chronic inflammation or hypertrophy, and usually associated with or causing a perverted local and systemic condition.

Pathology.—The faucial tonsils show a true chronic hypertrophy of the lymphoid and fibrous elements. According to the hyperplasia of the latter the organs will be smaller and more indurated. They may be rough on the surface from "distended lacunæ or ruptured follicles" (Berkley Robinson), the latter being in a state of chronic inflammatory thickening, and showing caseous degeneration of their contents. The growths in the vault of the pharynx are adenomatous papillomata; they are either sessile or pedunculated, and are fleshy in appearance and consistence and very vascular. They range in size from a grain of wheat to an almond-kernel (Allen), and project from the pharyngeal vault, lying in the depression posterior to and on a line with the fossa of the Eustachian tube (Rosenmüller's fossa). "Hypertrophy of the pharyngeal adenoid tissue may also be present without great enlargement of the tonsils proper" (Osler). A congestive type of nasal catarrh in adults often accompanies, or is the result of, neglected adenoid growths and hypertrophied tonsils that date from childhood. Chronic pharyngitis is also not infrequently associated.

Etiology.—The *predisposing causes* of chronic hypertrophy of the tonsils are—(*a*) heredity, especially in the scrofulous and syphilitic diatheses; (*b*) age, most frequently between five and fifteen years; (*c*) sex, boys appear to be affected more frequently; (*d*) hygienic surroundings.

The *exciting causes* are usually previous attacks of acute tonsillitis, either simple or that which is symptomatic of diphtheria or scarlatina. According to Harrison Allen, adenoid growths from the normal lymphoid tissue of the vault of the pharynx (pharyngeal tonsils) may be congenital, and are "in some way associated with the canal which is found in early fetal life penetrating the brain-case and uniting the anterior part of the pituitary body to the lining membrane of the pharynx."

Symptoms.—*Local.*—With slight or even moderate tonsillar enlargement there may be few or no symptoms attributable to it. There may be simply an increased secretion of mucus, and a susceptibility to fresh anginal attacks or to severe tonsillar manifestations in diphtheritic or scarlatinal attacks.

The first symptom to attract the attention is the direct effect of nasopharyngeal obstruction—*i. e. oral respiration.* This mouth-breathing is visibly labored and abnormally audible, and is especially marked at night, the child's respiration being noisy, snorting, and irregular. Sleep is disturbed by paroxysms of dyspnea, sometimes due, perhaps, to reflex spasm of the glottis. Nightmare follows as a result of imperfect aëration of the blood which supplies the brain on account of the obstruction to perfect respiration. The act of swallowing is rendered difficult by the faucial obstruction, and is often painful, owing to the superadded acute tonsillar trouble that is so liable to occur in the hypertrophied glands. Indirect results of chronic tonsillar enlargement are a *laryngeal stridor* and a croupy *cough*. Sometimes *asthmatic attacks* coexist, and seem also to be due to the hypertrophy. An excessive secretion of mucus in the pharynx is a common symptom, and causes hawking in subjects past young childhood. The *hearing* is often impaired, and tinnitus aurium is complained of, being the result of pressure of the growths against the orifice of the Eustachian tube or of clogging of the tube with mucus, due to the extension of inflammation from the naso-pharynx. Absolute deafness may result, and the senses of taste and smell are likewise diminished or perverted. *Inspection* of the fauces will show the tonsils bulging as two lumps covered with thick mucus, or the latter may ooze around the uvula from the pharynx. In mouth-breathers of long standing the superior dental arch is narrowed and the hard palate is highly arched. The breath is fetid, owing to the cheesy, inspissated exudate in the tonsillar crypts. In very old cases a tonsillar *calculus* may be felt, and is the result of calcification of the secretion.

The *facial expression* is characteristically stupid and pathetic; the disposition is dull, irritable, and stubborn; the lips are thick, and a vacant stare is in the eyes. *Speech* is slow, phonation nasal in quality, and articulation of the nasal consonants *n* and *m*, *l* and *o*, is changed or muffled. *Stammering* is not rarely associated with tonsillar hypertrophy. The anterior nares may be dilated and present a pinched appearance above their openings.

The prolonged interference with normal respiration gives rise to a peculiar chest-conformation, simulating that of rickets (*chicken-breast*). The ribs are prominent anteriorly, and there is a marked forward angle at the manubrio-gladiolar junction, as well as a grooved depression at the ensiform cartilage. Depressions between the widely-separated ribs exist anteriorly also. Posteriorly, and at the base of the chest in particular, the intercostal spaces are practically absent on account of the closeness of the ribs. The upper part of the chest is very narrow and the shoulder-bones quite prominent On percussion the hepatic area of dulness is diminished on the chest-wall, but increased downward and to the left. The first cardiac sound is weak. On inspiration there is a retraction of the intercostal spaces in the lower and lateral thoracic regions.

The resulting thoracic deformity may express itself principally as an excavation of the lower sternal area (*trüchter brust*). When chronic tonsillar enlargement leads to oft-recurring asthmatic attacks, the chest may become *barrel-shaped*, as in emphysema, at an early period of life.

The *general symptoms* of tonsillar hypertrophy are more marked when the growths exist in the pharyngeal vault alone. Developmental processes in children, such as dentition, and at puberty, particularly when the voice-changes are looked for, are often retarded or perverted. Anemia, headache, especially during study, cardiac palpitation, enuresis, and habit-chorea of the facial muscles, may be associated with general capriciousness, mental dulness, indisposition to intellectual exertion, drowsiness, and sullen irritability. The term *aprosexia* has been given to the loss of power to concentrate the mind for any length of time that is so characteristic of these cases.

Diagnosis.—Inspection of the fauces will reveal enlarged tonsils. It should be borne in mind, however, that the act of gagging often causes the tonsils to rotate forward and inward, making them appear larger really than is the case. Adenoid growths of the pharyngeal vault may exist without tonsillar enlargement, and can be detected by posterior rhinoscopy or by the insertion of the finger into the naso-pharynx; the vegetations may thus be felt blocking the vault.

Differential Diagnosis.—It is important not to attribute the obstructive symptoms to *nasal hypertrophies* or *atresia* or to *malignant growths* in the naso-pharyngeal space. The latter are infrequent at the ages at which chronic tonsillar enlargement of the fauces and pharynx is most apt to occur—*i. e.* early in life. Again, palpation of sarcomatous or carcinomatous growths gives marked differences in consistence, and there are usually spontaneous hemorrhages and local pain in attendance upon these neoplasms. "*Thumb-suckers*" differ from mouth-breathers in that in the former the incisors are inclined forward and cause slight protrusion beneath the upper lip; the dental arch is flat. In mouth-breathers, however, the incisors are vertical or nearly so, or incline so as to overlap each other; the dental arch is high and curved (H. Allen). *Retropharyngeal abscess* may be confounded with tonsillar enlargement, especially in children. But in this disease the attacks of dyspnea, the dysphagia, and the local distress are more marked. Again, in the pharyngeal disease the swelling is in the median line, pushing the soft palate forward perhaps, and on palpation it may give a sense of elasticity or fluctuation to the finger. Slight fever may also be present.

Prognosis.—Tonsillar hypertrophy is not a severe disease as regards life. There is, however, an increased liability to contract colds, recurrences of follicular tonsillitis, attacks of diphtheria, and severe scarlatinal angina. The prognosis in acute respiratory affections associated with chronic tonsillar enlargement is always more or less grave. Adenoid growths, even when neglected, tend to lessen in size after puberty, with a subsidence of local and reflex symptoms. After removal the growths, as a rule, do not return.

Treatment.—The old-fashioned use of astringent applications is probably useless when there is any marked chronic enlargement of the tonsils, and active surgical treatment alone is to be recommended for the condition. The use of absorbents and caustics, either externally or by parenchymatous injection, is, I think, objectionable on account of the necessarily protracted and painful course of treatment.

There are no more satisfactory means of doing radical good in cases of this kind than the galvano-cautery, scarification, and the removal of the tonsils with the tonsillotome, snare, or bistoury. In offensive follicular disease applications of chromic acid may give good results. Adenoid growths may be removed by means of the finger, curet, or forceps.

Constitutional treatment is often necessary in improving the nutrition of the patient. Good food, a change of air, systematic bathing, prudent habits, careful dress, and medicinal tonics and alteratives, as cod-liver oil, iodid of iron, and the hypophosphites, are usually indicated.

V. DISEASES OF THE PHARYNX.

PHARYNGITIS.

ACUTE PHARYNGITIS.

(*Pharyngitis Acuta Simplex.*)

Definition.—An acute catarrhal inflammation of the mucous membrane of the pharynx.

Pathology.—The mucous membrane is congested diffusely or in patches, and there may be an inflammatory exudate in, and a consequent swelling of, the submucosa and the contained glandular structures. The surface of the membrane is more or less coated with a viscid muco-pus.

Etiology.—*Predisposing causes* are—age, it being more frequent in adolescence and young adult life; a depraved constitution; digestive disorders, and a rheumatic, gouty, or scrofulous diathesis. The usual exciting cause is exposure, particularly of certain portions of the body, as the neck and chest, to cold or to sudden changes of temperature and to irritating vapors. An acute naso-pharyngeal catarrh, by bathing the pharyngeal mucosa with its irritating secretions, may set up the trouble. "*Epidemic pharyngitis*" is probably a manifestation of influenza. Acute simple pharyngitis may be a complication of scarlatina, measles, and small-pox (*exanthematous pharyngitis*). Micrococci are present, the streptococci often predominating.

Symptoms.—*Locally*, the affection is ushered in with a feeling of dryness and soreness, especially on swallowing. With the production of the muco-purulent secretion a tickling sensation provokes *hawking* or a slight "*throat cough*" and efforts at exspuition. The catarrhal process may extend to the larynx and cause some hoarseness, or to the Eustachian tube, causing dulness of hearing. Movements of the neck are *painful* and *stiff*, particularly if there is, as is often the case, slight involvement of the lymph-glands. *Inspection* of the throat shows the pharynx, often the posterior pillars of the fauces and the soft palate, and even the anterior pillars and tonsillar surfaces, to be deeply reddened and tumefied; the coursing veins are enlarged, and particles of a yellowish-white secretion appear here and there. Sometimes the pharyngeal follicles become subject to acute inflammation, and appear as elevated, discrete, shiny spots (*herpetic pharyngitis*—Mackenzie).

At the *onset* of this affection there may be chilliness, followed by slight fever, headache, an accelerated pulse, a dry skin, and anorexia. The *pharyngeal symptoms* seldom last more than from three to five days, when resolution takes place, some tenderness of the pharynx, however, remaining for a time.

Diagnosis.—On examination of the throat there should neither be any difficulty in diagnosing the affection nor any likelihood of confounding the affection with simple tonsillitis.

The **prognosis** is always favorable. In weakly patients, however, there is a liability to subsequent attacks.

Treatment.—In the early stages sucking of small pieces of ice does much to allay the congestion and irritability. A spray of cocain or menthol in albolene (2 per cent.) may also be used, followed by a 4 per cent. solution of antipyrin. Eucain may be substituted for cocain (2 per cent. solution), and is preferred by Gibbs and others. Dobell's solution is always to be recommended for its alkaline, sedative, and antiseptic action. Swabbing the pharynx with a silver-nitrate solution (gr. xl to the ounce—2.59 to 32.0) is, according to Sajous, of great benefit.

In well-established cases relief is often obtainable by medicated steam inhalation, as with the compound tincture of benzoin. In rheumatic cases lozenges of guaiac (gr. iij—0.194) are useful. The sipping of hot milk in which sodium bicarbonate has been dissolved is grateful.

The *general treatment* embraces measures directed at the fever and the diathetic condition. A hot foot-bath and a calomel purge, with belladonna, acetanilid, or aconite for the fever and pain, and sodium salicylate (gr. lx–lxxx—4.0–5.1—in the twenty-four hours), may be required. The diet, of course, should either be liquid or semi-solid.

Persons susceptible to repeated attacks must exercise caution in regard to exposure to severe cold and weather-changes, irritating vapors, and the like. Daily cold sponge-baths may be used to harden the skin. Tonic, nutrient treatment is also frequently called for.

MEMBRANOUS PHARYNGITIS.

(*Pharyngitis Crouposa.*)

Definition.—An acute superficial inflammation of the pharyngeal mucosa, characterized by the formation of a whitish false membrane, due usually to the streptococcus.

Etiology.—The principal causes of this form of pharyngitis are exposure of persons in debilitated health to cold or an impure or a septic atmosphere, particularly during epidemics of such diseases as scarlatina.

Symptoms.—The local and general symptoms are those of ordinary sore throat, though of a more severe type.

Diagnosis.—The pseudo-membrane is thin, of a yellowish-white color, and appears in small patches over the pharynx; it is easily detached, and is thus distinguished from diphtheria, with which alone it might be confounded. The presence of small vesicles or ulcers and the absence of grave constitutional disturbances are also features in this affection that serve to differentiate it from diphtheritic pharyngitis.

The **prognosis** is favorable.

Treatment.—Local applications of solutions of hydrogen peroxid or potassium permanganate (gr. x to the ounce—0.648 to 32.0) are very satisfactory. For the painful dysphagia the sedative and soothing remedies suggested for simple acute pharyngitis may be used. Internally, sodium benzoate (gr. v–xv—0.324–0.972) in glycerin, elixir of calisaya, and salol have each been recommended. Tonic treatment is nearly always needed.

CHRONIC PHARYNGITIS.

Definition.—A chronic inflammation of the mucous membrane of the pharynx. It may consist of either a hypertrophic or an atrophic involvement of the follicles, or both processes may coexist.

Varieties.—(*a*) Chronic naso-pharyngeal catarrh; (*b*) chronic hypertrophic pharyngitis or naso-pharyngitis (*pharyngitis sicca*); (*c*) follicular or granular pharyngitis. The last named is probably the result of, and nearly always is associated with, chronic simple (or hypertrophic) pharyngeal (or naso-pharyngeal) catarrh.

Pathology.—The mucous membrane in simple chronic pharyngitis is either reddened, thickened, and viscid (hypertrophic form), or pale, thin, and dry (atrophic form); in both instances dilated and tortuous veins are prominently shown. In the follicular variety the pharyngeal mucous glands are swollen into little red, glistening nodules studding the congested membrane. The enlarged follicles are due to a hyperplasia of lymphoid cells and an accumulation of retained dried-up secretions.

Etiology.—A protracted impairment of the general health, especially in those who over-exert mentally and are of sedentary habits, is a common *predisposing cause* of chronic pharyngitis. Repeated acute attacks may precede the affection. It is most common in adolescent and middle life.

The *exciting causes* are frequent and prolonged over-use and strain of the voice in clergymen, singers, teachers, army-officers, and street-venders; irritation from tobacco-smoke, chemical vapors, and continued exposure to cold air. Among prevailing causes may be mentioned post-nasal adenoids, deviations of the septum, and neoplasms. It may arise from gastric disorders.

Symptoms.—In all varieties of chronic pharyngitis the *local discomfort* is often very slight, and more annoying than painful, except when an exacerbation takes place. It is a particularly uncomfortable condition in those whose occupation requires more or less constant use of the voice. There is a sensation of *dryness* and *tickling* or *burning* in the throat and the desire to clear the throat of sticky mucus by *hawking* or a *short cough.* These symptoms are usually worse on rising in the morning, especially if some unfavorable influence has been exerted during the night previous, the throat being dry and a viscid secretion having collected. Swallowing is seldom interfered with.

If the larynx is somewhat affected by extension of the pharyngeal inflammation, *hoarseness* and a *dry, hacking cough* are produced. After using the voice there is a sense of fatigue, with huskiness and irritability.

The *local appearances* of chronic pharyngitis vary according to the form of the affection present in the case. In chronic catarrh of the pharynx a considerable collection of muco-pus is seen adhering to the mucosa and extending downward from the posterior nares. The senses of hearing and taste may be impaired. The uvula is frequently elongated, and its tip may rest on the base of the tongue. A nasal intonation of the voice is sometimes provoked. The posterior nares as seen by the rhinal mirror are often stopped up by foul secretions or by hypertrophy of the nasal mucous membrane. Headache and attacks of vertigo may occur.

Chronic hypertrophic pharyngitis and follicular pharyngitis ("***clergyman's sore throat***") are commonly associated. The thickened, reddened, pimply, vein-coursed appearance of the mucosa is characteristic. The follicles may be seen sometimes as polypoid elevations, and the pharyngeal tonsil may be found by the finger to be enlarged (Kölliker).

In the dry, *atrophic pharyngitis* that occurs more often in later life, and as a sequel of the simple chronic or follicular variety, a pale, smooth, relaxed, lustrous, and often quite painful membrane is observed.

The ***general symptoms*** are usually those of a weak, debilitated, nervous constitution, though in mild cases the general health may be unimpaired. In atrophic pharyngitis considerable cachexia may be present.

Diagnosis.—Care should be exercised in discriminating the variety of chronic pharyngitis present in any given case, so that the treatment may be planned accordingly. Careful and repeated inspection of the throat must render the diagnosis easy unless ulceration has taken place: in such cases a ***tuberculous*** or ***syphilitic*** sore throat must be eliminated by the superficial character of the ulcers, by their ready response to proper treatment, by the history of the case as to specificity, and by the absence of marked pain or symptoms pointing to tuberculosis. When due to gastric disturbance the lower throat will be deeply congested and the tongue will be irritable, with red papillæ standing over its base (Price-Brown).

Prognosis.—This should be guarded as to cure, on account of the stubborn resistance to treatment and the difficulty in removing unfavorable influences. Acute exacerbations are liable to recur unless rigid caution is practised at all times in avoiding the cause of the trouble.

Treatment.—The local use of astringent and alkaline antiseptic sprays or of the nasal douche is usually recommended, but has only a palliative effect. Silver-nitrate cauterization may be tried. The only effectual means, however, of curing the follicular or hypertrophic variety is that used by most throat-specialists—namely, the wire galvano- or actual cautery. Applications of silver nitrate (gr. x to the ounce—0.648 to 32.0) and the internal use of the oleoresin of cubebs have been recommended for the atrophic pharyngitis. Insufflation of powdered tannin or alum is also of service.

Systemic disturbances need attention according as they present themselves. Mineral baths are sometimes of great benefit, and tonics are usually indicated. All irritating causal factors must be removed or avoided before any favorable results can be hoped for from local applications. Tobacco-smokers and topers must deny themselves their habitual luxuries. Krause and Heryng recommend with favor curetting and the application of lactic acid to superficial tuberculous ulcers.

ACUTE INFECTIOUS PHLEGMON OF THE THROAT.

Definition.—An inflammation of the pharyngeal mucosa that passes rapidly into a suppurative process. It is exceedingly rare.

Its **etiology** is not definitely known. I have met with no cases except in my hospital wards, though they doubtless occur in general medical practice. The clinical features have been described by Senator.

The **symptoms** are *sudden in their onset* and quite intense. They are severe soreness of the throat, dysphagia, and hoarseness, as a rule; in advanced cases there has been difficult respiration. *Inspection* shows the pharynx to be deeply injected and the seat of marked inflammatory edema, the neck appearing greatly swollen as well. The general disturbance is correspondingly severe.

The **treatment** is wholly symptomatic.

RETROPHARYNGEAL ABSCESS.

Definition and Pathology.—A suppurative inflammation (rare) of the connective tissue lying anterior to the cervical spinal column.

Etiology.—The disease is relatively most common before two years of age. It is usually a primary affection, occurring without assignable cause, but a certain proportion of instances are doubtless caused by caries of the cervical vertebræ. It may rarely be secondary to any of the specific fevers. Traumatism causes occasional instances.

The **symptoms** are *pain* in swallowing, *impeded respiration*, soon becoming stertorous in character, the dyspnea meanwhile constantly increasing. There may be *couyh*, and the voice may present abnormal characteristics. The signs of *stenosis* finally declare themselves with considerable violence, and an examination of the pharynx usually serves to make the diagnosis positive; the projecting tumor is visible, and the palpating finger readily detects fluctuation. In children the *general features* (slight fever, anorexia, languor) overshadow for days the *local*, while in adults the condition develops acutely with severe faucial symptoms.

The **course** of the disease may be acute, lasting one or two weeks; more frequently, however, it is subacute (rarely chronic), as, for example, when it is due to caries of the vertebræ.

The **prognosis** is favorable in all cases that are early diagnosticated. If unrecognized until the later stages have been run, suffocation may ensue, or rupture into the larynx may cause death by asphyxia.

Treatment.—As soon as fluctuation is detected the abscess should be freely opened, and preferably, as a rule, through the mouth by means of a guarded bistoury. The throat, after the abscess is thoroughly evacuated, should be washed out with some mild antiseptic solution (salicylic acid 2 per cent. or boracic acid 2 per cent.). When pointing occurs at the side of the neck, as sometimes happens, the incision should be made through the skin in that locality. Constitutional indications are to be fulfilled in accordance with general principles, and the strength of the patient is to be maintained by a highly nutritious dietary.

VI. DISEASES OF THE ESOPHAGUS.

ESOPHAGITIS.

ACUTE ESOPHAGITIS.

Definition.—An acute inflammation affecting either the mucous membrane or submucous tissues of the esophagus, or both.

Pathology.—The ordinary morbid changes of an acute esophagitis are those of a simple catarrhal inflammation of the mucosa. It is rather characteristic of the condition that there is no increased secretion, a sponginess and rapid desquamation of the epithelium taking place instead, and causing a granular appearance of the membrane. Occasionally the mucous glands are swollen, and may break down, with the formation of small follicular ulcers. Catarrhal erosions may also be seen here and there. A croupous or diphtheritic exudate is seldom found in the lower portion of the esophagus, and small-pox pustules are rarely, if ever, seen. A diffuse or circumscribed purulent inflammation of the submucosa may dissect up the mucous membrane so as to considerably diminish the esophageal caliber; pus is usually discharged into the tube. In severe cases of poisoning (*corrosive esophagitis*) sloughing may extend into the muscular layer, and may produce a foul, dark, hemorrhagic mass. A fibrinous cast of the gullet has been vomited up by an hysteric woman (Birch-Hirschfeld).

Etiology.—The causes of acute esophagitis, other than traumatic, are rare. Under the latter are included the *mechanical*, *thermal*, and *chemical* irritants, such as the presence of foreign bodies and the swallowing of hot liquids, corrosive poisons, "concentrated lye," mineral acids, and arsenic. The condition may also be the result of the following: (*a*) an extension of catarrhal inflammation of the pharynx; (*b*) specific infectious fevers, as typhoid, typhus, and pneumonitis; (*c*) diphtheria (*pseudo-membranous esophagitis*) by the extension of pharyngeal diphtheria; (*d*) small-pox, giving rise to a pustular inflammation of the gullet; (*e*) local disease, as carcinoma of the esophagus, glandular or vertebral abscess, or laryngeal perichondritis (Strümpell).

Symptoms.—*Pain* during deglutition may be referred to the region of the esophagus, and a steady, dull pain may exist beneath the sternum. *Dysphagia* and *regurgitation of food* may be caused by spasm in severe cases. Mucus, blood, and pus may be discharged later. The absence or mildness of pain is not a true indication of the gravity and extent of esophageal inflammation.

Sequelæ.—Simple catarrhal or follicular ulcers may appear, and the necrotic form of the disease may be followed by suppurating ulcers, which, if healing takes place, may cause cicatricial stenosis.

Diagnosis.—This may be based upon the localization of pain, especially during deglutition; upon the pain occasioned by the passage of the esophageal sound; and upon the mucus, blood, or pus adherent to its bulb on withdrawal, provided carcinoma at the cardiac orifice of the stomach can be excluded. The expulsion of a pseudo-membrane (diphtheritic) from the gullet should be differentiated from esophagomycosis (thrush), especially in children. The diagnosis of the particular form of esophagitis will depend upon the facts elicited relating to the etiology.

The **prognosis** is good in mild cases, and should be guarded in those associated with grave disease. Death may occur in either the purulent or necrotic form.

Treatment.—This is entirely symptomatic, and in severe cases is of little value. A soft, bland diet, preferably of milk, may be borne in ordinary instances; if not, rectal alimentation should be resorted to. For the mild cases swallowing of bits of ice, and later of warm demulcent drinks, should be recommended. In cases of marked pain and esophageal spasm relief may be afforded by a hypodermic injection of morphin and atropin.

CHRONIC ESOPHAGITIS.

Chronic catarrh of the gullet may result from continued irritation by the causes of the acute form, and also from passive congestion due to hepatic cirrhosis, chronic cardiac or renal disease. The last-named conditions may also cause varicose esophageal veins, and fatal hemorrhage may result therefrom. The increased mucous secretion may cause eructations and nausea.

Postmortem evidence of esophagitis, either acute or chronic, is found with extreme rarity.

ULCER OF THE ESOPHAGUS.

THIS is a consequence of a simple or follicular catarrh of the gullet or of gangrene. " Catarrhal erosions " and follicular ulcers may occur, and also necrotic ulcers, in bedridden persons opposite the cricoid cartilage. The extensive purulent ulceration following the separation of necrotic sloughs may heal and cause stenosis of the tube, or it may rupture into the trachea, the posterior mediastinum, or the aorta. Ulcers simulating those occurring in the stomach (*ulceres ex digestione*) may sometimes be found at the lower end of the esophagus. *Postmortem* digestion, however, must not be mistaken for peptic ulceration. There may be localized points of pain on the passage of the esophageal bougie, with some pus and blood on the bulb after its withdrawal. Rest from swallowing should be secured as far as possible. The sipping of hot milk may be soothing, and the slow swallowing of mild boric-acid and sodium-bicarbonate solutions may be tried with benefit.

CARCINOMA OF THE ESOPHAGUS.

THIS is the most frequent affection of the tube, and, as it is the commonest cause of stenosis, it is important from a diagnostic standpoint.

Pathology.—Carcinoma of the esophagus is primary and of an epitheliomatous nature, the mucous membrane here being composed of pavement-cells. The new growth affects the mucosa first, and then, increasing in size and causing ulceration, it involves the entire circumference of the tube like a ring-like mass. This may either be hard and fibrous, or soft and jelly-like. The esophageal lumen is markedly diminished, though disintegrating ulceration or "flat" carcinoma may encroach upon the caliber but little. There may be a diffuse dilatation of the esophagus above the growth, as well as an hypertrophy of the circular muscular fibers. The cancerous tumor is found most commonly in the lower third of the esophagus (generally at the point where the left bronchus crosses the tube).

Etiology.—The *predisposing causes* of esophageal carcinoma are age and sex, males past forty years of age being the usual subjects of this neoplasm. The *exciting causes* are of uncertain origin. It has been alleged that various forms of protracted irritation of the mucous membrane may cause the development of carcinoma; and especially has this point been maintained in connection with the frequent occurrence of carcinoma of the gullet in topers. It is also believed by some that as gastric carcinoma may develop from the scars of old ulcers, a like condition in the esophagus may act as a nucleus for a carcinomatous growth.

Symptoms.—*Dysphagia* is the earliest symptom of esophageal carcinoma with beginning stenosis of the tube. This gradually and steadily increases, so that liquids alone can be swallowed, and later regurgitation even of liquid foods takes place. There may be considerable *pain*. I recently saw an instance with Dr. W. Frank Haehnlen in which enormous quantities of mucus were regurgitated and in which the symptoms of bronchiectasis developed near the close.

The *ejecta* may contain cancerous fragments, blood, and mucus. The dysphagic symptoms may subside spontaneously, owing to the disintegration and ulceration of the growth, or the dysphagia may be so slight as to be masked by the prominent symptoms of hepatic or pulmonary carcinoma and gangrene secondary to a very flat esophageal carcinoma. Or, without secondary manifestations of such a growth, the esophageal symptoms may rarely be latent. The cervical glands may be enlarged.

The most important *general symptom* of esophageal carcinoma, as of this malignant growth elsewhere, is the progressive emaciation, which increases with the stenosis and obstruction to the entrance of nourishment into the stomach. Though seemingly anemic, the patient's blood may contain an excessive number of corpuscles in a given bulk. This is due to inspissation from failure to absorb water and food into the body.

Course, Duration, and Termination.—The disease is chronic, becoming progressively worse, and is often beset with grave complications (*vide infra*). It seldom lasts longer than one and a half years, and the duration of medullary carcinoma of the gullet is usually much shorter. A fatal ending is inevitable, by inanition and exhaustion, or as the result of metastasis and secondary complications.

Complications.—These follow extension of the cancerous growth to neighboring parts. Thus, involvement of the larynx, trachea, and bronchi has been noted. The cancerous ulcer may also perforate the pleura, the pericardium, or the aorta or its branches, and cause fatal hemorrhage. The vertebræ have been eroded, and compression of the cord, with resulting paraplegia, may take place.

Paralysis of the vocal cords may be the effect of pressure by the growth upon the recurrent laryngeal nerve; most frequently pulmonary gangrene is due to perforation of the lung or to the inspiration of cancerous and decomposing particles that have been regurgitated.

Diagnosis.—As the dysphagia is a symptom of paramount importance in the diagnosis of esophageal carcinoma, all other causes of the symptoms must be excluded. The *enlarged tonsils*, *pharyngeal tumors*, pressure from without by cervical intrathoracic tumors, as *aneurysm*, or by displacement of the sternal end of the clavicle, and the presence of *foreign bodies* or *cicatricial strictures* of the gullet—all figure in the production of difficult deglutition. The history of the case, the age of the patient, the progressive emaciation (cancerous cachexia), and the obstinately increasing dysphagia will enable us to exclude the other affections named. In using the esophageal bougie for diagnostic purposes great care should be exercised, as an aneurysm may thus be ruptured or a deeply ulcerated carcinoma perforated. The withdrawal of cancerous tissue upon the bulb will decide the case. The esophagoscope may be useful in certain cases, but requires great care and special skill. The exceptional occurrence of latent cases must be remembered.

The **prognosis** is hopeless, and the supervention of grave complications or pulmonary gangrene renders the chances of an early demise very probable.

Treatment.—This is essentially symptomatic and sustentative. If feeding by the mouth is difficult on account of the extreme stenosis, although permitting the passage of an esophageal tube, the latter may be used for the passage of liquid nourishment. Rectal feeding may later become imperative. The mechanical treatment of the cancerous stricture by the passage of the graduated esophageal bougie is seldom of any avail, although temporary improvement may perhaps be obtained. Soft, disintegrating, and ulcerating carcinoma should thus be treated, though with the absence of any force whatsoever, lest perforation take place. The performance of gastrostomy may prolong life in some cases in which there is formidable difficulty in passing a tube into the stomach.

RUPTURE OF THE ESOPHAGUS.

THE first recorded case of this rare condition occurred under the observation of Boerhaave in 1724 in the person of the Baron Wassemar.

Pathology.—Softening, together with a great friability, of the esophageal walls may be found, this probably being the effect produced by the solvent action of the gastric juice upon the mucous membrane at a time when the local circulation is disturbed and the vitality of the tissues thus lessened.

The *postmortem* evidence of this accident consists of a longitudinal (as a rule) tear about 5 cm. (2 inches) in length, and situated in the lower half of the esophagus. Food and air may be found to have escaped into the left pleural cavity, and unless death occur at an early date signs of secondary purulent inflammation will probably be noticed. *Postmortem* digestion of the esophagus is more common (Osler). The perforation is often quite large, and is located in the posterior wall of the tube.

Etiology.—Softening of the walls of the gullet (*esophago-malacia*) is suggested by Zenker as a condition that always precedes spontaneous rupture, so called. The exciting cause is believed to be violent and persistent vomiting after a particularly heavy meal or during acute alcoholism.

Symptoms.—These come on *suddenly* or soon after a full meal, and commence with *nausea* and very *severe vomiting*, accompanied by *great pain* and rapid and extreme *collapse* of the whole body, due to the shock. A cutaneous emphysema of the neck and chest is manifested soon after the rupture.

The **diagnosis,** if made at all, must rest upon the clinical history. **Death** usually takes place in a few hours, or days at the most, and the **prognosis** is necessarily hopeless.

The **treatment** is equally so in the present status of surgery. Pain, if excruciating, should be dulled by the hypodermic administration of morphin.

NEUROSES OF THE ESOPHAGUS.

MUSCULAR SPASM.

(*Esophagismus.*)

Definition.—A spasmodic contraction of the muscular layer of the esophagus.

Etiology.—It is almost always a secondary affection, met with not infrequently in hysteria, hydrophobia, and rarely in chorea and epilepsy. I have seen one instance of the idiopathic form of the disease in a female possessing a highly neurotic constitution. In this case the esophageal bougie could be passed only with a great deal of difficulty during the spasm. It has usually been observed in aged males, and especially in those suffering from hypochondriasis. It may be due to reflex causes, originating, for example, in the uterus; thus, in some cases, it occurs only during the pregnant state. Rosenheim contends that cardiospasm may follow primary atony and ectasia of the esophagus.

Symptoms.—The chief subjective characteristic is *dysphagia.* Although liquids can be swallowed, solids, as a rule, cannot. Post-sternal *pain* is sometimes noticed, and choking signs are quite common. In the hysteric variety emotional disturbances are found among the prodromata, and most probably bear a causal relation.

Diagnosis.—The etiologic factors must be carefully weighed in connection with the symptoms and the valuable testimony gained by the use of the sound. The bougie on reaching the constriction is rather tightly gripped, though gentle pressure soon causes it to relax. After the subjective symptoms and spasm are over the sound passes without the slightest difficulty, providing a point of the greatest diagnostic import. The elderly hypochondriac is, as before stated, liable to develop a similar condition, which must not be confounded with true cancerous stricture. The **prognosis** is good.

The **treatment** is directed to the disease on which the condition is found to depend, and this must receive careful attention. The sound should be used as previously indicated under the discussion of Esophageal Stricture. Its passage has often been followed by speedy and permanent cures. A special electrode with which to apply electricity to overcome the spasm of the cardia has been employed.

PARALYSIS OF THE ESOPHAGUS.[1]

In extensive bulbar paralysis, when adjacent parts are involved, we may infer the existence of esophageal implication, though there be no objective evidence to adduce in confirmation. Doubtless the esophagus rarely shares in post-diphtheritic paralysis also. Dysphagia is the leading symptom. An invaluable peculiarity belonging to diphtheritic paralysis is the fact that solids are more readily swallowed than liquids.

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[1] For remarks on the treatment of this complaint the reader is referred to section on Nervous Diseases.

DILATATION OF THE ESOPHAGUS.

Pathology and Etiology.—Diffuse dilatation of the esophagus is usually secondary to stricture at or near the cardiac orifice. In accordance with the common law of compensation, the first effect of the stenosis is to engender hypertrophy of the muscular layer above it with a view of overcoming the resistance caused by the obstruction. The wall of the esophagus becomes thickened, and the tube is generally somewhat narrowed, above the seat of the stenosis; but finally, as a result of degenerative changes, the muscular coat weakens, the esophagus dilates, and food accumulates above the stricture—a condition that, once begun, progresses. Dilatation may rarely follow hysteric spasm of the cardia.

Congenital dilatation, in which the whole extent of the tube participates, has also been met with, though such a condition is rare indeed. It sometimes results from fatty degeneration of the muscular wall, and a predisposition to the complaint may be acquired as the result of injury or prior inflammation.

Symptoms.—The essential symptom is chronic *dysphagia*. When dilatation follows stenosis the patient often locates the point at which the food lodges in the esophagus. Most of the ingesta are regurgitated several hours after eating, and this process is often attended by more or less severe strangling. The *esophageal sound* comes upon the stricture, and is either gripped firmly or totally resisted; in the latter event the bulb can be moved about above this point with abnormal freedom. In the rare cases of spindle-shaped dilatation without stenosis the sound usually detects no obstacle on its way into the stomach. A *sac* is occasionally formed, however, as the result of localized bulging of the paralyzed wall, in which food may collect or the exploring sound may catch, thus leading to erroneous inferences. Dysphagia is present, though it presents peculiarities, in that the food may either pass down very slowly until it reaches the stomach, or it may find its way down for some distance and then lodge in the shallow pouch, as above described. In the latter event the food may be gulped up from time to time. If the sound can be easily introduced into the stomach, we may safely eliminate stricture as the cause of the dilatation.

The **prognosis** is good as long as sufficient food can be gotten into the stomach for the support of life.

Treatment.—The chief object in the treatment of this condition is to keep the patient well nourished. If sufficient food cannot be swallowed, a Symond's tube should be inserted and nourishment given through it; and when this mode of feeding is no longer feasible, the physician has to choose between gastrostomy and rectal feeding. There can be no doubt that by means of nutrient enemata nutrition may be fairly well maintained for a considerable period of time, but not indefinitely, as these cases would seem to demand. In the hands of a competent surgeon, on the other hand, gastrostomy is often fruitful of brilliant results. Galvanism has been recommended on high authority, but I cannot speak from personal experience in its use. Local lesions, when present, must be dealt with in accordance with the rules governing the treatment of the several causal conditions.

ESOPHAGEAL DIVERTICULUM.

(*Pharyngocele.*)

Definition.—A circumscribed sac in the wall of the esophagus.

Pathology and Etiology.—Two varieties are met with, which Zenker has termed *pressure* and *traction* diverticula; the latter are rare. Diverticula that occur at or near the inferior constrictor, and more particularly the larger ones, are *congenital* in origin. When *acquired* they are the result of a localized lesion in the muscular coat, through which the mucous membrane bulges like a hernia. This is owing to repeated slight pressure occasioned by the passage of food. When once such a process is started, various factors tend to continually enlarge the pouch. Chief among these are the morsels of food that find lodgement and naturally tend to augment the size of the diverticulum by dragging it downward. The sac may finally attain a diameter of not less than 4 inches (10 cm.). Its situation is nearly always on the posterior wall at the pharyngo-esophageal junction, and its form is usually saccular or pear-shaped. Most instances have been met with in males after middle life. The cause of the weakened area at which the diverticulum occurs is to be found sometimes in injury, but more frequently in an antecedent inflammation. Histologic changes are observed only in the mucous and submucous layers, these anatomic elements together forming the pouch.

Traction diverticula are produced by the fringe of tissues that often becomes adherent to the upper aspect of the esophagus, and from their mode of occurrence they will obviously be more or less funnel-shaped. Their dimensions are small. They are more common in children than in adults, for the reason that in the former, more frequently than in the latter, do the bronchial glands suppurate, with subsequent cicatrization. This circumstance affords an explanation of the fact that traction diverticula are usually seated on the anterior wall of the esophagus, near the bifurcation of the trachea.

Clinical History.—*Traction* diverticula do not, as a rule, give rise to clinical symptoms. Exceptionally, however, as the result of the mechanical irritation caused by bits of food that are retained in these funnels, ulceration may occur and be followed by perforation of their apices. In this manner the main bronchi are often perforated (causing pneumonia and pulmonary gangrene), also the pleura (causing empyema), and, more rarely, the pericardium (causing suppurative pericarditis).

Pressure diverticula when small cannot be recognized, owing to the absence of signs and symptoms. When they attain considerable size, however, they are often attended with severe symptoms. The earliest clinical manifestation is difficulty in swallowing; some of the food enters the sac, and, if allowed to remain, undergoes putrefactive decomposition, causing *fetor of the breath*. From time to time, and especially on attempting to swallow, the partly or wholly filled condition of the pouch excites *nausea* and *vomiting*, associated with prolonged *strangling;* this results in the ejection of a portion of the accumulated contents. After such an attack the patient is unable, temporarily, to swallow food, and in consequence of the limited amount of food taken signs of inanition soon appear; this may finally become extreme, and is sometimes the immediate cause of death. The appearance of a *pear-shaped swelling* in the side of the neck has been observed. As the tumor enlarges it displaces the larynx and presses upon the enlarged vessels—more rarely upon the superior laryngeal nerve, giving rise to dyspnea and distressing fits of coughing.

Diagnosis.—A point in the differentiation of this affection is the enlargement of the sac after meals (not all the food passing into the stomach), and its disappearance after being emptied. Another discriminating sign is the effect of compression by the hand in causing the contents ("air and sodden food") to flow back into the mouth. In those instances in which the tumor is absent we may demonstrate its existence by the use of the esophageal sound. If the sound passes into the sac, the descent will soon be arrested. If, however, the instrument fails to enter the mouth of the pouch, it readily glides into the stomach. An elbowed sound, bent at an obtuse angle near the tip, is especially useful in such cases. It may be inserted in different directions, so as to avoid entrance into the sac. Schwalbe and Rosenfeld have been able to recognize the condition by the aid of radiography.

Prognosis.—The outlook is unfavorable in the absence of operative treatment, though modern surgery gives promise of curing a certain proportion of cases. Wheeler has operated successfully in one instance at least. The physician may prolong life by directing attention to the nutrition of the patient, but he cannot hope to promote a cure. If the patient cannot swallow an adequate amount of nourishment, he may be successfully fed for an indefinite period through a tube, which he himself should be allowed to pass. When sufficient food cannot be introduced by this method to maintain the powers of the patient, rectal feeding should be instituted. If excision of the diverticulum be deemed impracticable by the surgeon, then the establishment of a gastric fistula is worthy of extended trial in cases in which the above-mentioned modes of feeding have failed.

STRICTURE OF THE ESOPHAGUS.

Etiology.—A stricture of the esophagus may be due either to (*a*) Congenital narrowing (exceedingly rare); (*b*) Squamous epithelioma, usually producing an annular constriction; (*c*) Rarely to polypi protruding from the mucosa, which almost occlude the lumen of the tube;

(*d*) Rarely to specific inflammation, as syphilis and tuberculosis; (*e*) Simple stricture generally results from the ingestion of corrosive fluids, which cause extensive sloughing of the mucosa, followed by cicatricial contraction; (*f*) Rarely as a sequel of typhoid and peptic ulceration.

Clinical History.—The symptoms vary with the special cause and with the degree of stenosis. The first and most prominent indication of narrowing of the gullet is a very slowly increasing *dysphagia*. The patient for a long time complains of a *sense of pressure* at a certain substernal point on swallowing solid food, or, more rarely, an apparently healthy person will suddenly experience painful pressure in attempting to swallow a larger quantity of food than usual. By and by even fluids cause dysphagia, and the patient observes that the time required for the food to reach the stomach is lengthened. The impediment to the act of swallowing is not due alone to mechanical stenosis, but partly to the weakness of the muscular coat, sometimes owing to its partial destruction, and in exceptional cases partly to spasmodic contraction. When due to carcinoma, difficult deglutition is, as a rule, the only symptom complained of. When occasioned by corrosive fluids or traumatism, *pain* is prominent from the onset.

Above the seat of stricture the esophagus is often *dilated* and contains accumulations of the ingesta. The latter, together with considerable mucus, are regurgitated three or four hours after meals, and we may be certain that the materials thus ejected do not come from the stomach if they are alkaline in reaction. The leading clinical features are the gradually *increasing debility* and *emaciation*, finally reaching an extreme degree.

Diagnosis.—However characteristic the symptoms may be, the bougie should invariably be passed before pronouncing a positive diagnosis. By this means we ascertain the degree and the seat of the stricture. To begin with, a medium-sized gum-elastic bougie (No. 16 English scale) should be employed, after warming it and lubricating with glycerin. Its use should be preceded by a cocain-spray to prevent spasm. The patient should occupy a low seat, with his head supported by an assistant from in front of the operator. The head should be only slightly thrown backward. The forefinger of the left hand should then be passed back over the tongue until it touches the epiglottis, and the bougie inserted along it with the right hand, thus avoiding the error of passing it into the naso-pharynx or the larynx. When the bougie reaches the cricoid cartilage it is sometimes gripped pretty firmly even in a healthy person—a fact that is always to be remembered. No force should be applied. The instrument may pass the constriction with a jerk, or it may not only be gripped, but distinctly arrested, when a smaller bougie should be tried. By moving the instrument upward gently we may detect sometimes several strictures lying one above another. To locate the obstacle, the distance from the teeth to the point of stricture is measured on the instrument, and the results compared with the normal measurements, which are as follows: from the teeth to the cricoid cartilage, 7 inches (17.7 cm.); to the left bronchus, 11 inches, (27.8 cm.); and to the opening into the diaphragm, 15 inches (37.9 cm.).

Auscultation of the esophagus has been practised, but the clinical indications afforded are of little practical value. The stethoscope is placed to the left of the spine, and the patient takes a mouthful of water, when, if a stricture be present, a splashing, cooing sound will be heard at the seat of the stricture instead of the normal esophageal bruit.

Differential Diagnosis.—It is important to determine not only the existence of a stricture, but also the diseased underlying process, since without this knowledge rational methods of treatment cannot be instituted. First and foremost, we must exclude those affections that simulate simple and malignant stricture, in certain of which the introduction of the sound would be attended with grave dangers. *Compression of the esophagus* by enlarged or accessory thyroids, aortic aneurysms, vertebral abscess, enlarged lymphatic glands, and occasionally pericardial effusions, may produce dysphagia, and on passing the bougie resistance is offered at the seat of the external pressure. As a rule, the extent of the stenosis is moderate. If the narrowing be due to aneurysm—"(*a*) rhythmic movement is sometimes communicated to the free end of the sound introduced as far as the stenosis." Careful physical examination will often reveal the presence of an aneurysm or other pressing tumor, and should never be neglected. A passage of the sound in cases of aneurysm has even caused rupture of the sac and death. (*b*) Spasm of the esophagus or paralysis (the latter rarely) may closely resemble true stenosis. These neurotic forms are almost exclusively met with in hysteric females; on the other hand, malignant strictures are found generally in males over forty years; while in simple stricture there is usually a definite history and certain etiologic factors.

To discriminate between simple and malignant stricture is not difficult, as a rule. When a clear history of gumma, of tuberculous disease, or of injury (from corrosive liquids) is obtainable, the presence of a simple stricture may be safely inferred after eliminating the affections previously mentioned. In the absence of etiologic data pointing to the simple form, cases occurring in the male after forty years of age may be looked upon as malignant.

Prognosis.—In forming a prognostic opinion the chief factor to be considered is the nature of the stricture. Practically, so long as the stenosis is dilatable, the prognosis is not unfavorable provided sufficient nourishment can be taken ; moreover, not a few cases of simple stricture are curable. The majority, however, come to a fatal termination finally.

Treatment.—The chief object of the treatment is to gradually and methodically dilate the stricture in a mechanical manner. The flexible English bougie above mentioned is the best for the purpose, commencing with one of good size; conical ivory bougies, having a flexible whalebone handle, may also be used, though, being quite hard, they are apt to inflict injuries unless used cautiously. It is sometimes necessary, on account of the tightness of the stricture, to begin with a catgut sound. The method of introducing these instruments has already been given. They should be used once daily, and often can be passed successfully by the patient himself. At intervals of three or four days trials of bougies of larger size should be made. I have seen truly remarkable results from this treatment when carried forward systematically in cases due to cicatricial contraction, the patients increasing in bodily weight and strength. In annular constrictions of a malignant type, however, it is productive of temporary benefit only.

The *diet* deserves most careful attention. When the stenosis is so pronounced as to prohibit sufficient food being swallowed, a Symonds tube should be passed into the stomach, and through it liquid food is introduced. Concentrated forms of nourishment, as raw eggs, bovinin, and the various infants' foods, may be administered with milk.

When the passage of the bougie is no longer possible relief may be secured in one of two ways: (1) rectal feeding; (2) gastrostomy, if the seat of the stricture be near the stomach, and esophagostomy if at the upper portion of the gullet. I have recently witnessed favorable results from gastrostomy in a case of simple stricture operated upon by Laplace. It is important that the patient should thoroughly masticate the food before introducing it into the stomach. Before resorting to operative procedures, however, careful trial should be made of rectal feeding. Various forms of nutritious enemata and other points regarding rectal alimentation will be found in the Treatment of Gastric Ulcer.

VII. DISEASES OF THE STOMACH.

METHODS OF DIAGNOSIS.

EXAMINATION OF THE GASTRIC FUNCTIONS.

Secretory Function.—When food enters the stomach the glands immediately begin to secrete their various juices, and continue to do so until the food has passed into the duodenum. During the later stages of gastric digestion the activity of the secretory function of the stomach diminishes, and to obtain accurate knowledge of any pathologic condition of the organ, examinations of the gastric contents must be made under conditions as nearly like the physiologic as possible. Reliable results cannot, therefore, be obtained from an examination of ordinary vomita, but the contents of the stomach must be procured at a definite period after a so-called test-meal (*vide infra*).

Numerous test-meals have been offered to the profession, but those that I have found most satisfactory are "the test-breakfast of Ewald and Boas" and "the test-dinner of Leube-Riegel." The former being simpler and easier of preparation than the latter, it is the oftenest used.

The *Ewald-Boas test-breakfast* consists of one or two rolls (50–70 gm.) and one cup of tea or water (300–400 c.c.). I constantly advise the use of one roll and a glass of water. About an hour after this meal has been taken the contents of the stomach are to be withdrawn, and at such a time HCl should be the only acid present.

The *Leube-Riegel test-dinner* consists of a large plate of soup (300–400 c.c.), a large piece of beefsteak (150–200 gm.), and some potatoes (about 50 gm.) or a roll—practically, a large plate of soup, a piece of meat (preferably beefsteak), and a roll of bread. The examination is to be made about three and a half to four hours after the meal.

To obtain the contents of the stomach we should use a soft, flexible-rubber tube with an end-opening, or, better still, with several additional openings on the sides, and it should be marked at a point 23.5 to 25.5

inches (58–64 cm.) from the end introduced, this helping the examiner to determine whether it has entered the fundus. The tube is moistened with water and the end carried back to the pharynx; the patient is now asked to swallow, and the tube is gently pushed down the esophagus, these acts being repeated until the tube reaches the stomach. An ordinary Politzer bag is now attached to the tube (Ewald), or a Boas aspirator (which consists of a rubber bulb having a soft-rubber tube at one extremity with clamps). If the Politzer bag be employed, it is compressed and allowed to re-expand, the contents being thus withdrawn into the bag. There are cases in which it is safer to empty the stomach by siphonage. This is readily accomplished by using a long tube and exerting gentle pressure on the abdomen to start the current.

The method I have most frequently used is that of "expression," as follows: The patient is asked to take a deep inspiration, and then to contract his abdominal muscles as in the act of having a stool: in this way the contents are quickly expelled through the stomach-tube above described. These should be first examined microscopically to detect any residue from previous meals, such as meat and the like, and the quantity obtained should be 20 to 40 c.c. After filtering the gastric contents thus obtained they are variously tested.

Among qualitative tests the following are important:

To determine the *reaction*, ordinary litmus-paper is used; if acid, the blue turns red.

The presence of *free acids* is determined—(*a*) By Congo-red, a solution of which is turned blue by the addition of liquids containing free acids. The use of Congo-paper (prepared by dipping ordinary filter-paper in Congo-red solution) is the easiest method.

(*b*) *Tropeolin O. O.*—Alcoholic solutions of tropeolin are turned by the addition of liquids containing free acids to a brownish-red, deep red, or deep mahogany-brown, according to the amount of acids present. Tropeolin-paper (filter-paper immersed for some time in an alcoholic solution) may be used, but must not be kept too long.

Free HCl.—Günzburg's test—phloroglucin gr. xxx (2.0), vanillin gr. xv (1.0), absolute alcohol ℥j (30 c.c.). To two or three drops of this reagent add an equal number of the gastric filtrate in a porcelain dish, and slowly evaporate to dryness over a flame; and if free HCl is present, a rose-red tint appears along the edges. Blowing at the edge will hasten the reaction. The great delicacy of this test is conclusively shown by its availability when HCl is present in the proportion of 1 to 20,000. There are no recognized interfering conditions.

Boas' Resorcin Test.—Resublimed resorcin 5 parts, white sugar 3 parts, and diluted alcohol 100 parts. The method of procedure is the same as in Günzburg's test, and a purple-red color appears. More caution is required in evaporating, but this method will also detect the presence of free HCl in the proportion of about 1 : 20,000.

Töpfer's Test.—To a few c.c. of filtered (or unfiltered) stomach-contents, 1 to 4 drops of the reagent (dimethylamidoazobenzol in a 0.5 per cent. alcoholic solution) are added; in the presence of free HCl a rose- or cherry-red color is produced. Combined HCl gives a negative result. The presence of acid salts, peptones, mucin, and starch (in the usual percentage) do not interfere with this reaction.

Lactic Acid.—Uffelmann's Test.—The reagent should always be freshly made, as follows: To 10 to 15 c.c. of a 2 per cent. aqueous solution of carbolic acid add 1 or 2 drops of neutral ferric chlorid, when an amethyst-blue color will appear. To 1 or 2 c.c. of the mixture add a few drops of the filtrate, and if lactic acid is present it changes to a canary-yellow color. Sources of error may be overcome by shaking 5 or 10 c.c. of the filtrate with double the quantity of ether, and, after allowing the ether to separate and pouring it off, more is added, the whole shaken, and the washing is repeated. The ether is then decanted and evaporated almost to dryness in a water-bath. To the residue about 1 c.c. of water is added, and to this an equal quantity of the Uffelmann reagent from a pipette; and if a canary-yellow now appears, positive proof of the presence of lactic acid is afforded. Boas and others hold that the presence of lactic acid in the gastric contents during the first stage of digestion has pathologic significance. He also found that ordinary bread contains lactic acid, and hence he has adopted a thin gruel made by adding to a quart of water flavored with salt half an ounce of oatmeal-flour. Boas states that no lactic acid is present in the filtrate several hours after this test-meal, except in cases of carcinoma of the stomach. The use of this test-meal for usual clinical purposes is now generally held to be superfluous. Lactic acid in the stomach-contents occurs with fermentation-stagnation from either obstruction or deficient motility.

A more reliable test for lactic acid than the foregoing is that of Boas, as follows: Digest the filtrate several times with ether to remove the fatty acids; add a few drops of phosphoric acid and boil. Transfer the mixture to a distillate flask; add H_2SO_4 and MgO_2; heat, and lactic acid will be distilled over. This can be conducted into a strongly alkaline solution of iodin and potassium iodid. The presence of lactic acid is then shown by the production of iodoform, which can be recognized by its odor and by the precipitate that is formed.

Fatty or Volatile Acids.—Heat to boiling a few c.c. of the filtrate in a test-tube, over the mouth of which place a strip of moistened blue litmus-paper; the presence of fatty acids will change the paper to red.

Acetic Acid.—In large quantities this acid is detected by its odor, and in smaller quantities its presence is determined by neutralizing with sodium carbonate the watery residue of the ethereal extract, and adding neutral ferric chlorid, when a blood-red color will be struck. Quantitative estimation of certain constituents is desirable.

Total Acidity.—To 10 c.c. of the filtrate add 1 or 2 drops of a 1 per cent. alcoholic solution of phenophthalein, and as many cubic centimeters of a decinormal solution of sodium hydrate are added slowly from a buret until the reddish color that appears fails to disappear on shaking. The number of cubic centimeters of the decinormal solution normally required ranges from 4 to 6; hence, if these be multiplied by 10, we have 40 to 60 as the percentage of acidity. Under pathologic conditions these numbers may be either higher or lower. This total represents both free and combined acids. If no organic acids be present, the above figures will represent the percentage of HCl. The latter is also reckoned thus: If it required 5 c.c. of the decinormal solution of sodium hydrate to be added to 10 c.c. of the filtrate to get the red color (alkalinity) with the phenophthalein, we say the acidity is 50, and multiplied by 0·003,646 = 0.1823 per cent. of hydrochloric acid. It should be stated that the normal range of percentage is from 0.14 to 0.24.

Estimation of Free HCl.—Mintz's method: To 10 c.c. of the filtrate add a decinormal solution of sodium hydrate from a buret until no reaction is given with Günzburg's reagent. The number of c.c. of the decinormal solution used, multiplied by 10 and then by 0.003,646, gives the percentage of free hydrochloric acid.

Töpfer's Method.—To 10 c.c. of filtered gastric juice 1 or 2 drops of Töpfer's reagent are added, and then also a decinormal solution of soda, drop by drop, until the last trace of red has changed to yellow. To estimate the percentage of HCl, the number of c.c. of soda solution required to neutralize the free HCl in 100 c.c. of stomach-contents is multiplied by 0.00365. Example: To remove the red color 4 c.c. of soda solution are required; hence, 0.00365 × 40 = 0.14, the percentage of free HCl.

Estimation of Lactic Acid.—If the volatile acids are present, they should be removed by boiling. Take the total acidity of 10 c.c. of the filtrate; then to a second 10 c.c. add 25 to 30 c.c. of ether; shake well, allow the ether and filtrate to separate, remove the ether, and again add 25 to 30 c.c. of ether; shake, and repeat the process. Next obtain the acidity of the watery solution, and the difference between this and the total acidity, multiplied by 10 × 0.09, will give approximately the amount of lactic acid.

In the *gastric digestion* of the *albuminoids* (proteolysis) the proteids are converted into peptone. Although commenced in the stomach, this function is dependent in greater part upon the action of the pancreatic ferment in the small intestines. Among the substances earliest engendered by this process are the *albumoses* (propeptone), whose separation may be thus effected: Add a small quantity of a saturated solution of sodium chlorid to an equal amount of gastric filtrate, and if it becomes cloudy propeptone is present, the degree of the cloudiness indicating the amount present. If the mixture does not become turbid, add a few drops of acetic acid, when it will become so in the presence of this substance, however slight the quantity. If heated, the solution becomes clear, and if allowed to cool, the propeptone precipitates and may be obtained by filtration.

In a later stage of the process of albumin-digestion peptone is produced and its detection is easy. To a small quantity of the filtrate (the propeptone having been removed) add enough sodium or potassium hydrate to render the solution alkaline; then add a few drops of a 1 per cent. solution of cupric sulphate, and, if peptone be present, a purplish color is presented.

The Test for Pepsin.—In a test-tube containing 15 c. c. of filtrate add a small piece of egg-albumin, and keep at a temperature of about 100° F.; if present, the albumin disappears in from two to six hours. If hydrochloric acid is absent from the filtrate, it is necessary to add a few drops of the dilute acid. It should be pointed out that laboratory attempts to estimate the rate of albumin-digestion are unreliable.

Rennet Ferment.—To 5 or 10 c.c. of raw milk add a few drops of the gastric filtrate, and keep it at a temperature of about 100° F.; if rennet is present, coagulation into a single cake occurs in from a few minutes to an hour or more.

Rennet Zymogen (which is converted into *rennet ferment* in the presence of an acid).—To 5 c.c. of gastric filtrate add enough sodium carbonate or sodium hydrate to make it slightly alkaline; then add calcium chlorid (1–2 c.c. of a 2 per cent. solution); then mix with an equal quantity of milk, and, if zymogen is present, coagulation occurs as in the case of rennet ferment. Both rennet ferment and rennet zymogen may be assumed to be present when HCl has previously been found.

Starchy Derivatives.—To a small quantity of gastric filtrate add 1 or 2 drops of Lugol's solution; the presence of dextrin gives a blue reaction—erythrodextrin purple, achroödextrin, grape-sugar, and maltose (intermediate substances)—showing a yellowish color. If there is a mixture of these starchy derivatives, as when the digestion of starches proceeds naturally, the first few drops of Lugol's solution may produce no color-reaction, or it may be taken up by the dextrose or maltose, while the addition of more of Lugol's solution will give a purple (if erythrodextrin be present) or a blue color, due to starch.

Indeed, if a minute quantity of the solution strikes a blue or purple tinge, conversion of starch into maltose has been abnormally tardy. I believe this is oftenest due to hyperacidity, though it may also more rarely be due to a defective ptyaline-supply.[1]

The Tests for the Motor Function.—More important than the secretory is the motor function of the stomach. There are three recognized tests:

The oldest method is that of *Leube.* It consists in washing out the stomach from six to seven hours after a large meal, preferably consisting of beef-soup (13 oz.), beefsteak (6½ oz.), bread (1½ oz.), and water (6½ oz.), or from two to two and a half hours after Ewald's test-breakfast. Normally, the stomach should be empty within these periods of time, so that if a residue remains it denotes a lack in the motor force.

Salol Test of Ewald and Sievers.—Salol being composed of phenol and salicylic acid, it is not acted upon in an acid medium; therefore, when introduced into the stomach it remains a stable compound, and is only broken up in the intestine by the action of the pancreatic juice. The salicylic acid is absorbed into the blood and eliminated through the urine, in which it can be detected by adding a few drops of neutral ferric chlorid, a violet color appearing. The patient is given 15 grains (1 gm.) of salol in two thin gelatin capsules; the bladder is emptied, and the patient told to urinate every half hour for two hours. Normally, it requires from three-fourths to an hour for the salicyluric acid to appear in the urine, but when the motor function of the stomach is much impaired it may require two or more hours. In order to detect the earliest traces of the eliminated salicyluric acid, Ewald and Einhorn have suggested moistening a piece of filter-paper with the urine, and then allowing a drop of neutral ferric chlorid solution to come in contact with it, the edges of the drop showing a violet color in the presence of the slightest traces. The varying reaction of the intestinal contents renders the salol test very unreliable. Sometimes in healthy individuals the decomposition of the salol is retarded, and to overcome this objection Huber has suggested the determination of the precise time when the salicyluric acid no longer appears in the urine. He found that normally it required from twenty-four to thirty hours. If, then, the reaction outlasts this period, it shows peristalsis or the motor function to be unduly tardy. Leube's test is much more reliable.

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[1] The tests for the estimation of the combined acids, of some of the fatty acids, and of many of the products of proteolysis are complicated and unnecessary in an ordinary clinical examination.

Klemperer's Oil-test.—The stomach is thoroughly washed, and 3½ ounces (100 c.c.) of olive oil are poured into it through the tube. Two hours later the remaining oil is withdrawn by aspiration. As the stomach-wall does not absorb oil, the difference between the original amount and that withdrawn shows the condition of the motility. Klemperer states that at this time the residue should not exceed 20 to 40 c.c. This test is also unreliable.

To Test the Absorptive Power.—The method described by Penzoldt has been almost universally adopted: A capsule containing grains 1½ (0.1), of potassium iodid is given to the patient, care being taken that the capsule is first carefully wiped. The iodid is absorbed from the stomach and appears in the saliva, which is to be examined for the presence of iodin. For this purpose strips of starch-paper (filter-paper moistened in a solution of starch and dried) are used; they are moistened with the saliva of the patient, and the moistened areas treated with a drop of fuming nitric acid. As soon as the iodin enters the saliva, the characteristic reaction for starch is struck—a blue color. Normally, this reaction occurs in from ten to fifteen minutes; under abnormal conditions it may be delayed for half an hour or more. Rarely it fails to occur. This test cannot be strongly depended upon.

PHYSICAL OR EXTERNAL EXAMINATION.

This implies the well-known physical signs—inspection, palpation, percussion, succussion or splashing, and auscultation.

Inspection.—(*a*) *General.*—This may give an idea of the nature of the illness as well as its severity by noting whether the patient appears to belong to a neurotic group, the general health often being good, or whether the patient is emaciated, or has with the latter the cachexia of a malignant growth. In diseases of the stomach attention should be directed to the mouth, and especially to the teeth, because the latter are often of causal importance in many gastric ailments. Dental affections often prevent the possibility of curing the various chronic diseases of the stomach.

(*b*) *Local Inspection.*—In patients with thin and relaxed abdominal walls the contour of the stomach can be plainly noted; especially is this the case in very large, dilated stomachs or in those that have been displaced. The examiner is greatly aided by inflating the stomach with air or gas. The former is to be preferred, for the reason that the supply is easily regulated; he is enabled to watch the different steps of the distention, and after the examination is completed the air is allowed to escape through the tube. For this purpose an ordinary stomach-tube is most convenient, and its passage is to be effected in the same way as in removing the gastric contents. A double bulb-attachment is connected with the external end of the tube, by means of which air is readily forced into the stomach (*Runeberg's method*).

Frerichs' method is sometimes used. It consists in administering ʒj (4.0) of tartaric acid, dissolved in half a glassful of water, and immediately afterward ʒj (4.0) of sodium bicarbonate, dissolved in the same amount of water. Effervescence now occurs, with a progressive visible distention of the organ. The chief objection to this method is the fact that either too much or too little distention is obtained.

The inflated stomach presents a circumscribed protuberance, usually in the epigastric, and also in the umbilical region if the organ is dislocated or dilated. The air may find its way into the intestine, producing a visible change in the contour of the abdomen. Tumors and other abdominal enlargements may also be recognized, and an idea obtained as to which organ is involved, after making due allowances for displacement, as in gastroptosis and pyloric carcinoma. Exaggerated peristaltic waves may also be noticeable in the upper portion of the abdomen, usually when associated with the stomach, and in the lower portion if it is in the small intestine. Peristalsis is increased from various causes—inflation of the stomach, external tapping, neuroses, pyloric obstruction, and the like.

The value of the gastroscope in inspecting the interior of the stomach is, I think, doubtful. Gastro-diaphany (illumination of the stomach) is sometimes useful in showing the fundus extending to a lower level (at the navel) than is indicated by percussion, and in indicating the presence of tumors in the anterior wall of this organ. The Röntgen rays show the outline of the stomach, though indistinctly, after the administration repeatedly of 20 grains of bismuth subnitrate.

Palpation.—This elicits at times more trustworthy information than inspection. The patient should be in the recumbent position, the lower limbs partially flexed on the abdomen and the head low. The examiner should stand at the right side of the patient and use the right hand, which should be warm. With the palmar surface down gentle pressure should be made with the fingers and the ulnar side of the hand. If the abdominal wall is tense, it is best to distract the attention of the patient from the examination by talking to him. In this manner we can corroborate the inspection as to the size, shape, and position of the stomach, and can detect morbid growths and determine their consistency and movability.

Deep palpation, by increasing pressure with a slightly rotatory movement, elicits the degree of sensitiveness, tenderness, or pain, whether circumscribed as in ulcer or diffuse as in generalized inflammatory states (enterocolitis, peritonitis). In deep-seated tumors palpation should also be made in the knee-elbow position, and if movable they may drop to the abdominal wall. Gurgling and succussion-sounds of some diagnostic value may be elicited. In some instances relief from pain may be noted on pressure with the broad hand in neuroses. Variations in the degree of tension and of resistance are found and prove helpful.

With Boas' algesimeter we are enabled to detect the amount of pressure necessary to be exerted over a given area to cause pain by reading the number of kilograms from a scale. In some instances this is a serviceable instrument, but in ulcer, when palpation must be done with the greatest gentleness, it would not be a very safe procedure as compared with the soft hand—nature's own instrument.

Percussion.—The patient is placed in the recumbent position; the examiner uses his fingers and endeavors to discriminate the slightest differences in the note, and percusses lightly. If the stomach is empty or partially filled with gas, it gives a lower tympanitic sound than the colon. To ascertain the size and position of the stomach by percussion the process should begin at the symphysis pubis and follow the median line upward. The upper border of the stomach is at the ensiform cartilage, the lower about two fingers' breadth (3 cm.) above the umbilicus. If the upper margin is some distance below the ensiform, displacement of the organ is indicated; this depression may be occasioned by various diseases of the thorax. The stomach may be elevated by great distention of the gut or peritoneal sac.

It is well to trace the limits of resonance of the stomach and of any areas of dulness met with, so that their size and position may be graphically represented. The differences in the percussion-note over the stomach and colon may be greatly exaggerated by inflating the former. Runeberg's method is to be preferred. By employing light percussion the limits of the stomach can now be easily and accurately defined, unless the transverse colon be at the same time greatly distended with gas. In such instances Dehio's modification of Piorry's method is to be resorted to. It consists in giving about 1 liter (1 quart) of water in fractional doses while the patient is standing; one-quarter of the liter is swallowed and percussion practised, when a dull note will be obtained over the most dependent portion of the stomach. A second quantity of equal amount is given and a re-examination made, and so on, the object being to ascertain to what point the lower border sinks on the addition of more fluid. Boas holds that this method tests effectively the tone of the stomach, and that a *marked* descent of the lower border after each addition of water is indubitable evidence that there exists weakness or atony of its walls. If a neoplasm originates posterior to the stomach or colon, inflation of the latter may cause the previous circumscribed dulness to disappear.

By striking the abdomen in the epigastric region splashing-sounds may be produced. This sign is of diagnostic value in dilatation of the stomach, though its absence does not contradict the presence of the dilatation. Again, if the splashing-sound is obtained in a fasting stomach, it may give a clue to some abnormal condition. The stomach may contain large quantities of fluid and no splashing-sound be obtained. Caution should be exercised lest the splashing-sound sometimes produced in the transverse colon be mistaken for that originating in the stomach; in the former the sound is usually associated with diarrhea, while in the latter constipation usually obtains. The outlines of the stomach can be most satisfactorily determined by auscultatory percussion.

Auscultation.—Various sounds are heard, none of which are pathognomonic of any diseased condition.

Succussion-sounds are produced by shaking the patient, and, if the stomach is dilated and contains fluid, the sounds may be audible some distance from the patient. If these sounds are heard after digestion should be completed they indicate some abnormal condition—defective motility, in particular. If heard below the umbilical line, it usually indicates dilatation. In motor insufficiency (atony) of the stomach-walls splashing-sounds are audible after swallowing a few ounces of water. Partial obstruction of the cardiac orifice causes a delay of the "deglutition murmur" (a hissing sound followed in six or seven seconds by either gurgling, clucking, sprinkling, or splashing), as heard over the esophagus with the stethoscope while the patient is swallowing a liquid, "while in complete or almost complete closure of the cardia, this murmur is absent" (Ewald).

MALPOSITION OF THE STOMACH.

THE stomach may occupy a truly vertical position in consequence of the persistence of the normal infantile condition or of improper clothing—*e. g.* long-continued pressure from corsets. Unless an angular condition of the duodenum, causing obstruction to the outflow of the gastric contents, followed by dilatation of the stomach, be engendered, the malposition is of little or no clinical significance. Transposition of the stomach, with the organs occupying the right hydochondrium, is rarely met in association with transposition of other viscera.

GASTROPTOSIS.

Definition.—Downward displacement of the stomach. The lesser curvature of the organ lies about midway between the ensiform cartilage and the umbilicus, and the greater curvature near the symphysis pubis.

Etiology.—So far as our present knowledge extends, the conditions and circumstances contributing mostly to the origin and development of gastroptosis are—(*a*) Age and sex. Meinert of Dresden found among girls of fourteen years gastroptosis in 80 per cent., and among the women who presented themselves at his private clinic in 90 per cent. According to my observation, gastroptosis is not as frequent among American girls and women as among the Germans. "Dislocation occurs in about 5 per cent. of the male population of Dresden." (*b*) Improper clothing, particularly tight lacing. (*c*) Dislocation of the right kidney. This operates potently, and prolapse of other abdominal organs, as the liver and intestines, is often associated, and may constitute the chief point of departure. (*d*) Repeated pregnancies, inducing a relaxed state of the abdominal wall. (*e*) Muscular strain and local injury, by diminishing the tonicity of the gastrohepatic omentum. (*f*) Abnormalities of the chest-formation (kyphosis); great meteorism, and enlargement of the abdominal organs, especially of the spleen and liver. Certain chronic diseases may be active—*e. g.* chlorosis, tuberculosis.

Symptoms.—Malposition of the stomach may exist without symptoms, but in most instances it produces functional disturbances of clinical importance. The latter are due, first, to the difficulty that the stomach experiences in emptying its contents. Soon functional disorders arise in consequence of gastric atony, and later there is apt to be a greatly diminished gastric secretion, associated with a nervous dyspeptic condition. Especially to be emphasized is the fact that the stomach may be of natural or of diminished size (as the primary result of the compression of the corsets—Fleiner), or it may be dilated—a not uncommon event that often colors the clinical picture in a peculiar manner. *Constipation* due to defective peristalsis, and *colicky pains* due to spasm of the intestinal muscles, are important features.

Physical examination of the inflated stomach[1] permits the accurate demonstration of gastroptosis. The epigastrium is hollowed, while the lower quadrants of the abdomen are prominent. The percussion-note now indicates the position of the organ. It is to be borne in mind that the cardiac end remains fixed at the twelfth dorsal vertebra, while the pylorus moves downward and to the left: this will explain why the epigastrium is free of gastric tympany. *Succussion splashing-sounds* may be heard if atony, with retained gastric contents, obtains. The differentiation of gastroptosis from *dilatation of the stomach* is also accomplished by the method of inflation, since this makes plain the course and position of the lesser curvature and of the pylorus.

The **prognosis** is not bad; it is modified in some cases by the presence of special causal agencies, and in others by the occurrence of certain complications, as dilatation of the stomach.

The **treatment** has relation to the removal of all causes that favor the condition. Nervines and nutrients are especially to be employed. Gastropexy and gastrorrhaphy have given promising results.

DILATATION OF THE STOMACH.

The condition is to be subdivided, clinically, into acute and chronic forms. The normal capacity of the stomach varies within rather wide limits, though the maximum normal capacity, according to Ewald, does not exceed 1600 c.c. (1.5 quarts); enlargements above this capacity may then be said to fall under the heading of dilatation.

Pathology and Etiology.—The chief factor in the production of chronic dilatation is **pyloric stenosis.** This is usually due (*a*) to such diseases of the stomach as carcinoma, cicatrix of an ulcer, fibroid overgrowth and contraction of the pylorus, or the contraction consequent on the action of corrosive poisons; (*b*) to the external compression arising from carcinoma of the liver, pancreas, or gall-bladder, from the omental lymph-glands, and not infrequently from a displaced right kidney, or from large gall-stones; (*c*) congenital pyloric stenosis and adhesions about the pylorus may also act as causes of dilatation.

Obviously, in all such instances increased force is necessary to propel the food from the stomach into the duodenum, thus leading gradually to a hypertrophy of the muscular fibers; this is noted in an especial degree in the immediate vicinity of the pylorus. So long as this hypertrophied state of the muscular layer compensates for the obstructive lesion no pathologic dilatation can occur. Just as soon, however, as the muscles prove to be inadequate on account of secondary degenerative changes, accumulation of the undigested food in the stomach ensues. This tendency for the contents of the stomach to accumulate is very much augmented by the increasing weakness of the muscle on the one hand and the increasing degree of stenosis on the other. A further step in the anatomic alteration is the development of a chronic gastric catarrh in consequence of the chemical and mechanical effect of the undigested food, the latter inevitably undergoing fermentative aud putrefactive changes from prolonged retention. The degree of dilatation is, to some extent, augmented by the generation of excessive amounts of gases under these abnormal conditions, as well as by the great weight of the accumulated gastric contents. When produced in this manner the stomach attains enormous dimensions, and one instance has been recorded in which it was capable of containing 90 pounds of fluid (Loomis). Dilatation is usually general, though there may be mere diverticula corresponding to the seats of ulcers or to erosions of the walls.

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[1] Inflation may be accomplished by the use of effervescent mixtures or by the introduction of atmospheric air (*vide ante*).

Dilatation may also occur *independently of pyloric stenosis*, though this class of cases is not so large as the preceding, nor does the condition attain so pronounced a degree. In this variety of dilatation there is atony of the muscular coats, due to various and dissimilar causes: (*a*) repeated over-strain of the muscular layer, due to over-filling of the organ with food and drink, is a comparatively frequent cause, and one met with in diabetics and in those who habitually drink large quantities of beer; (*b*) chronic gastric catarrh frequently weakens the muscle, and more especially when associated with an over-indulgence in food and drink; (*c*) fatty and other forms of degeneration or nutritional disturbances associated with certain constitutional diseases (particularly carcinoma, anemia, and tuberculosis); (*d*) congenital weakness of the muscular coat; (*e*) impaired innervation, leading to imperfect peristalsis and consequent dilatation; (*f*) omental hernias (Bamberger) that drag down the stomach; (*g*) fibrous bands, by binding the stomach to other organs, will occasionally cause slight dilatation.

Acute dilatation has for its chief causes—(*a*) specific fevers, producing parenchymatous degeneration of the muscular coats; (*b*) the acute paralytic distention of Fagge, who ascribed the condition primarily to chronic catarrhal inflammation; (*c*) the drinking of large quantities of effervescing liquids; (*d*) following shock (Boas, Rosenheim); (*e*) sudden obstruction of the pylorus and of the duodenum (Bettmann).

Clinical History.—Since the diseases causing dilatation are numerous and diverse, the clinical history presents great variations. Associated with the symptoms of dilatation are usually those of the causal affections, and the latter sometimes overshadow the former. Among the earlier symptoms, *increased hunger and thirst* are frequently observed, partly due most probably to the general condition of inanition. The thirst is also due, according to Von Weinig, to the fact that the stomach does not readily absorb water, and the pyloric obstruction prevents the passage of water into the intestines. *Vomiting* occurs at intervals of several days, the matter ejected amounting to from 1 to 3 gallons (4–12 liters). The clinical characters of the *vomitus* are strikingly peculiar. Occasionally the vomiting occurs more or less regularly some hours after feeding. The ejecta often contain remnants of previous meals, are, as a rule, excessively acid, emitting a sour odor, and on microscopic examination they show bacteria, sarcinæ, and torulæ in great numbers. The vomitus undergoes fermentative changes very rapidly, is ill-smelling, the odors being mainly due to sulphuretted and phosphuretted hydrogen. It consists of acetic, butyric, and lactic acids and partially decomposed food (HCl being usually absent), and on standing separates into three layers—an upper layer of brownish froth, a middle one of grayish-brown fluid, and a lower one composed of remnants of food. The acid contents of the stomach are not infrequently regurgitated, causing *pyrosis*. Eructations of foul gases are also common, and certain *general symptoms* almost invariably ensue. Progressive emaciation naturally follows, sometimes becoming extreme. A characteristic symptom is *muscular cramp* affecting the calves of the legs and sometimes spreading to the flexors of the arms and the abdominal muscles. Owing to the fact that but a small amount of liquid reaches the intestines, and also to the impaired absorption from the stomach, there are *constipation* and *scanty* urine, the latter usually being alkaline in reaction. The nervous phenomena of chronic gastritis are in evidence and insomnia is often pronounced. *Loss of consciousness* has been met with. *Tetany*, particularly after lavage, has also been observed. Cardiac palpation and arrythmia are often present and are induced principally by the effects of the dilatation. Nocturnal dyspnea (asthma ?) may develop. A striking instance is reported by J. T. Whitcomb in which nearly all the muscles of the body, including those of the esophagus, appeared to be in a tetanic condition. Dreschfield calls attention to *dilatation of the epigastric veins* in both inguinal regions as an evidence of dilatation of the stomach; he has observed it in more than 60 cases, and refers to compression of tributaries of the portal vein by the dilated and displaced stomach as the supposed cause of the condition.

Physical Signs.—*Inspection* reveals a rounded prominence just above the umbilicus in the supine and just below the umbilicus in the standing posture. In the epigastric region there is sometimes a noticeable depression. Obviously, then, the abdomen is asymmetric in appearance. The outlines of the stomach may be made distinct by the patient taking an effervescing draught, and may sometimes be readily seen, particularly the greater curvature, "passing obliquely from the tip of the tenth rib on the left side toward the pubes, and then curving upward to the right costal margin" (Osler). Sometimes peristalsis is visible through the abdominal walls, and rarely the peristaltic waves are seen passing from right to left. These movements may be excited mechanically by various manipulations. *Palpation.*—The increased resistance of the walls of the stomach and their peculiar elasticity aid us in mapping out the contour of the stomach with more precision by palpation than by inspection alone. The movements of the organ can be plainly felt, frequently leading up to a pyloric mass. A sign of considerable diagnostic importance is the loud splashing sound obtained by tapping the region of the stomach with the finger-tips of both hands alternately, or by shaking the body while the hand is placed over the epigastrium, though this should not be mistaken for a similar sound that may be obtained when the colon contains fluid. The patient may produce and maintain similar splashing-sounds by voluntary efforts. A tumor of the pylorus, which is often displaced, may be palpable. *Percussion* furnishes subsidiary evidence as compared with palpation. The examiner should first percuss the empty, and then the filled stomach, if he would obtain reliable aid from this sign. When empty, an increased area of tympanitic resonance will be obtained, extending from above downward to a point several inches below the umbilicus. If now water amounting to 1 quart (1 liter) be introduced into the organ, and, in consequence, a line of dulness at or below the navel be noted where tympanitic resonance had been found, we have good evidence of the existence of dilatation. The posture of the patient should next be changed, when it will be found that the line of dulness has also altered, The stomach may be expanded by gas or air (*vide* Physical Examination) and its limits mapped out by *auscultatory percussion*. *Auscultation* reveals little that is of diagnostic value. The transmitted sounds heard over the stomach have a metallic ring. I have confirmed the observation by Franck and others, who claimed to have heard peculiar gurgling sounds produced by the heart's action and systolic in rhythm. Fluids swallowed by the patient may be heard dropping into the dilated stomach, and succussion-sounds may be elicited by shaking his body. *Measurements* made by introducing a probang into the stomach until it reaches the greater curvature are valuable only when the degree of dilatation is considerable, on account of the obvious chances of error. In health the instrument passes about 60 cm. (24 inches), reaching a point more or less nearly on a level with the umbilicus, while under conditions of extreme dilatation it may be introduced 70 cm. (28 inches).

Diagnosis.—The diagnosis embraces, first and foremost, the recognition of the special causes. The unmistakable clinical manifestations are the characters of the vomitus and the peculiar manner of recurrence of the vomiting. These, together with the physical signs and a knowledge of the causal condition in the individual case, are adequate for a positive diagnosis.

Differential Diagnosis.—The condition is most apt to be confounded with *ascites* or *over-distention* of the bowel, and in the female with *ovarian cyst*. In *dilatation of the intestines* the gastric symptoms of dilatation of the stomach are wanting; moreover, the physical signs are dissimilar. The splashing sounds on manipulation, the line of dulness below the umbilicus after filling the stomach, and other signs so significant of gastric dilatation are absent in over-distention of the intestines. In addition, we should make trial of the salol test, though this is now considered of little value (*vide* Chemical Examination). In dilatation of the stomach salicyluric acid appears in the urine two or three hours after salol has been taken, while in health as early as from one-half to one hour. From dilatation of the stomach we may discriminate *ascites* by the history and by the characteristic gastric symptoms belonging to the former affection. In dilatation the abdomen is asymmetric, the projecting prominence being in the vicinity of or just below the umbilicus. In ascites the lower portion of the abdomen is chiefly distended, and on assuming the recumbent posture the abdominal area becomes broadened and flattened. On palpation fluctuation may be elicited in the hypogastric and iliac regions. *Megalogastria*, or simple " big stomach," is distinguished by its absence of symptoms, and especially by the fact that the food is passed into the intestines as quickly as in health. *Gastroptosis* is easily distinguished from gastric dilatation (*vide* article on Gastroptosis). The distinction is aided by the use of the x-rays.

Acute Gastric Dilatation.—Acute dilatation of the stomach has a sudden onset, and gives rise to all of the above-mentioned physical signs.

In this type of the disease, however, vomiting is more frequent and severe than in the chronic form. Cyanosis is a common symptom, and pain often a prominent one. The patient frequently passes into a condition of collapse that may prove speedily fatal. Acute dilatation may arise in the course of chronic gastrectasis. Some of these cases probably represent a mere episode in the course of the chronic disease (Veeder, Todd).

Prognosis.—The prognosis in the *acute form* is uncertain, though the majority of cases recover; the condition may, however, tend to merge into the chronic form.

Chronic dilatation offers a bad prognosis, most instances being utterly incurable. Obviously, it depends greatly upon the causal conditions. A resort to surgical interference sometimes gives promise of a more favorable subsequent course in cases of cicatricial stenosis. Cases of dilatation that are not secondary to pyloric obstruction, however, give a more favorable prognosis on the whole.

Treatment.—One of the chief aims of the physician should be to lessen the labor of the muscular coat and to prevent the continual necessity of passing the contents of the stomach into the intestines. This is to be accomplished by careful attention to the character and amount of food taken and by frequent cleansing of the stomach. It is necessary to thoroughly empty the organ by lavage, this being repeated daily. A thorough and safe manner of washing out the stomach is by means of the soft Nélaton catheter, its introduction being unattended by injurious local effects. Perhaps the best way in which to thoroughly empty the stomach is by the use of the stomach-tube, as will be detailed under Chronic Gastritis. Recently this has been replaced by the siphon apparatus as a simpler and more convenient mechanism than the former, and one not so likely to be attended with harmful effects, though perhaps less efficacious. The long course of these conditions renders it desirable that the patient should, whenever possible, be taught to wash out his own stomach. On account of the fermentative and putrefactive changes going on in the ingesta it is necessary to use weak antiseptic solutions for this purpose, suitable ones being a 3 per cent. solution of boracic acid or a 1 per cent. solution of salicylic acid. Subsequently warm water alone may be employed. The *diet* should be composed chiefly of fluids, given in small quantities and at stated intervals. If the pyloric obstruction be not too far advanced, tender meats, eggs, and other easily digested albuminous articles of food may be allowed in moderate quantities. Since gastric digestion and absorption are very often markedly impaired, it is well also to include those substances that are readily digested and assimilated after leaving the stomach, though the latter must be given in a fluid state. In no other manner can we bring such marked relief from disagreeable gastric symptoms as by a suitable dietary, and in no other manner can the general nutrition of the patient be so markedly improved. The weakened condition of the muscle-walls is due to over-strain and to degenerative processes; hence. after having removed as much of the labor thrown upon it as possible, we should attempt to overcome its paretic state by the employment of such agents as strychnin and electricity. Stockton, and later, Reed and others, have obtained good results from direct electrization of the stomach by the use of special electrodes; it improves motility and lessens the size of the organ. Exercises to develop the muscles and abdominal massage are useful. For the associated catarrhal state the remedies recommended under Chronic Gastric Catarrh may be employed.

Since some of the more annoying symptoms and remote evil consequences are directly attributable to the fact that too small a proportion of the stomach-contents finds its way into the intestines, we should compensate for this deficiency of intestinal fluid by rectal injections of a weak solution (gr. v to ℥j—0.324 to 32.0) of sodium chlorid, not less than one pint of this solution being injected twice daily. In addition, nutrient enemata should be employed when, despite proper regulation of the dietary, loss of flesh and strength continue. For the anemia and debility tonics are indicated, particularly iron, which may be administered hypodermically in the form of the albuminate. Finally, surgical intervention may become necessary.

INFLAMMATORY DISEASES OF THE STOMACH.

ACUTE CATARRHAL GASTRITIS.

(*Acute Gastric Catarrh.*)

Definition.—An acute catarrhal inflammation of the mucous membrane of the stomach, attended with more or less severe local and constitutional symptoms.

Pathology.—The postmortem evidences of an acute inflammation of the gastric mucosa are distinctive only of the graver forms, since the latter alone usually terminate fatally. Observations upon cases of gastric fistula, however, have shown that in the milder grades of acute gastric catarrh the morbid appearances are similar to those characteristic of acute catarrhal inflammations of the portions of mucous membrane normally exposed to view. Thus, at first there are small irregular patches of redness, slight swelling, dryness, and ecchymosis. Later, serum effused from the congested vessels, and mixed with an increased quantity of mucus, escaped leukocytes, and desquamated epithelium, is present. Hemorrhagic erosions may be seen; the mucous membrane is now thickly swollen, softened, and covered with a tenacious mucopus, which, from an increase in the number of leukocytes in a more intense inflammation, may pass into a purulent exudate. Infiltration and swelling of the solitary lymph-follicles are frequent; these sometimes form minute abscesses that burst and result in follicular ulcers. The gastric tubules may be filled with a granular débris of epithelial cells that have undergone cloudy swelling and fatty degeneration. The above changes are more commonly seen near the pylorus.

Etiology.—The *predisposing causes* of acute gastric catarrh embrace those various impairments of the system in which the normal functional activity of the stomach is altered or diminished. These are seen as the result of (*a*) improper hygienic surroundings; (*b*) malnutrition; (*c*) the various anemias; (*d*) in gouty and rheumatic subjects; (*e*) in the tuberculous, cancerous, and malarial dyscrasiæ; (*f*) associated with chronic passive hyperemia of the stomach due to emphysema of the lungs, cirrhosis of the liver, and renal and cardiac diseases; (*g*) in sickly and delicate children, in convalescents from acute diseases, and in enervated chronic invalids. (*h*) Persons having chronic gastric catarrh are predisposed to superadded attacks of the acute disorder.

The *excitants* are mainly (1) dietetic. These include the ingestion of much indigestible food; food or drink that is too hot or too cold (*thermal*); sour and highly-seasoned articles; the too free use of condiments; and especially the eating of decomposed canned goods and tainted meats. In cases due to the latter the fermentative and putrefactive agents (acetic, lactic, and butyric acids, and the ptomaïns) are the immediate causes of the catarrhal inflammation and tend to produce the constitutional disturbances, sometimes typhoid or septic in nature, that give rise to the so-called "gastric fever." The term "*crapulous gastritis*" has been applied to those cases due to gluttonous meals. (2) Toxic gastritis. Excessive indulgence in spirituous liquors is a common cause. Certain drugs, as the salicylates, iodids, bromids, arsenic, and mercury. (For the intense form of toxic gastritis, *vide* p. 756.) (3) Acute infectious fevers, as measles, typhus fever, and scarlatina, provoke the disorder ("*infectious gastritis*"), as do also remittent and intermittent fevers, especially when of the pernicious variety. (4) The influence of cold as an excitant of this disease has very probably been overestimated. (5) Since the publication of the observations by Klebs, Fränkel, and others, the *mycotic* origin of the condition cannot any longer be doubted. Among the microörganisms incriminated are the anthrax bacillus, the favus fungus, the Oidium albicans, and the yeast fungus.

Clinical History.—The symptoms of the ordinary or milder variety of acute gastric catarrh are embraced in the description of the "subacute gastritis" or "acute dyspepsia" of some writers. Soon after eating there are uneasiness, fulness, pressure, distress, and, perhaps, a dull pain referred to the epigastrium. Thirst is common, also nausea, eructations of gas or liquid, and, less often, vomiting. The *vomitus* consists of undigested food, considerable mucus, and fluid constituents that are sometimes bile-stained. The percentage of HCl in the stomach-contents is variable, although either absent or greatly diminished as a rule. The *tongue* is coated. The general condition of the patient remains unimpaired, and the average duration is less than twenty-four hours. In *severer cases* the symptoms before stated are intensified, and particularly the nausea and vomiting. Physical exploration discloses slight prominence of the epigastric area, with more or less tenderness on palpation. The *tongue* is dry and heavily coated, the breath unpleasant as a rule, the patient complaining of a flat or bitter taste in the mouth. *Constitutional symptoms* appear early, and the onset is often marked by rigor and a febrile reaction, the temperature rising to 102° or even 103° F. (38.8°–39.4° C.). *Herpes* may appear on the lips and skin—a fact that points to the infectious nature of this complaint. The *pulse* is usually accelerated, and there are indisposition to exertion, headache, dulness, and other nervous symptoms. An erythematous cutaneous eruption is often present, and particularly in febrile cases in children. The marked general disturbance is due to the toxic effects of the products of fermentation and decomposition.

Complications.—Constipation is a comparatively frequent complication, and diarrhea a comparatively infrequent one. Either coincidently or by direct extension the duodenum is similarly affected, and in some instances jaundice becomes an accompanying feature. The duration of this variety of the disease rarely exceeds four or five days.

Diagnosis.—The diagnosis of the lighter, afebrile forms of the disorder is not attended with the slightest difficulty. A logical diagnosis in cases in which well-marked local and general symptoms appear is not easy. The definite etiology, the vomiting (affording temporary relief), the pain or tenderness, the sudden rise of temperature, and the equally sudden fall at the end of a few days, however, are almost unequivocal.

Differential Diagnosis.—The absence of prodromata, of rose spots, of the peculiar temperature-range, and of enlargment of the spleen serve to distinguish this complaint from *typhoid fever*. The instances of indeterminate etiology may present a clinical picture not to be differentiated from certain infectious diseases. Here a careful analysis of the local symptoms and signs will usually lead to a correct conclusion, despite the apparently complete identity of the general disturbances. Close observation of the behavior of any given obscure case for a couple of days will usually enable the physician to arrive at a correct diagnosis. In children headache and vomiting are symptoms often so well marked as to create a striking resemblance to *tubercular meningitis*, but the latter can be discriminated by the history and longer duration. In children acute gastritis with an erythematous rash is often mistaken for *scarlet fever*. The final elimination of the latter disease is usually easy, however, in consequence of the absence of angina, of the typical tongue, the hard and very rapid pulse, and the peculiar desquamation affecting the hair and the nails.

Prognosis.—Quite generally the prognosis is good. When, as sometimes happens, however, the disease is purely secondary, the prognosis must depend largely upon the primary affection. I have found that many persons suffer from repeated attacks of gastric catarrh, each increasing the liability to subsequent attacks.

Treatment.—Our chief aim should be to remove the cause and then to give the stomach complete rest. Hence, whenever the disease is distinctly traceable to errors of diet, emetics of the blandest sort should be employed; large draughts of warm water usually suffice, but lavage is to be preferred in some cases. This should be followed by a purge made up as follows:

℞. Hydrarg. chlorid. mit.,	gr. j	(0.0648);
Sodii bicarb.,	gr. xviij	(1.16);
Sacchari lactis,	gr. xij	(0.777).

M. et ft. chart. No. vj.

Sig. One, dry on the tongue, every hour; the last to be followed in two hours by a wineglassful of Hunyadi Janos or other saline laxative.

The stomach must now have absolute rest for about twenty-four hours, when pancreatized milk or milk boiled with lime-water may be given at stated intervals. If nausea and continued vomiting prohibit the use of milk by the mouth, I resort to rectal alimentation early, and particularly in children. Certain symptoms, as *nausea*, *pain*, and *rest-*

lessness, demand as early relief as possible, and can be most successfully met by the use of morphin in small doses hypodermically at intervals of twelve hours. When constant nausea is the symptom chiefly complained of, I have found creasote combined with bismuth or cocain in small doses to be highly serviceable. Convalescence is usually uninterrupted, and is soon complete. When protracted it is often on account of the too early return to solid articles of diet or the too early use of bitter tonics. The mineral acids should first be administered, well diluted, after the local symptoms have in a great measure subsided, and to these the bitter vegetable tonics are later to be gradually added. Locally, I employ sinapisms at the beginning of severe types of the affection, and follow these with warm linseed poultices lightly applied to the entire epigastric and hypochondriac regions.

TOXIC GASTRITIS.

Pathology and Etiology.—This is an intense form of acute gastritis, produced by the ingestion of irritant and corrosive poisons, among the former being such agents as phosphorus, antimony, and arsenic, and among the latter concentrated mineral acids and strong alkalies. When caused by the non-corrosive poisons intense hyperemia and tumefaction, leading to desquamative changes in the glandular structure, ensue. When excited by corrosive substances necrosis of the mucous membrane may occur, leading even to an involvement of all the coats, and terminating in perforative peritonitis. The lesions may be of various grades of severity, and are either localized or general.

Symptoms.—The symptoms vary somewhat with the nature of the special poison, though they are usually quite violent. *Incessant vomiting*, great pain in the epigastric region, and, later, *diarrhea*, and excessive thirst, together with such symptoms as intense *burning pains* in the mouth and throat and dysphagia, are the most characteristic signs. The *vomitus* contains mucus, sometimes blood, and rarely shreds of mucous membrane. The *physical examination* reveals a marked distention of the abdomen, which is also, as a rule, very painful on pressure over the epigastric region. The *general condition* of the patient soon becomes one of profound prostration; the skin-surface is cold and clammy, and the pulse and respiration are greatly hurried, terminating at times in fatal collapse within a few hours. Sometimes there is a marked febrile movement; the temperature may reach 104° F. (40° C.); the pulse ranges from 100 to 130; and if life be spared long enough toxic nephritis, with or without hematuria, develops. The nervous symptoms (convulsions, stupor, sometimes ending in coma) may be due in part to the renal lesions, though mainly to the diminished alkalinity of the blood. Symptoms of gastric ulcer or of esophageal stricture may be sequelæ.

Diagnosis.—The diagnosis rests upon the history of the ingestion of some poison, upon the character of the symptoms (referable not only to the stomach, but also to the mouth and the pharynx), and upon the results of an inspection of the mouth, pharynx, and the vomitus. A chemical examination of the stomach-contents and urine may be necessary.

Prognosis.—This depends upon the nature of the poison and its dose. When free emesis occurs early the prognosis is thereby rendered more favorable, since under these circumstances both the local and constitutional effects are mitigated. Among unfavorable symptoms may be mentioned the development of signs of collapse or of peritonitis.

Treatment.—To ascertain, in the first place, the special cause of the gastritis, and when this is found to administer the proper antidote to that poison, are measures of prime importance. The stomach should be washed out with warm water containing some demulcent substance and a small proportion of the appropriate antidote. While lavage may be resorted to, it must be cautiously undertaken. Subsequently measures should be employed to combat the active local inflammation. Externally, leeches, followed by the ice-bag, have proved to be the best agents in my own hands; internally, opium, bismuth, and demulcents, with bits of ice, are most useful. Rectal alimentation should form the sole method of feeding so long as the signs of severe inflammation along the upper alimentary tract are present. The indications presented by the general conditions will vary with the general effects of the peculiar poison in each case.

DIPHTHERITIC GASTRITIS.

This form of gastritis is always a secondary condition, though it is not, as has often been stated by others, always caused by a direct extension of the diphtheritic process from the pharynx down through the esophagus to the stomach. It arises more frequently in the course of some other acute infectious malady, as pneumonia, scarlet fever, or small-pox. Though it is regarded as a rare disease, the fact that it is unrecognizable during life renders it certain that the affection is sometimes overlooked. I have seen two instances associated with croupous inflammation of the intestines, both occurring in greatly debilitated children. Osler saw a case which occurred as a secondary process in pneumonia.

ACUTE SUPPURATIVE GASTRITIS.

(*Phlegmonous Gastritis.*)

Definition.—An acute suppurative inflammation of the submucosa.

Pathology and Etiology.—Phlegmonous gastritis is confessedly a rare, and almost invariably a secondary, disease. I have observed pathologic evidences of its presence, however, in two cases that came to autopsy, both patients having died of sepsis. It is excited by an invasion of pyogenic cocci. It may very rarely originate spontaneously or follow an injury, though more commonly it is merely a symptom of a general septic process or a complication of an acute infectious malady. Two forms are described—namely, a *diffuse purulent infiltration* and a *circumscribed form* (*stomach-abscess*). The morbid process begins in the submucous layer, and then spreads in various directions, involving soon the muscular and serous coats on the exterior and the mucous coat on the interior. The limited variety results in the formation of true abscesses that may attain considerable size and rupture either into the peritoneal cavity or into the stomach.

Symptoms.—There may or may not be an initial rigor. Whether the case is ushered in by a chill or not, however, the *temperature* soon rises to 103° or 104° F. (40° C.), and subsequently pursues an irregular course. The symptoms of the *typhoid state* supervene, and are usually associated with the symptoms of the primary affection. Hence the clinical picture is greatly diversified. For a variable period prior to the fatal issue the patient passes into coma. The *local symptoms* and *physical signs* are rarely diagnostic. There is a constantly increasing epigastric *pain; emesis* also appears, the *vomita* often containing a notable quantity of pus-cells.

The **physical signs** reveal but little in most instances, and vary with the form of the complaint. Inspection shows in the *diffuse* form a considerably distended abdomen. On pressure the stomach is found to be quite tender. In the *limited variety* the gastric abscess sometimes gives rise to the physical signs of a tumor, and a localized prominence may be seen over the seat of the abscess; the tenderness to the pressing finger may be confined to the same area. Palpation has served to elicit fluctuation and to define the limits of the tumor, the latter sometimes attaining the size of a cocoanut; on percussion either dulness or a muffled tympanitic resonance is elicited, according to the size of the mass.

Diagnosis.—The diffuse variety cannot, as a rule, be positively distinguished from certain other gastric affections. The detection of pus-cells is, however, of the utmost diagnostic value. Gastric abscess, on the other hand, is often recognizable, since the physician has not only the history to aid him, but also the physical signs, which may demonstrate the presence of a fluctuating tumor.

Course and Prognosis.—The majority of cases reach a fatal termination within one week, and those that do not terminate in death thus early pursue a subacute or even chronic course. They present such symptoms as local pain, chills, and fever, and death results, sooner or later, either from exhaustion or such complications as peritonitis and metastatic abscess with jaundice.

Treatment.—The treatment in the diffuse form is, at best, only palliative. In the circumscribed variety the aid of the surgeon should be invoked as soon as a probable diagnosis has been made.

CHRONIC CATARRHAL GASTRITIS.

(*Chronic Catarrh of the Stomach; Chronic Catarrhal Dyspepsia.*)

Definition.—A chronic catarrhal inflammation of the gastric mucous membrane, presenting various degrees of intensity and embracing the symptoms that are more or less characteristic of widely different clinical forms of gastric derangement.

Pathology.—The anatomic changes are most marked near the pylorus, where the mucous membrane often presents a distinctly wrinkled, mammillated appearance. The mucous membrane looks either red or gray (the latter hue being due to pigmentation), and is pretty generally covered by tenacious mucus, mingled with detached epithelium. Ewald describes the histologic changes thus: "The minute anatomy shows the picture of a parenchymatous and an interstitial inflammation. The gland-cells are in part eroded or show cloudy, granular swelling or atrophy. The distinction between the 'haupt' and 'beleg' cells cannot be recognized, and in many places, particularly in the pyloric region, the tubes have lost their regular form and show in many places an atypical branching like the fingers of a glove. Individual glands are cut off toward the fundus, but appear at the border of the submucosa as cysts, partly empty, with a smooth membrane, partly filled with remnants of hyaline and refractile epithelium. An abundant small-celled infiltration presses apart the tubules, and is particularly marked toward the surface of the mucosa, and from the submucosa extensions of the connective tissue may be seen passing between the glands. The mucoid transformation of the cells of the tubules is a striking feature in the process and may extend to the very fundus of the glands." Hemorrhagic abrasions may be found in cases due to cardiac disease or to portal engorgement. Superficial ulcers may form, usually in the pyloric region or along the lesser curvature, varying in size from a few lines to an inch or more in diameter. and nearly circular in shape. Long-standing cases also present sclerotic changes of the mucous membrane. Of these, two forms are distinguished. In the one variety the mucous membrane is perfectly smooth and atrophied; the glands are displaced, narrowed, and shortened, while the gap thus formed is more or less filled with connective tissue. There is a thinning of the stomach-wall, with enlargement of its cavity. The other form presents a hyperplasia of the mucous membrane, the glandular structure, and the submucous layer, sometimes resulting in enormous thickening of the stomach-walls, with great diminution in the size of its cavity (*gastrophthisis*). The contraction of the new-formed connective tissue may cause polypoid projections. I have seen one instance in which the stomach held less than a half pint.

Etiology.—It is evident that the factors which produce acute gastric catarrh will, if long continued, produce a chronic condition. The causes of chronic gastritis act either as mechanical, chemical, thermic, or biologic irritants, and fall naturally into the following classes: (*a*) Errors of diet (referring more particularly to important articles of food), its variety, and preparation; excessive alimentation; the habit of eating at irregular intervals or with undue haste, and thus not allowing time for perfect mastication of the food. The too free use of ice-water, tea, and coffee during meals plays an important *rôle* in the causation of dyspepsia in America. (*b*) The immoderate use of alcohol stands second in order of importance. Those persons who habitually indulge in alcoholic beverages to excess are prone to an irregular mode of life, which leads to digestive disturbances. Such patients are apt to suffer from the more active forms of the complaint, and, at intervals of time, from genuine acute gastritis. In the same category should be mentioned certain toxic irritants, as the over-use of tobacco and the prolonged use of tonics and purgatives. (*c*) Functional derangements of the stomach sometimes merge into the disease under consideration. This is true of that form in which there is a deficiency in the gastric juice. Under these circumstances fermentative and putrefactive changes set in. Stockton holds that the majority of cases of chronic dyspepsia are of nervous origin. (*d*) Local mechanical influences (portal congestion) may offer resistance or obstruction to the outflow of venous blood from the stomach to the right heart. In this way chronic gastric catarrh is a secondary process in chronic affections of the liver, heart, and lungs. (*e*) Such constitutional conditions as gout, chronic rheumatism, chronic tuberculosis, Bright's disease, diabetes, anemia, chlorosis, chronic malaria, syphilis, and chronic forms of skin-disease. The explanation of the peculiar liability of these conditions to catarrh of the stomach lies in the obstruction offered to the passage of blood through the hepatic and cardio-pulmonary circulation. This is true in an especial degree in chlorosis, anemia, chronic tuberculosis, and malaria; in gout, chronic Bright's disease, and syphilis it is probably due largely to the action of chemico-vital irritants in the circulating medium.

Clinical History.—The *local symptoms* bear a striking resemblance to those of other forms of gastric disturbance. They vary greatly in severity, though never entirely absent, as in the case of purely functional disorders. Deficient secretion of the gastric juice, due to the anatomic changes in the gastric tubules, is a potent factor in the production of the symptoms directly referable to the stomach. It is the function of hydrochloric acid, normally present in the gastric secretions, to destroy the ferment-producing spores; hence when, owing to lack of free HCl, the latter are not destroyed, deleterious products of fermentation are the result, these in turn aggravating and prolonging the course of the affection. Recent investigations go to show that deficient motor power is more important than a deficiency in the secretions in bringing about the clinical phenomena of the disease. The presence of an inordinate amount of mucus which is alkaline in reaction neutralizes in part the HCl; it may also more or less completely cover the ingesta, thus preventing the gastric secretions from reaching them, and lengthening, at the same time, the period of digestion.

Among the *earlier symptoms* directly attributable to the gastric lesions are anorexia (though at times the appetite may be moderately good or even keen); fulness and distress; burning sensations and dull pain in the epigastric region; eructations of gas, which may be either offensive or odorless, during and immediately after meals; regurgitation of fluid, either acid (heartburn), due to the presence of organic or hydrochloric acid, or a bitter form of peptones. These symptoms are usually increased in intensity after meals. The *tongue* frequently appears broad and flabby, and almost constantly the edges and tip are somewhat reddened, whilst the papillæ are enlarged. Occasionally it is small, with enlarged and red papillæ, or it may look healthy. A bad or, at times, a persistently bitter, taste in the mouth and great thirst may be complained of. There is often a profuse secretion of saliva, but the mouth may be dry. *Nausea* is common, and is most marked in the morning hours; it is frequent before or after meals, and often *vomiting* occurs either immediately after meals or a couple of hours later. The *vomitus* will vary somewhat with the time of the occurrence of emesis. Usually it consists of food in the first stages of digestion, mixed with large quantities of mucus. In alcoholic catarrh morning vomiting occurs quite commonly, and consists of a watery fluid composed of saliva and mucus. This class of sufferers from chronic gastritis not infrequently exhibits well-marked evidences of salivation. I have repeatedly found the material vomited in chronic gastric catarrh to be acid in reaction, unless, as occasionally happens, the vomiting takes place several hours after eating, when it is sometimes faintly alkaline or neutral. The acidity of the vomitus is not due to the presence of free HCl, but possibly in small measure to combined HCl, and partly and sometimes largely to acid salts or the weaker acids (lactic, butyric) resulting from the abnormal processes of fermentation.

Microscopic examination sometimes reveals the presence of sarcinæ ventriculi, yeast fungi, and numerous bacterial organisms. The relations of these low forms of life to the pathologic processes going on in the stomach are not well understood, except in the case of the yeast fungus, which is concerned with the process of fermentation. The sarcinæ ventriculi may, however, exercise a causative influence, since certain cases yield readily to the antiseptic method of treatment.

A *chemical examination* of the contents of the stomach for purposes of diagnosis according to the methods laid down in the preliminary section (*vide* p. 735) should not be neglected. In simple chronic gastric catarrh the hydrochloric acid is found to be diminished, and rarely lactic, butyric, and acetic acids are present. In many cases of chronic catarrhal gastritis there is an abundance of mucus (*gastritis mucipara*—Boas); and in other cases there is present a normal amount of acid or even hyperacidity—the *gastritis acida* of Boas. In protracted forms free HCl is sometimes greatly diminished or entirely absent—*gastritis anacida*. According to Boas the difference between this and the atrophic form is but one of degree, all secretion being lost in the latter. In atrophic gastritis then there is little or no mucus in the gastric contents, and in established cases an absence of HCl and of the gastric ferments (*gastritis atrophicans*). Ewald has subdivided all cases into three varieties: (*a*) *Simple gastritis*, in which the fasting stomach contains only a small quantity of slimy fluid, while after the test-breakfast the HCl is diminished in quantity, and lactic acid and the fatty acids are usually present. (*b*) *Mucous gastritis*, in which class the acidity is always slight and the condition is distinguished from simple gastritis by the large amount of mucus present. (*c*) *Atrophy*. Here the fasting stomach is always empty, while after the test-breakfast HCl, pepsin, and the curdling ferments are wholly wanting.

The *absorbent* and *motile* powers of the stomach are both more or less diminished.

Physical Signs.—Sometimes there may be observed an undue distention of the stomach, the prominence being more marked toward the left. On making *firm pressure* over the epigastric region tenderness is often elicited. This is not present in the early stages, nor constantly later, since the degree of inflammatory action is subject to great oscillation. Diffuse tenderness in the absence of a new growth is of great diagnostic value. It is to be recollected, however, that resistance may be felt when the stomach is thickened in chronic interstitial gastritis. Dilatation of the organ may be indicated by splashing-sounds, and these may be present in the absence of gastrectasis at a time when the stomach should contain no food.

On *percussion* we may note alterations in the size of the organ.

Among the general or indirect symptoms manifested the *nervous phenomena* are of first importance. So prominent are they in the clinical picture ofttimes as to lead the incautious physician to the conclusion that his patient is suffering from some primary disease of the brain or nerves. The nervous derangements have been attributed solely to morbid sympathetic disturbances; it is quite probable, however, that we should ascribe a share of the morbid influence to the absorption of toxic materials from the stomach and intestines. Headache is frequently complained of; it is generally frontal, though also occipital, and tends to appear before meals. The so-called sick headache more rarely occurs. Indisposition to mental or physical exertion, vertigo, depression of spirits, and well-marked hypochondriasis are common concomitants. Patients complain of wakefulness and disturbed dreams, though drowsy after meal-time. There is a sympathetic disturbance of the cardiac rhythm, and sometimes dyspnea, owing to the same cause. The urine is often highly colored, scanty, and deposits an abundant uratic sediment; occasionally, however, it is of low specific gravity, rather copious in amount and pale in color, owing to the influence of phosphates. This condition is found in cases in which the nervous element is notably prominent.

Complications.—The intestines often become involved, and usually by direct extension. Implication of the duodenum may lead to jaundice and to obstinate constipation, though only moderate constipation is the rule in catarrh of the stomach. When the process extends to the large intestines diarrhea develops. Alternating constipation and diarrhea are often observed. The nutritive system is, in confirmed cases, seriously implicated, as shown by the anemia, emaciation, and general debility present. It is particularly in examples of combined intestinal and gastric catarrh that we observe the most notable impairment of the general health, and the reason of this will be clear when it is recollected that under these circumstances all the digestive fluids are lessened in amount. The gases generated in the stomach often find their way into the intestinal canal, giving rise to distention, and sometimes to colicky pain. Perhaps many reflex sympathetic disturbances are of intestinal origin. The gastric catarrh may extend upward to the oral cavity. Under such circumstances the tongue is large and heavily coated, with impressions of the teeth upon its edges. Since the mucous membranes are unhealthy, there is produced an abnormal condition of the secretions that renders the breath foul and causes thirst. Certain skin-eruptions, as eczema, lichen, and urticaria, are common concomitants of this disease. By some authors these disorders of the skin are supposed to be caused by the catarrh of the stomach. I have frequently observed, however, that when present their improvement has been followed by an aggravation of the gastric symptoms, and *vice versâ*. A sequel of the disease is dilatation of the stomach, but I believe this to be less frequent than was formerly supposed. The course of chronic gastric catarrh is long, the average duration being considerably more than one year. Its duration may be much abridged by early recognition and proper treatment of the condition. The symptoms at first intermit and are mild, but later are persistent.

Diagnosis.—A positive diagnosis may be based on a clear etiology, the presence of persistent symptoms and signs of chronic disturbance of digestion, a diminished (*usually*), normal, or even increased, amount of HCl (the atrophic form apart, *vide supra*), an abundance of mucus in the gastric contents, and deficient absorptive and motor power. The points of difference between the more serious affections of the stomach (carcinoma, ulcer, and dilatation) and chronic gastric catarrh will be detailed when the former diseases are considered. As I have said, Ewald makes three leading forms of the complaint, based on the results obtained from an analysis of the stomach-contents, but transitional or atypical types are constantly met with in practice.

Prognosis.—Chronic catarrh of the stomach may be said not to manifest an innate lethal tendency. It, however, predisposes to, as well as aggravates, the symptoms of existing forms of acute and serious forms of chronic diseases, especially other organic affections of the stomach. The prognosis depends considerably upon the stage that has been reached when first met with, since the condition is amenable to treatment only when not too far advanced. The prognosis is rendered somewhat more grave by the presence of certain complications, particularly the presence or absence of intestinal involvement. I have seen one case that proved fatal in consequence of stricture of the pylorus.

Treatment.—It must never be forgotten as far as possible to search for and remove the causal affections in every case. When associated with grave forms of cardiac, hepatic, or renal disease these must receive careful attention primarily.

The masticating apparatus must be looked after by the physician, who must also instruct his patient in the art of eating slowly, so that insalivation of the food is thoroughly effected. Too often the quantity of aliment consumed is beyond the need of the bodily functions, and the method of preparing the same faulty. All food eaten should be fresh and pure. Such patients should eat oftener than in health, taking four or five meals in the twenty-four hours. The physician must with untiring diligence attend to every dietetic, sanitary, and therapeutic detail. The major portion of the treatment has relation to—

(1) *The Diet.*—In the matter of arranging the dietary in separate cases the general condition and peculiarities of the individual must be taken into account. The wise physican will be guided to some extent by the dictates of his patient's experience, and will not fail to avail himself of any information obtainable upon this head. In *severe cases* an exclusive milk diet for a period of two to four weeks often gives the best results. The daily amount requisite to meet the demands of the vital functions is 4 to 8 pints. Of this, 5 to 8 ounces are to be taken *slowly* every two hours during the day. The beginning amount, however, must occasionally be smaller—2 to 3 ounces—to be gradually increased. A pinch of salt or from ½ to 1 ounce of lime-water may be added to each feeding, or the milk may be diluted with Vichy. The milk should not be taken iced, but warmed or at the temperature of the room. Boiled milk is objectionable. The stools are to be watched for curds, and when the digestive capacity is exceeded the amount of the nutrient should be lessened and other articles cautiously added.

When *whole* milk cannot be digested on account of an actual loathing for it, skimmed or partly skimmed milk or buttermilk should be substituted. If the latter cannot be utilized in proper amount, animal broths, together with some of the artificial foods (panopeptone, liquid peptonoids), may be added. As tolerance for a liberal amount of milk becomes established the appetite is no longer satisfied, and then I begin to add the light solids in a gradual manner; for example, white meat of chicken or game (except tame ducks and turkey), stale or twice-baked bread, milk or dry-toast or zweiback, soft-boiled eggs, oysters, fish, and, later, Hamburg steaks, stewed sweetbread, and the like. For dessert, junket or custards, sweetened with saccharin, are well borne as a rule. Subsequently, farinaceous articles, if thoroughly cooked (except oatmeal), and certain plain vegetables, may be allowed, but their effects must be minutely observed. The former are to be eschewed in cases in which acid-fermentation or flatulency is a prominent feature. Among the latter, rice, spinach, lettuce, and macaroni (stewed in milk) are to be selected. Peas and beans, if green and succulent, may be tried, but if ripe are to be discarded. The only form of fat permissible is good butter. Stewed fruits, Graham bread, and soft, green vegetables are often well borne and tend to overcome constipation. Pig's- and calf's-foot jelly may be allowed.

In light cases and in those of moderate severity, particularly if the cause of the complaint is removable, the dietary need not be rigid at the start. Indeed, to minimize the saccharine articles and starches and to avoid the coarser vegetables, hot bread, pastries, and the like, is all that is required. In the case of confirmed dyspeptics the following articles are to be scrupulously avoided: very fat meats, fat fish-foods, condiments, certain fruits (strawberries, bananas), hot bread, saccharine articles of diet and farinacea, potatoes, particularly sweet potatoes, and other coarser vegetables.

Of *drinks*, the best during meal-time is simple hot water, to which a little milk may be added, or a single coffee-cup of weak tea. Occasionally cocoa is allowable, but ordinary chocolate, coffee, and strong tea are harmful. Too much liquid should not be taken during a meal, since it dilutes the gastric secretion to a deleterious extent, and cold drinks are to be interdicted during the same period. Alcohol, and particularly concentrated spirituous liquors, exert an irritating effect, and hence should be forbidden. In cases in which there is no gastric fermentation certain wines may be allowed (Oporto, Malaga, imported Hungarian Tokay).

(2) *Hygienic measures* are of signal value in this disease. Of these the most important are forms of fresh-air exercise, as bicycling, walking, boating, and horseback-riding. Suitable indoor apparatus for physical exercise is now easily obtainable at little cost, and therefore open-air exercise may be supplemented by the latter. Physical exercise must be carefully supervised, so as to avoid the deleterious effects of over-exertion. I am convinced of the superior advantage of travel, including a sea-voyage, and an appropriate change of air—for example, to the seaside or mountains—particularly for the large class of self-centered and low-spirited dyspeptic patients. A cold sponge-bath, followed by brisk friction of the skin, is to be advised. An abdominal bandage, made of woollen or silk material and constantly worn, tends to increase the patient's comfort.

(3) *Medicinal Treatment.*—Saline laxatives, as sodium phosphate, Rochelle salts or Carlsbad salts, taken fasting in hot water, are advantageous, since they serve to regulate the bowels, to deplete the engorged gastro-intestinal vessels, as well as to rinse the stomach. Hunyadi Janos or Carlsbad mineral waters may be substituted. Their efficacy is much

enhanced when the alkaline carbonates are administered simultaneously. Further than this, little is needed in the majority of instances. The use internally of antiseptics, combined with alteratives and mild astringents, is often beneficial. I can speak most positively in favor of the following pill:

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Hemmeter recommends silver nitrate, in the form of lavage (1 : 2000), or in the form of solution 0.3 to 120 of peppermint-water; of this one tablespoonful three times daily on an empty stomach.

In the *fermentative* form of chronic gastric catarrh the hyperacidity is, in reality, often dependent upon the lack of free HCl; hence this agent should be supplied. It is best administered immediately after meals, the dose being not less than 10 minims (0.666), well diluted, and this may be repeated in the course of ten or fifteen minutes in obstinate cases; it may be combined advantageously with pepsin (gr. v–x—0.324–0.648) or pancreatin (gr. x—0.648). Pancreatin is better associated with sodium bicarbonate in the form of a tablet containing each gr. ij (0.129). Of these two or three may be administered fifteen to thirty minutes after meal-time. Care is to be taken to use only the best articles of pepsin and pancreatin. When hyperacidity exists, diastase and ptyalin may be exhibited, but I have failed to obtain encouraging results from their employ. This class of cases represents an aggravated or advanced form of the disease (atrophic stage), and demands prolonged and varied treatment. At the end of the digestive process it is well to thoroughly irrigate the stomach (lavage), and more particularly if evidences of dilatation be present. The stomach may also be cleansed and prepared for the reception of the next meal in a very agreeable manner by having the patient sip a 2 per cent. solution of borax in warm water or a 2 per cent. solution of sodium chlorid half an hour before meals; indeed, the continued use of simple hot water for the same purpose has, in my hands, often given excellent results. With it must, of course, be combined the saline laxatives and the restricted diet. Not less than 1 pint of water, hot as it can be taken by the patient, should be sipped at each sitting.

To assist the appetites of these patients and to stimulate the secretory function a few drops (not more than 5) of the tincture of nux vomica may be given fifteen minutes before meals, with gr. ij–iij (0.129–0.194) of sodium bicarbonate. These indications are also fulfilled by lavage once daily or bi-daily (if the patient be feeble). If hyperacidity, due to the organic acids, persists despite the measures already recommended, we may combine bismuth subnitrate with magnesia and a few grains of charcoal, this being administered when the stomach is empty. We may also check fermentation by the exhibition of salicylic acid (gr. v—0.324) thrice daily or creasote (gr. ½—0.0324) thrice daily. Germain Sée has recently found strontium bromid (ʒss to ʒj; 2.0–4.0) to be of great value in cases in which gaseous fermentation with hyperacidity is combined with permanent tenderness. Happy results often follow a course at some spa if the patient be under the charge of a competent physician during his sojourn. The robust or plethoric should go to Carlsbad, Ems, and Kissingen abroad, and to Saratoga in this country, using more especially the Hawthorne water. The anemic should go to Franzenbad or the spa near Brussels, and in this country to the iron springs at Bedford, Pennsylvania. A course of the alkaline mineral waters may be successfully taken at home in many instances, though patients are much more apt to obey the physician's injunctions as to diet, exercise, and the like when at a spa than when at home. These waters do not simply act as purgatives, but also as antacids. It has been experimentally shown that sodium chlorid, sodium carbonate, as well as carbon dioxid, promote the secretion of the gastric juice. In the more chronic cases belonging to this class or those that have resisted other forms of treatment intestinal complications are usually found. Here the alkaline waters are to be alternated with calomel in small doses, prescribed thus:

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In the *mucous* variety of gastric catarrh additional indications for treatment are presented. The chief aim should be to limit, as far as possible, the production of mucus and to cleanse thoroughly the stomach prior to each meal, thus preparing the organ for the reception and better digestion of food. Here, again, at least one pint of hot water, containing the substances before mentioned, should be sipped half an hour before each meal. This mode of cleansing the stomach is usually successful; if unsuccessful, however, it should be supplemented by lavage once daily, using the same solutions as above indicated, though in larger quantities. The siphon is also highly useful in cases of this sort in which stricture of the pylorus is suspected and when the food is retained in the stomach much longer than the normal period of digestion. This frequently happens, for the reason that the mucous covering which the food receives not only prevents it from being acted upon by the gastric juice, but also renders absorption tardy. The therapy of this form of chronic gastritis requires, in addition to what has before been given, the more potent astringents for the purpose of arresting hypersecretion of mucus. The best way to use these agents is topically. The stomach may be washed out (at bed-time or early in the morning) with a 2 per cent. solution of alum or a 1 per cent. solution of tannic acid; antiseptic solutions are employed in like manner, a 2 per cent. solution of salicylic acid being especially efficacious. If lavage cannot be practised, such astringents as catechu, cerium oxalate, and silver nitrate, with small doses of opium (*vide supra*), should be tried. For use internally one of the very best remedies is atropin sulphate.

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The ulcer often heals by cicatrization. The resulting scar is pale and stellate, and there is puckering of the surrounding mucous membrane. If the ulcer has not extended deeper than the mucous membrane, granulation-tissue develops from the edges and base; this tissue slowly contracts, uniting the margins and leaving a comparatively smooth scar. On the other hand, if the ulcer be large and involve the muscular and serous coats, stricture of the pylorus, followed by dilatation, may result. The stomach may present an hour-glass shape, due to the contraction of a girdle ulcer in the central part of the organ. Nearly all gastric ulcers would perforate the coats were it not for the development of a localized peritonitis with the establishment of protective adhesions. The ulcers being usually situated on the posterior wall, the surface of the pancreas forms the point of attachment most frequently, though the stomach may also become adherent to the left lobe of the liver, the spleen, omentum, diaphragm, or the transverse colon. The organs with which the stomach becomes agglutinated may be penetrated by the ulcerative process, resulting in suppurative inflammation (*abscess*); or. guided by the limiting adhesions, fistulous connections of the stomach with the transverse colon, the pleura, the pericardium, lungs, gall-bladder, and the duodenum may be established. Of these, gastrocolic fistulæ are the most common. Osler states that there are two instances on record in which the ulcer perforated the left ventricle. Penetration of the ulcer through the posterior gastric wall opens the lesser peritoneal cavity, in which case the base remains limited, producing a condition known as subphrenic pyo-pneumothorax. When the anterior surface of the stomach, which has no anatomic relations with other organs favorable for the establishment of protective adhesions, is perforated, general infectious peritonitis rapidly supervenes if a fatal end be not reached immediately. Intense hyperemia or the erosion of small vessels gives rise to small or moderate hemorrhages. If the ulcer penetrate one of the larger vessels, as happens not rarely, then profuse and even fatal hematemesis is the result. This accident is doubtless frequently prevented by the development of a "protective thrombosis." In several instances small aneurysms have been found at the bases of the ulcers (Douglas, Powell, Welch).

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Clinical History.—In *typical cases* of gastric ulcer the clinical symptoms are almost positively diagnostic. The earliest manifestations commonly point to chronic or subacute gastric catarrh, these being followed, soon or late, by those that are characteristic, as *pain*, *vomiting*, and *hematemesis*. Of these, pain is most constantly present, and presents certain peculiarities that demand rather elaborate mention. It is commonly dull, at times burning, and is associated usually with great oppression. These symptoms are doubtless often due to coexisting catarrhal gastritis. The character of pain that is most diagnostic is an *intense gnawing*, *burning* or *boring* in the epigastrium, more or less *periodic* and *strictly localized* in a circumscribed area. These paroxysms usually come on almost immediately after eating, occasionally one or two hours later, and disappear quite promptly when the stomach is emptied either by vomiting or by its contents passing into the duodenum. From the time of its occurrence, the quality, and strict localization of the pain, it may safely be assumed that it is due to direct irritation, set up by the food, of the sensory fibers occupying the base of the ulcer. In addition, there are paroxysms of diffuse pain (*gastralgia*) that are often strictly intermittent, though not necessarily excited by the partaking of food. This pain is due to a sympathetic nervous disturbance or reflected irritation. Finally, sharp, intense, lancinating pains, that are caused by local or general peritonitis, may appear suddenly, ceasing only with the death of the patient. We often meet with the four kinds of pain described above in a single case, though they vary in relative intensity in different cases. The pain in round gastric ulcer is greatly modified by numerous conditions, all of which are largely under human control. The *effect of taking food* has been already referred to, though it should be added that, obviously, undigestible, imperfectly masticated, highly-spiced food, sweet and hot substances, cause the paroxysms to be more intense than less irritating articles of diet. *Rest* diminishes the severity of the pain in that it prevents traction on the ulcer. *Certain postures* may aggravate it, and, though not a trustworthy guide, we may often determine the situation of the ulcer by the effect of posture after taking solid food. The severity of the pain is often increased by bodily fatigue or even moderate exercise, and, to a greater degree, by special emotional influences. The situation of the pain when strictly localized is of the utmost importance in diagnosis. I have found it almost invariably from one to two inches below the ensiform cartilage, though it has been rarely observed in the umbilical and hypochondriac regions. It is absent in one half of all cases. Here may be mentioned a pain-point in the dorsal region (often at a level with the tenth to the twelfth thoracic vertebra) on the left side.

Vomiting, next to pain, is the most frequent symptom, but unless the vomitus contains blood, which is present in less than 50 per cent. of all the cases, it has little diagnostic importance. Nausea and eructations of acid or food often precede or accompany the emesis. Vomiting usually occurs about two hours after eating, and is often coincident with the height of the paroxysm of pain, which the vomiting relieves as a rule. The *vomitus*, as first shown by Riegel, usually contains an increased proportion of HCl (superacidity).

Hematemesis is a symptom of unequalled clinical significance. On it alone frequently rests a positive diagnosis. When the hemorrhage is considerable, pure blood, more or less clotted, may be ejected, this being highly characteristic of gastric ulcer. Frequently, however, the blood oozes gradually into the stomach and mingles with the gastric juice, and in consequence the oxyhemoglobin of the blood is converted into hematin, the vomitus presenting the appearance of coffee-grounds. On *microscopic examination* under these circumstances only larger or smaller pigment-masses can be seen, and no blood-corpuscles.[1] Vomiting of blood may recur at intervals of a few hours or on each successive day. The *amount* also varies within the widest limits according to the size of the vessel eroded. Some of the effused blood passes through the pylorus, escaping with the feces and giving to the latter a tarry, black appearance. A few cases have been reported in which all the blood was evacuated with the stools except that which was absorbed from the alimentary tract. Either as the result of a single copious hemorrhage or of repeated smaller bleedings a *pronounced anemia* is produced, the objective signs and the cerebral and cardiac manifestations of the latter disease at once becoming evident. As a rule, however, the evidences of anemia are only moderately well marked, and to assume that the anemia is due solely to the hemorrhages would probably be an error. A slight rise of temperature is often observed under these circumstances; this is to be regarded as the so-called *anemic fever*. The pain and the most unpleasant local symptoms have been frequently observed to disappear after its cessation—a circumstance that, as Strümpell observes, may be owing in part to the extreme caution of the patient thereafter. Not infrequently convalescence sets in almost immediately.

Physical signs are few and slight. On *palpation* tenderness is found, though not in all cases. The spot of localized agonizing pain before alluded to is often excessively tender on pressure—a valuable sign. The true gastralgic attacks, so common in gastric ulcer, are at times relieved by making firm pressure with the broad hand over the epigastrium. Near the pyloric end of the stomach palpable tumors may be felt, due to the thickened floor of the ulcer. When these indurated masses become adherent to adjacent organs—the pancreas, for example—epigastric tumors of considerable size may be felt, suggesting the presence of carcinoma. *General symptoms* often do not appear until late in the disease, the patient continuing to look as well as usual. Anemia is usually noted first, to be followed by debility and emaciation; the degree of the general disturbances is in direct proportion to the severity and duration of the causes producing them—namely, the coexisting catarrh, hemorrhages, pain, and vomiting. In some instances the cachexia is pronounced, and the face, on account of the prolonged suffering, assumes a drawn, haggard appearance.

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[1] The blood, however, can be identified by chemical tests and the spectroscopic appearance of the hematin.

Other Clinical Forms.—These have been subdivided into numerous types, some of which merge into one another and cannot be separated clinically. The following atypical forms should be distinguished: (*a*) Latent ulcers, whose existence is not suspected during life, but which are revealed, should they come to autopsy, as open ulcers or cicatrices. (*b*) An explosive form, in which the ulcer may or may not give rise to gastric disturbances prior to the occurrence of perforative peritonitis. (*c*) A recurrent form, described by Welch thus: "In this the symptoms of gastric ulcer disappear, and then follow intervals, often of considerable duration, in which there is apparent cure, but the symptoms return, especially after some indiscretion in the mode of living. This intermittent course may continue for many years. In these cases it is probable either that fresh ulcers form or that the cicatrix of an old ulcer becomes ulcerated."

Complications.—Perforation of the ulcer (most common when it is situated in the anterior wall) leads to peritonitis, which almost always ends fatally. Rarely a localized peritonitis is the result, owing to rapidly formed limiting adhesions or perforation into the lesser peritoneal cavity. The symptoms of this complication will be given in their proper place (see also *Pain*, p. 769). Hemorrhage may prove a serious complicating accident, being in not rare instances an immediate cause of death. I recently saw a case in which hemorrhage caused rapid dissolution.

General Course.—This presents wide variations in different cases. It may be, though seldom, limited to a few hours, as in the explosive form. Innately, the disease is an exceedingly chronic one, often lasting several, and sometimes ten or fifteen, years. Its duration in curable cases may be lessened by proper treatment. Recovery may be incomplete, and the scar resulting from the healing process may give rise to true attacks of gastralgia. Again, if the cicatrices be situated at the pyloric orifice, dilatation will almost invariably develop.

Diagnosis.—The typical cases in which the characteristic symptoms above mentioned are conspicuous are easy of diagnosis. Hemorrhages occurring with gastralgic attacks are almost pathognomonic. A considerable proportion, however, offer formidable difficulties. Without the presence of hematemesis, for example, a positive diagnosis should not be made, and yet this symptom does not appear in 50 per cent. of all cases. In the absence of hemorrhage we may, however, infer the altogether probable existence of ulcer if there be a history of the more important etiologic factors; if there be gastralgia, hyperacidity, local pain and tenderness, a dorsal pain-point; and
symptoms be excited or greatly aggravat

long course and liability to remission, to be followed by exacerbations of the symptoms, are strongly confirmatory.

Differential Diagnosis.—This disease may be mistaken for *gastralgia*, *chronic gastritis*, *the passage of gall-stones*, *cirrhosis of the liver*, and *carcinoma of the stomach*. The differentiation of the latter complaint will be given later. (*a*) In certain cases of cirrhosis of the liver hematemesis is met with, but here there is absence of all the other characteristic symptoms of ulcer, and the presence of a group of symptoms and physical signs pointing to disease of the liver. (*b*) Hepatic colic simulates ulcer of the stomach without hemorrhage. The sudden onset, the longer duration of the attack of pain, its sudden complete cessation, the presence of jaundice and certain physical signs presented by the liver, suffice to distinguish this affection from gastric ulcer. (*c*) Chronic gastric catarrh with hematemesis resembles ulcer of the stomach in many particulars. The great diminution in the proportionate amount of hydrochloric acid found in chronic gastric catarrh and the increased amount in gastric ulcer are facts that will help materially in discriminating these two diseases. When they are associated with one another my observation teaches that there is an excess of HCl present; hence a proportionately diminished amount of HCl probably argues against the presence of ulcer. The vomiting in ulcer is combined with severe paroxysms of pain; not so in chronic gastritis, and the vomit in the former contains larger quantities of blood than in the latter disease. (*d*) Doubtless ulcer of the stomach has often been mistaken for neurotic gastralgia, and the discrimination cannot always be accomplished to a certainty. Their chief differential points may be conveniently arranged thus:

Gastric Ulcer.	Gastralgia.
History of certain occupations, anemia, chlorosis, amenorrhea, tuberculosis, and diseases of the heart common.	History of neurasthenia, neuralgia, and hysteria common.
Most frequent from fifteen to thirty-five years of age.	Most frequent before or near the menopause (in the female).
The paroxysms of pain usually come on at a definite period after eating.	Paroxysms more frequent when the stomach is empty than soon after meals.
Eating rarely relieves pain.	Eating usually brings relief.
Tenderness on pressure over a certain limited area in the epigastrium.	Tender spot absent. General hyperesthesia of the skin often present.
Pressure usually aggravates, and only occasionally relieves patient during paroxysm of pain—not during the intervals between seizures.	Pressure almost always relieves the pain.
In the intervals between the attacks gastric disturbances, more or less severe, are present; also tender point frequently.	In the intervals between attacks no gastric disturbances present, as a rule.
Hematemesis present in nearly one-half of the cases.	Hematemesis absent.
General health often much impaired, particularly late in the affection.	General health less affected than in ulcer.
Physical signs of a mass may be present.	Signs of tumor always absent.
Dilatation may coexist in the late stage.	Dilatation never present.
Hyperacidity of gastric juice usually present.	Hyperacidity present only in certain forms (*supra*).
Improvement follows rest and regulation of diet.	Regulation of diet has no effect.

The **prognosis** is obviously uncertain. The average mortality is about 20 per cent. Such grave complications as free bleedings and peritonitis have been discussed sufficiently in the Clinical History. Among thoracic complications, pneumonia, tuberculosis, and left-sided perforative empyema are those most frequently encountered. They all render recovery almost positively hopeless. The possibility that the resulting scar may cause persistent gastralgia, and the probability that a cicatrix surrounding the whole or any part of the pylorus may cause obstruction at this orifice, followed by dilatation, must be kept in remembrance. Carcinoma may develop in the floor of an old ulcer in subjects who, on account of a predisposition, furnish a suitable soil.

Treatment.—The treatment of gastric ulcer embraces three leading objects: (1) Of paramount importance is *absolute rest for the stomach*. This is to be accomplished by maintaining the recumbent posture in bed, on the one hand, and by rectal feeding, wholly or partly, on the other. This mode of alimentation will be discussed presently. Perfect rest constitutes the best-known safeguard against those serious accidents that intervene suddenly in the course of this affection. It also ensures more rapid cicatrization than any other single agent. The process of repair is very slow under the most favorable circumstances; hence the patient should be informed at the outset that from four to six months, at least, must be spent in bed. (2) *The careful regulation of the diet.* It is not possible for the stomach, when the seat of ulcer, to digest the normal amount of nitrogenous food without being injuriously affected thereby. Those articles of diet should be employed that are digested and assimilated chiefly in the intestinal tract. But, though the patient is fed by the mouth, this should be supplemented by rectal feeding almost from the beginning. By pursuing this combined method and giving per rectum but a limited amount of albuminous food the vital forces can more effectually be supported. Failure to cure cases of gastric ulcer is often due to the fact that but little nourishment is supplied to the system, the patient's general strength being allowed to become exhausted quite early. Frequently the stomach is so irritable as to render it exceedingly difficult to introduce into it even a fractional part of the amount of food necessary to support life properly; and in all cases that I have seen the amount of food that could be taken by the mouth was really inadequate, considering the dietetic requirements of the disease. Should nutrient enemata not be well borne, they may be discontinued until the unpleasant symptoms have subsided, and then resumed immediately. By giving only a portion of the food in this manner rectal feeding may be continued for a long period without disagreeable intestinal symptoms. The following dietary will be found useful: At 7 A. M. give 100 c.cm. (℥iij) of Leube's beef-solution; at 11 A. M., 200 c.cm. (℥vj) of pancreatized milk-gruel;[1] at 3 P. M., 200 c.cm. (℥vj) of peptonized milk or skimmed milk or buttermilk; and at 7 P. M., 200 c.cm. (℥vj) of pancreatized milk-gruel; in addition, the following by rectal injection: at 8 A. M., 6 ounces of pancreatized milk-gruel, with ½ ounce of bovinin and 10 drops of tincture of opium, this to be repeated at 2 P. M. and 8 P. M. If the nutrient enemata must be discontinued for a time, the regular diet must be increased proportionately. If, on the other hand, the stomach rejects the above-mentioned food, then the feeding must be, for a time, exclusively rectal; this is quite practicable if the proper choice be made of nutrient preparations. In addition to the substances before mentioned we may employ from 4 to 6 ounces (150–200 c.cm.) of Leube's beef-solution, or the same amount of defibrinated blood or pancreatized milk with brandy. DaCosta reported recently a number of instances that were cured by a diet of ice-cream. It has been recommended to employ lavage when the stomach is exceedingly irritable, but the use of the stomach-tube is liable to damage the ulcer even in the most careful hands. The good effects from washing out the stomach for uncontrollable vomiting and pain have, however, been frequently witnessed. It may often be satisfactorily accomplished by the use, internally, of 1 pint (½ liter) of warm water containing a few grains of sodium bicarbonate, sipped slowly when the stomach is empty. If at the expiration of two months the condition of the patient indicates that the reparative process is far advanced, then well-boiled rice, stale bread, and potatoes may be allowed; and later eggs, oysters, fish, and sago, the patient not being allowed to resume an ordinary solid diet for at least six months. (3) *The medicinal treatment*, which is altogether subsidiary to the dietetic, has reference to two ends: (*a*) Promotion of the healing process. We cannot be certain that any remedial agents at our command can accomplish this object, and yet it is our duty to attempt it. Of the efficacy of alkaline remedies in this disease we are thoroughly convinced; in neutralizing the hyperacidity of the gastric secretions they fulfil an important indication, since the excess of HCl must have an unfavorable effect upon the ulcer. Of these, sodium bicarbonate (in full doses) or the alkaline purgative mineral waters, as Carlsbad, Kissingen, Hunyadi Janos, are most useful. The Carlsbad salts are also highly beneficial. They may be prepared artificially as follows: sodium sulphate, 50 parts; sodium bicarbonate, 6 parts; sodium chlorid, 3 parts—of which a teaspoonful may be taken in hot water, fasting, in the morning. The preparations of bismuth may be given in combination with antiseptics, which latter are especially to be recommended. Fleiner's method of giving 10 gm. of bismuth in 200 gm. of warm water on an empty stomach, and then allowing the patient to drink several swallows of water, and afterward placing him in the horizontal position with the hips elevated for about an hour, has yielded gratifying results. About 200 gm. of bismuth administered in the above manner usually suffice to effect a cure (Savelieff). Silver nitrate has long enjoyed an enviable reputation in this disease. For the chronic gastric catarrh which is very generally associated with ulcer, silver nitrate, as before stated, is most efficient, and may be combined with small doses of opium or hyoscyamus. The previous general condition of the patient is frequently unfavorable to the successful healing of the ulcers, and to combat the anemia and chlorosis that are often present we may employ iron and arsenic. The albuminate of iron has been warmly recommended, and small doses of Fowler's solution of arsenic are generally well borne by the stomach; the former may also be given hypodermically. When organic cardiac diseases are concomitants they should receive careful attention, and the recognition and treatment of all associated diseases must not be overlooked.

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[1] The milk-gruel is prepared with wheaten flour or arrowroot [illegible] equal quantity of milk.

(*b*) The relief of urgent symptoms. The preceding measures relating to the diet and treatment, and particularly small doses of the extract of opium combined with silver nitrate, often relieve the pain. Mild counter-irritation is also of service, but warm poultices should not be employed. The application of cold to the epigastrium (ice-bag) sometimes alleviates the pain, though quite as often it fails to benefit. For the severe gastralgic attacks morphin should be administered hypodermically.

Vomiting, when not excessive, will be allayed by the use of the agents already mentioned, and bismuth, creasote, silver nitrate, and opium are especially useful; chipped ice, with a small amount of brandy thrown over it, is also of value. When obstinate the following remedies, in small doses, may be tried separately: cerium oxalate, potassium bromid, tincture of iodin, cocain, chloral, and hydrocyanic acid.

For the *hematemesis*, rest, the application of a broad, flat ice-bag, together with the use of ergot hypodermically, will usually suffice. For exhaustive hemorrhages infusion into the veins or into the subcutaneous tissue (hypodermoclysis) of normal salt solution is an important measure. In recurring hematemesis W. L. Rodman advises operation between attacks—always after the third bleeding.

Perforation of the gastric ulcer calls for surgical intervention as soon as the diagnosis is established with any degree of certainty. Nothing more should be given by the mouth. In the cases of perforation which have been operated upon within the first twelve hours during the past three years, 83.78 per cent. have been saved (Tinker). Most of the cases not cured by medical treatment are also savable by operation.

CARCINOMA OF THE STOMACH.

Pathology.—Next to the uterus, the stomach is the most favored seat of carcinoma. In a total of over 30,000 cases studied by Welch, 21.4 per cent. were found to show involvement of this organ. With reference to the parts of the organ most frequently attacked, Welch analyzed 1300 cases with the following results: pyloric region, 791; lesser curvature, 148; cardia, 104; posterior wall, 68; greater curvature, 34; anterior wall, 30; fundus, 19. The forms of gastric carcinoma noted are columnar epithelial (including colloid) and the glandular carcinomata (embracing encephaloid and scirrhous). The epitheliomata grow from the lining epithelium whilst the encephaloid and scirrhous are new growths from the glandular epithelium proper. The last two forms are therefore similar in structure, the differences possibly being due to variations in their growth (rapid in the encephaloid and slow in the scirrhous variety), and to the consequent varying proportion between fibrous tissue and cells; the encephaloid cancers are thus soft, and readily break down on their surface, forming large ulcers that have a clean floor, while the scirrhous cancers are hard and firm. Columnar epitheliomata are frequent, and are situated at the pyloric end of the stomach, where the glands are formed of a single layer of columnar cells on a basement membrane. They are often the seat of colloid degeneration. Squamous epitheliomata occur only at the cardiac end. All the varieties mentioned are prone to produce secondary new growths in adjacent organs, the scirrhous, however, manifesting a less marked tendency to metastasis than the others. They occur either as circumscribed tumors or as a diffuse infiltration, and in the immediate vicinity of the gastric carcinoma there is a marked thickening of the muscularis. Perforation of the stomach-walls occurs in 3.3 per cent. (Brinton). Welch collected 37 cases of secondary carcinoma of the stomach; 17 were secondary to carcinoma of the breast.

Etiology.—The factors bearing upon the etiology of gastric carcinoma may all be regarded as predisposing causes. Of these *age* is the most potent. Of 2038 cases examined by Welch with reference to this point, 75 per cent. occurred between the fortieth and seventieth years, 24.5 per cent. between forty and fifty years, and 30.4 per cent. between fifty and sixty years. According to Lebert the maximum liability lies between the forty-first and sixtieth years. It is comparatively rare under thirty years of age, and almost never occurs during the first decade. *Heredity* stands next to age as a causal factor, though it is far less influential. Welch analyzed 1744 cases, and found that a family history of carcinoma was present in about 14 per cent. *Sex* has little if any influence. The extent to which gastric carcinoma is dependent upon previous disease of the stomach is not definitely determined, but in persons that are predisposed, by reason of age or heredity, the cicatrix of an old ulcer or a pre-existing chronic catarrh of the stomach may become additional causative factors. Strümpell has called renewed attention to the probable relation between gastric ulcer and gastric carcinoma. Schmidt also has recently found the same cell-degenerations around both cancerous and ulcerous growths.

Clinical History.—Prior to the development of gastric carcinoma the symptoms of catarrhal dyspepsia may be present for a variable period of time. The onset, however, is often comparatively abrupt. Again, it may be insidious, and be marked more by the evidences of failing general health and strength than by distinct local subjective symptoms. Osler and McCrae[1] have reported cases of latent carcinoma of the stomach. *Anorexia* is commonly present, though occasionally the appetite remains unimpaired. A sense of oppression, rarely amounting to true cardialgia, and frequent eructations, come on soon after eating. In many cases but little *pain* is complained of, whilst in a lesser number pain is a prominent symptom throughout the entire course of the affection. Its character is very often described as lancinating, less often as burning or gnawing; the latter form of pain is due, most probably, to associated and secondary ulcers. The pain is often referred to the shoulders and the back or loins. *Vomiting* is infrequent, excepting in the more advanced stages of the disease, when it is almost constantly present to a greater or less degree. During the early stages it is due to the catarrhal irritation, later to obstruction. When the latter is at the cardiac orifice, the pain occurs at once after eating; when at the pylorus, it appears several hours after meals. Vomiting may also be caused by the occurrence of fermentation in large accumulations in the stomach. The *vomitus* has few, if any, of the physical characteristics noted in simple ulcer of the stomach. Free hematemesis is very rare; when, however, the surface of the new growth ulcerates, there is almost invariably an occasional slow oozing of blood into the stomach. It is here acted upon by the altered gastric juice, and the black hematin resulting from the transformation of the red hemoglobin gives rise to the well-known "coffee-ground" vomit of carcinoma of the stomach.[1] It is to be recollected that the chocolate-colored appearance of the vomitus is not found alone in carcinoma of the stomach, but may also occur under other abnormal conditions of the stomach-contents.

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[1] *Philadelphia Medical Journal*, February 3, 1900.

The *chemical examination* of the gastric secretions is of prime diagnostic importance, showing as it does the almost constant absence of free HCl after the *test-meal* (see p. 739). Riegel has recently given emphasis to the fact that the presence of free HCl, supposing the examinations to be properly made (by the use of the color test) and sufficiently often repeated, speaks almost positively against carcinoma. In not one of 154 artificial digestive experiments was albumin digested in this disease. Rare cases do, however, occur in which free HCl is present, as when carcinoma of the stomach is secondary to an ulcer, the free acid in the latter being usually increased. More important than the foregoing are those instances in which free HCl is absent, as in carcinoma of the esophagus, extensive amyloid disease, advanced cases of renal disease, and the febrile state. Riegel also noted the absence of free HCl in carcinoma of the duodenum.

Two leading views are held concerning the cause of the failure to find HCl: (1) That it is due to inflammatory degeneration of the mucous membrane, commencing as a catarrhal inflammation and advancing to interstitial change and atrophy (*Rosenheim's view*); (2) that the absence of the acid is due not so much to non-secretion as to its combination with some substance arising from the carcinoma (*Riegel's view*). *Lactic acid* in excess occurs in the stomach in carcinoma. Hemmeter has found lactic acid in 82 per cent. of his cases and Oppler-Boas bacilli in 94 per cent. The *microscopic appearances* of the vomitus and wash-water are in some ways identical with those observed in gastric ulcer, and if they be examined speedily, red blood-corpuscles may rarely be seen. The microscope also, very occasionally, reveals pieces and bits of cancer-tissue, and Kaufmann, Hammerschlag, and others emphasize the frequency of long bacilli. It has been claimed that sarcinæ are present, but Riegel states that they are infrequent. Chronic interstitial gastritis and atrophy of the mucosa may, among other conditions, show symptoms in addition to the absence of HCl.

Physical examination often discloses the presence of a tumor. *Inspection* may reveal an irregular tumor, particularly if the patient be much emaciated. When dilatation exists the outlines of the organ may be seen. On *palpation* the new growth, in a majority of cases, may be felt through the abdominal walls, though often not clearly, as a hard, nodular, and sometimes movable mass. Though this generally appears in the epigastrium, it must be recollected that it depends upon the part involved; also that a tumor united with the wall of the stomach, particularly if situated at the pylorus, sags downward more and more, even to a point below the umbilicus. Less frequently, on account of its mobility, it is discovered in such unlooked-for situations as the right or left hypochondriac region. The varying degree of fulness of the stomach will obviously alter the position of the tumor. When situated at the cardia it is beyond reach of palpation; when attached to the lesser curvature of the stomach or the posterior wall, it is rarely to be felt unless it be of large size. The new growth cannot be definitely made out when it assumes the form of a diffuse infiltration, though it offers increased resistance to the palpating fingers and exhibits more or less tenderness on pressure. Usually the patient lies in the dorsal position during the examination, with the limbs drawn up, breathing regularly while the mouth is kept open. The detection of a tumor when in an unfavorable situation may be facilitated by shifting the patient's position from the dorsal to the lateral, the standing, or the knee-elbow position respectively; at the same time one or two tumblers of some carbonated water should be given with a view to distending the stomach and carrying the tumor downward. Pulsations are frequently communicated from the aorta to the palpating hand through the tumor. If the growth is situated at the lesser curvature, a deep inspiration will often cause the tumor to fall lower, and thus become accessible to palpation. *Percussion* over the seat of the new growth causes a muffled tympanitic resonance; superficial percussion, however, frequently gives absolute dulness.

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[1] *Teichmann's test* for hematin crystals may be employed as follows: Place a drop of the "coffee-ground" material upon the slide and add a few crystals of sodium chlorid. Then introduce a few drops of acetic acid beneath the cover-glass, and on warming hematin crystals will form.

The presence of *metastatic new growths* in the liver and enlargements of the supraclavicular or inguinal lymph-glands are of value in the diagnosis. In one instance that I saw in the Philadelphia Hospital a nodule the size of a walnut protruded from the umbilicus, leading to the suspicion that gastric carcinoma might be present, though the general symptoms pointed strongly to chronic gastric catarrh at the time. Subsequently, however, a round and somewhat nodulated pyloric neoplasm could be readily held in the grasp. In several instances in which the lymph-glands in the groins and in the supraclavicular spaces were the seat of enlargement a probable diagnosis of abdominal carcinoma was made in the absence of positive symptoms and physical signs, and the diagnosis was borne out at the autopsies.

General Symptoms.—Quite early in the disease such evidences of general nutritional disturbance as loss of flesh and anemia may be observed, and, obviously, cases attended with constant anorexia and vomiting will earliest manifest the wasting process. Almost from the beginning the face gradually assumes the cachectic appearance which, in the advanced stages, becomes so characteristic of gastric carcinoma. Anemia soon becomes a prominent feature. There is a waxy pallor of countenance, and the cerebral symptoms as well as the peculiar cardiac murmurs of anemia appear. The blood frequently presents peculiarities that bear a resemblance to those seen in pernicious anemia, and sometimes there is a marked leukocytosis, particularly near the close of life. Osler and McCrae have shown that the number and kind of leukocytes present are of no value in the diagnosis of this condition, and also that the presence or absence of digestive leukocytosis is too uncertain to be of diagnostic importance. The blood-count rarely shows the marked *oligocythemia* seen in progressive pernicious anemia (*vide* Pernicious Anemia). "When any degree of anemia is presented nucleated red corpuscles may be found in dry and stained specimens, and this method of examination may be of much service when an actual blood-count is impossible. The condition is, however, an anemia with wasting, and the layer of panniculus is not retained as in the ordinary forms of pernicious anemia" (Osler). The causes of the profound anemia met with in this affection are not quite plain, since frequently it becomes pronounced before the nutritional disturbances (shown by a loss of flesh) have become marked. The fact that metastatic carcinoma has been found to be abundant in the marrow of the bones is significant in this connection, as pointing to the probable interference, in some instances, with the blood-producing function of the bone-marrow. In advanced cases moderate *edema* of the ankles and of the backs of the hands is frequently observed, and is probably dependent upon excessive anemia. The *temperature* at first shows no abnormalities, as a rule, though after the cachexia has become decided it is often subnormal. Sudden elevations of temperature (103° to 104° F.—40° C.), preceded by rigors and followed by profuse sweating, are rarely observed. The explanation of their occurrence is to be found in the fact that suppuration sometimes takes place in the bases of the cancerous ulcers. The mind almost invariably remains clear to the last, though delirium may be a late-appearing symptom.

Complications.—*Intestinal symptoms* are frequently observed, and *constipation* in particular is quite common. It is apt to alternate with *diarrhea* toward the close of the disease, or diarrhea may in the later stages become a persistent and obstinate symptom. Some of the complicating conditions have reference to the *secondary* new growths. When, as frequently happens, the liver is implicated, *jaundice* is not uncommon, being associated with signs of hepatic enlargement. Indeed, so prominent may be the symptoms and physical signs referable to secondary carcinoma of the liver as entirely to mask the more or less hidden forms of carcinoma of the stomach. The mesenteric and retroperitoneal lymph-glands or the lungs may be the seat of secondary carcinoma, though in these situations it rarely gives rise to characteristic symptoms. Occasionally the new growths spread to the peritoneum and sometimes give rise to ascites. As has been stated under Pathology, perforation may rarely occur, and we then have the pronounced and rapidly supervening symptoms of diffuse peritonitis. *Fistulous communications* between the stomach and the transverse colon or the small intestine—the latter rarely—may also occur. *Nervous symptoms* may be regarded as complicating conditions, and sometimes hasten the fatal termination; the patient becomes somnolent or, rarely, even comatose: the breathing is difficult and the respiration deep and labored. This mode of termination I noted in one of my own cases. Traces of *albumin*, and in the later stages tube-casts, may be present in the urine. An increased quantity of *indican* has frequently been noted; acetone and diacetic acid are present in rare instances.

Atypical Forms.—The disease rarely is entirely latent, and most often in persons previously much enfeebled and in the aged. In other instances the pain is wanting; hence it frequently happens that the presence of cancerous tumors in the stomach is not suspected until accidentally discovered, the symptoms being attributed to less grave conditions. In still other instances the development of a pronounced anemia and of the progressive cachexia alone furnish ground for suspicion.

General Course and Duration.—The course of gastric carcinoma is invariably toward a fatal issue, death usually taking place before the expiration of two years. The average duration of the disease is about one year. According to my own observation, when it occurs in emaciated persons it pursues a slower course than when occurring in fleshy individuals. Carcinoma of the stomach below thirty years of age usually runs a rapid course (Mathieu).

Diagnosis.—A positive diagnosis of gastric carcinoma is easily made when a tumor is demonstrable. It is, however, possible to diagnosticate the disease in the absence of a palpable new growth. The history, the presence of such characteristic symptoms as pain, coffee-ground vomit, the existence of dilatation of the stomach, the constant absence of free hydrochloric acid, the almost constant presence of lactic acid after the Boas test-meal—all occurring in an elderly person, together with the existence of progressive cachexia, are sufficient to warrant a diagnosis. It is needful, however, to exclude the diseases other than gastric carcinoma in which the absence of free hydrochloric acid has been noted. Hemmeter states that the early diagnosis of carcinoma of the stomach is possible in a certain number of cases from histologic examination of small fragments of gastric mucosa, either detached accidentally or removed intentionally. Exploratory laporotomy may be safely advised at the present day if improvement does not follow medical treatment in strongly suspicious cases within a reasonable period.

Differential Diagnosis.—A gastric carcinoma presenting a discernible mass is liable to be mistaken for a *cicatrized ulcer*, for *carcinoma of the pancreas*, of the *transverse colon*, *duodenum*, *omentum*, and the *left lobe of the liver*, as well as for *aneurysm of the abdominal aorta*. The *aneurysmal tumor*, however, is smooth, and is not nodular like the cancerous growth ; moreover, it gives rise to an expansile impulse. In aneurysm the characteristic gastric symptoms and the cachexia are wanting. In *pancreatic carcinoma* the tumor is always fixed; there is an absence of the coffee-ground appearance of the vomit and of dilatation, and free HCl is present in the gastric contents. Further than this, fat may be present in the stools and sugar and fat in the urine. *Carcinoma of the transverse colon and omentum* will be excluded by the presence of such significant symptoms as gastric hemorrhage and the consequent hematemesis, by a chocolate-colored appearance of the vomitus, the permanent absence of HCl, and persistent presence of lactic acid.

Simple round ulcer of the stomach may in cicatrizing give rise to a small tumor, followed by pyloric stenosis and secondary dilatation—an exact counterpart of the course of gastric carcinoma. Great reliance should be placed on the age of the patient, the presence of HCl in the gastric secretions, the grave gastric disturbances, particularly the points of pain (dorsal and epigastric), and localized tenderness with hematemesis, coupled with the longer duration of ulcer (more than two or three years). Ulcer with tumor-like thickening may show an excess of lactic acid, due to associated motor insufficiency, rendering a differential diagnosis exceedingly difficult. Finally, carcinoma may originate in ulcer. Hypertrophic stenosis of the pylorus is also closely simulated.

Simple gastric ulcer and chronic gastritis are often confounded with carcinoma of the stomach. The facts of greatest value in the discrimination of these three affections are so well presented by DaCosta that they are, in the main, here subjoined:

CHRONIC GASTRITIS.	GASTRIC ULCER.	GASTRIC CARCINOMA.
Not confined to any age. More common in middle-aged or elderly people.	May occur in middle-aged persons, but is most frequent in young adults, especially women.	Most common in elderly people; rarely occurs in persons under thirty years of age.
Pain at the epigastrium somewhat augmented by food; soreness is also present. Both are constant, although comparatively slight.	Pain at the epigastrium much augmented by food; subsides when this is digested; paroxysms of pain, not lancinating; strictly localized soreness to touch in epigastrium; sometimes a painful spot over lower dorsal vertebræ. Intermissions in the pain of considerable length are frequent.	Pain frequently of a radiating kind, often paroxysmal, not infrequently severe and lancinating, but not of necessity associated with soreness. Little or not at all affected by food. Pain rarely remits; never intermits for any considerable time.
Symptoms of indigestion marked.	Symptoms of indigestion sometimes very slight.	Symptoms of indigestion marked. Anorexia; extreme acidity of stomach.
Sometimes vomiting.	Vomiting may be present or absent.	Vomiting a very frequent symptom.
No hemorrhage, or but trifling hemorrhage; at most blood-streaks in vomited matter.	Abundant hemorrhage from the stomach common. Stools may contain blood (tarry).	Hemorrhage not very abundant, but frequently occasioning coffee-ground-looking vomit.
Bowels constipated.	Bowels may or may not be constipated; usually are.	Bowels obstinately constipated.
No fever.	No fever.	Intercurrent attacks of slight fever may occur; but temperature often subnormal.
Not much emaciation; no cachectic appearance.	Frequently extreme pallor and debility, especially if preceded by anemia.	Gradual and progressive loss of flesh, and debility; and at times, with the cachexia, hypertrophy of the peripheral lymphatic glands, especially above the clavicles.
Disease may be relieved or cured; is often of very long duration.	Duration uncertain; may get well, may run on rapidly to perforation; on the other hand, may last for years.	Average duration one year; may be shorter, but seldom longer.
No tumor.	Rarely a tumor.	Generally a tumor.
Contents of stomach almost always contain free hydrochloric acid.	Hydrochloric acid in excess in contents of stomach.	No hydrochloric acid in contents of stomach.
No lactic or fatty acids after the rigid Boas test-meal.	No lactic or fatty acids after the rigid Boas test-meal.	Lactic acid present after Boas's test-meal.
No dropsy.	No dropsy.	Edema of ankles often met with.

Treatment.—The diet should receive careful attention, and it will be necessary to adapt it to the peculiarities of the individual case. In general terms, articles of food that are digested and assimilated in the intestines should be employed. After well-mar [illegible] of pyloric obstruction appear we may add greatly to the comfort of the patient by limiting the dietary to liquids, and by predigesting them if they are not otherwise well borne. Should the stomach reject all food, rectal alimentation should be promptly instituted. The medicinal treatment of gastric carcinoma is altogether symptomatic, no remedy with any power over the lesion having been found. The more troublesome symptoms—namely, pain, vomiting, and constipation—are to be met on general principles. Should free hematemesis occur, it should be treated as previously indicated under Gastric Ulcer. The claims that have been advanced in favor of arsenic and other preparations as possessing power to control the progress of gastric carcinoma have not been confirmed by any extended experience. If dilatation coexists, it is to be managed in accordance with the recommendations found under Dilatation of the Stomach (p. 752). Surgical treatment may also be called into service.

Hypertrophic Stenosis of the Pylorus.—By this term is meant pyloric obstruction due to hypertrophy, principally of the circular layer of the muscularis with hyperplasia leading to secondary dilatation of the stomach. This may be (*a*) congenital; (*b*) acquired. The *etiology* is unknown, although spasm of the pylorus has been suggested. The *symptoms* are those of dilatation of the stomach and a pyloric tumor is sometimes palpable. The resemblance to ulcer with tumor-like thickening and to *ulcus carcinomatosum* may be striking (*vide* also p. 781). Congenital atresia proves rapidly fatal, while the adult form may run a long course. Exceptionally other forms of gastric tumor occur—*lipomata*, *sarcomata*, *fibromata*, and *cysts*.

HEMATEMESIS.

HEMATEMESIS is a symptom. Inasmuch as it is due to various causes and diseases other than those of the stomach, it is hardly to be properly classed among gastric affections, and, at all events, is not entitled to more than a brief separate description.

Etiology.—Among the causes of hematemesis are—1. Traumatic injury to the stomach; 2. Diseases of its coats (carcinoma, ulcer, miliary aneurysm of the arteries); 3. A mechanical impediment to the portal circulation; 4. Vicarious menstruation; 5. Alterations in the blood; 6. A disease of some neighboring organ, such as carcinoma of the pancreas, may perforate the gastric coats and open its vessels.

Symptoms and Diagnosis.—If the fact that it is always a symptom, and not the disease itself, be recollected, the importance of recognizing its special causal condition in each instance will be obvious. The manner of its occurrence and the characteristics presented by the blood often give a clue to its nature and origin. Thus, we have seen that the clinical signs in hematemesis due to carcinoma and ulcer of the stomach vary greatly, being almost peculiar to each. This fact must, however, be weighed with the history and symptoms of the case in which it may occur; in this manner, and in this manner only, can errors be avoided. A process of exclusion is the best way to reach a decision. If a careful inquiry determines the absence of morbid lesions of the stomach, such as carcinoma, ulcer, or chronic gastritis, then the other organs of the abdomen, and more particularly the liver, must be examined. If this and the heart be found to be healthy, attention should then be turned toward the state of the blood, since the presence of any specific fever may readily account for the hematemesis. Should the blood present nothing abnormal, it may be found that the menstrual or other habitual discharge has become suppressed.

Differential Diagnosis.—It is to be recollected that the source of the blood may be other than the stomach. Rarely, an abdominal aneurysm bursts into the stomach; occasionally, too, a thoracic aneurysm opens into the esophagus, whence the blood speedily finds its way into the stomach. A careful consideration of the history and of the attending symptoms, together with a thorough physical examination, will, after excluding the various conditions causing true gastric hemorrhage, lead to a correct interpretation of the phenomena. Blood coming from the *throat*, *tonsils*, *mouth*, or the *respiratory organs*, including the nose, is sometimes swallowed, and afterward ejected by vomiting. To discriminate from this condition it is only necessary to make an examination of the lungs and elicit most carefully the history. It must also be recollected that *hysteric females* and *malingerers* have been known to swallow the blood of animals and other dark fluids, and vomit them subsequently. The vomitus may resemble dark blood in appearance when stained by bile or iron or after a free indulgence in wine. The points of contrast between hematemesis and *hemoptysis* have been placed side by side in the following table:

HEMATEMESIS.	HEMOPTYSIS.
The history points to gastric, splenic, hepatic, or cardiac disease.	History of cough and other symptoms points to pulmonary or cardiac disease.
A feeling of uneasiness, and sometimes of nausea or faintness, precedes the hemorrhage.	A feeling of weight and uneasiness in the chest, a saline taste, and a tickling in the throat precede the hemorrhage.
The blood is ejected by vomiting; violent vomiting may excite cough.	The blood is raised by coughing, though, if it be swallowed, vomiting may follow.
The blood is either clotted or fluid and dark; it may be mingled with remnants of food, and is acid in reaction.	The blood is bright-red, frothy, in small coagula, sometimes mixed with mucopus, and alkaline in reaction.

Prognosis.—Hematemesis, except it be due to rupture of an aneurysm, rarely presents a hopeless prognosis. In cases of splenic enlargement, hepatic cirrhosis, or gastric ulcer, it may prove fatal.

The **treatment** has been detailed in the discussion of Gastric Ulcer.

NEUROSES OF THE STOMACH.

NERVOUS DYSPEPSIA.

(*Neurasthenia Gastrica.*)

Definition.—A functional disorder of the stomach, usually characterized by regularly (and sometimes irregularly) recurring attacks of gastric disturbance, followed by almost complete freedom from symptoms. Under the term nervous dyspepsia I shall embrace a general consideration of all the forms of gastric neuroses to be hereafter described. There are no local lesions detectable.

Etiology.—The vast majority of cases occur in highly emotional and hysteric persons, under such exciting conditions as great anxiety, violent passion, dissipation, social excesses, mental over-exertion in business life, grievances, and any startling news or sudden excitement. The condition is most commonly met with in healthy-looking, ruddy-cheeked adults, though it may also occur in the weak and pale-faced. It is more common in females than males. Persons living amid luxurious surroundings suffer most. It is probable that a nervous temperament operates as an underlying cause for an exacerbation of nervous dyspepsia. Gastric neuroses may be of reflex origin in various organic affections, particularly of the nervous system and in irritation of the genito-urinary organs of both sexes.

Symptoms.—The symptoms follow immediately upon the action of the exciting cause and are largely under the influence of the emotions. In the ordinary form the gastric secretions are often normal, and the stomach is found empty after a test-meal within the physiologic time-limit. There is anorexia, which occasionally alternates with a voracious appetite. After meals the patient complains of distress and oppression in the epigastrium; eructations, and an occasional regurgitation of the acid liquid or solid contents of the stomach, with heartburn, will also be noted. Vomiting is not rare, and occurs independently both of the time of eating and of the character of the food. *Gastric peristalsis* is sometimes so well marked as to be readily felt and even visible through the stomach-wall. Kussmaul has called special attention to this symptom, which, I believe, belongs largely to nervous dyspepsia (*vide peristaltic unrest*, p. 788). The increased peristaltic waves, especially under emotion, excite cooing, gurgling sounds that are a source of great annoyance to the patient.

The *physical examination* sometimes reveals abdominal distention and hyperesthesia of the surface, but no localized tenderness, pressure with the broad hand usually affording relief from pain. *Nervous phenomena* always exist, and their correct interpretation is of the utmost importance in the diagnosis. Neurasthenic and hysteric manifestations are commonly associated. The mental condition is unstable and illy regulated, and this fact furnishes a satisfactory explanation of the operation of the etiologic factors. The general health is in many instances not noticeably impaired; in others, particularly in those subject to frequent vomiting and complete anorexia, the general nutrition suffers considerably.

Complications.—The bowels are often constipated, are apt to be distended with gas, and may be the seat of an abnormal peristalsis. The *course* of nervous dyspepsia, in all of its clinical varieties, is chronic, and it may terminate in catarrh of the stomach.

Nervous dyspepsia with hypochondriasis forms a group of cases in which the hypochondriasis may sustain a causal relation; it may, however, be secondary to the gastric disturbances. In either event it is apt to become pronounced after the gastric symptoms have lasted a long time. The symptoms other than the nervous phenomena are quite similar to those previously described.

Diagnosis.—The diagnosis is based on the following points: (*a*) The etiologic factors. Here it is important to ascertain the particular causative influence that produces the gastric symptoms, taking also into consideration any well-recognized predisposing causes. (*b*) The course of the complaint and the absence of some of the physical signs and symptoms that would point positively to anatomic lesions of the stomach. When there is a catarrhal process, the symptoms become more pronounced immediately after taking food than in neurasthenia gastrica. The influence of the ingestion of indigestible substances upon sympathetic dyspepsia is often to relieve, or is of neutral effect, whereas in catarrhal indigestion it decidely aggravates the condition. The dull pain after eating and the tenderness on pressure are usually more marked in the catarrhal variety, and the stomach contains large amounts of mucus. The symptoms of the latter do not intermit from time to time, as in nervous dyspepsia, but are more constantly present. The analysis of the stomach-contents obtained after a test-breakfast will also assist in the diagnosis. The gastric contents in cases of nervous dyspepsia are often found about normal, though any abnormality (even to complete achlorhydria) may be present (*vide* Special Forms of Gastric Neuroses, p. 786).

Prognosis.—If there be an absence of an inherited predisposition, and if the cause is removable, complete recovery may be prognosticated. In a neurotic constitution, however, the tendency to recurrence, even after a decided improvement has taken place, is very strong. The most unpromising cases are those in which the cause is irremovable, though as to life the prognosis is not unfavorable. The patient himself is always of the opinion that he is suffering from a serious and incurable affection.

Treatment.—Every causal factor must be recognized and mitigated or removed. If we fail to accomplish this end, our efforts at cure will be unsuccessful. The dietary should be generous and composed of highly nutritious articles of food, and to convince the patient that his stomach is capable of digesting a full meal is the first duty of the physician, though the task is confessedly difficult. So soon as the patient realizes the truth in reference to his digestive capacity his sufferings are largely at an end. It is the nervous system that demands especial attention, and the internal treatment of the stomach is merely placeboic. Nerve-tonics combined with nerve-stimulants are often serviceable.

A change of air from the city to the country, the mountains, or the sea-coast is usually followed by improvement. In some manner the patient must be extricated from the old surroundings under the influence of which the disease was started and has continued. Sea air has seemed to me to be more serviceable than mountain air in these cases, though I believe it to be an axiom in climatic therapeutics that the latter confers more lasting benefits than the former. These patients are often averse to taking exercise, but so great is the value of this sanitary measure that it should never be overlooked. Cold sponging of the surface, followed by friction to the skin, should be practised daily for its effect upon the skin-circulation and the nervous system. Occasional lavage, hot and cold douches, electricity (intra- and extra-gastric), and gastric massage, may all be tried, and may prove of distinct advantage. In highly neurotic and hysteric females the S. Weir Mitchell treatment is often attended with good results. The hypochondriac form is often intractable. Strychnin, however, if perseveringly used, and if coupled with a change of air, often proves beneficial. One of the most obstinate examples of this nature that I have ever seen occurred in a retired merchant living in Philadelphia. This man was finally cured in consequence of his own suggestion, resulting in his removal to the country and engaging in farming.

SPECIAL FORMS OF GASTRIC NEUROSES, CHARACTERIZED BY MARKED AND PECULIAR ANOMALIES OF SENSATION, MOTILITY, AND SECRETION.

NEUROSES OF SECRETION.

HYPERCHLORHYDRIA.

(*Hyperacidity.*)

Definition.—An augmentation of the secretory function of the stomach during the digestive period, resulting in the presence of an excessive amount of hydrochloric acid.

Etiology.—Hyperacidity is common during digestion, and in most instances is due to some one of the causative influences mentioned under Nervous Dyspepsia (grief, great anxiety, mental over-taxation). The disease is common among the professional classes, and it affects men oftener than women. Highly-seasoned foods and alcoholic intoxicants may occasion the condition.

Symptoms.—Hyperchlorhydria may be *continuous*, though more often it is *discontinuous* and lasts from a few hours to several days. After the periodic form has lasted a long time it may gradually become a permanent condition. The patient at first complains of *uneasiness* in the epigastrium one or two hours after meals. Later, this amounts to pain of moderate intensity, and soon follows every meal after a like interval. The duration of the pain is from one to three hours. Acid eructations are frequently noted. The increase of hydrochloric acid interferes with the digestion of starches, and thus tends to increase the pain. On the other hand, however, a diet composed of albuminoids often affords relief, and the salts of the alkalies also ease the pain. Associated *nervous symptoms* (headache, dizziness) are often observed, though the bodily nutrition is usually well maintained. *Palpation* of the epigastrium may show a diffused tenderness. Evidences of moderate dilatation of the stomach sometimes appear, and splashing sounds may be detectable.

Diagnosis.—Though the diagnosis of hyperacidity is made probable by the above-mentioned symptoms, it is rendered certain only by a repeated analysis of the gastric contents. The findings, according to Einhorn, are—(1) On examination of the stomach in the fasting condition, the organ either is found empty or contains only a few cubic centimeters of juice; (2) one hour after Ewald's test-breakfast the hyperacidity is greatly increased, owing to the great amount of free HCl.

Gastric ulcer must be eliminated. In this disease the pain is aggravated immediately after eating, and is not relieved by albuminous food, nor by large doses of alkalies as in hyperchlorhydria. In ulcer, moreover, the pain often leads to vomiting, and severe, painful attacks frequently occur at night.

Gastro-succorrhœa (*Reichmann*); **Gastroxynsis** (*Rossbach*).—In this affection there is an increase of hydrochloric acid, either constantly or intermittently, when no food is present. An *epigastric gnawing pain* and nausea appear in the full bloom of health. The *nausea* soon results in the *vomiting* of enormous quantities of gastric contents. The *appetite* is lost, but the thirst is excessive, and the amount of drink taken and of liquid vomited are proportional. During the night or in the early morning hours the patient commonly vomits large amounts of a clear or bile-tinted liquid containing hydrochloric acid and the gastric ferments in excess. This may be followed by persistent vomiting, attended with much retching. After a lapse of a few hours the ejection of a large quantity of highly acid liquid may be repeated. The *pain* often becomes intense, headache is common, and a tendency to collapse is usually marked. The attacks last, as a rule, about two or three days, when they quite abruptly give place to apparent good health. *Recurrence* at the end of periods ranging from a few months to a year or more are common. A physiologic form has been advanced.

The *diagnosis* is made upon the presence of the clinical symptoms and course, as well as upon the results of oft-repeated analyses of the vomitus. Gastric ulcer and certain organic spinal and cerebral nervous affections, in which there is excessive gastric secretion, must be excluded before an absolute diagnosis can be made.

Gastro-succorrhœa Continua Chronica.—Reichmann first described a condition characterized by a *constant secretion of gastric juice* either in the absence or presence of food. The *symptoms* are much the same as those in *hyperacidity*, but tend to become continuous, so that the vomiting finally becomes a daily occurrence. In the fasting state a highly acid secretion that contains no food-particles flows through the tube from the stomach. Albuminoids are rapidly and starches slowly digested by these patients, as is shown by an examination of the gastric contents three or four hours after the Leube-Riegel's test-meal (one plate of soup—400 c.c.—a large portion of meat, some potatoes, and a roll). The disease is quite rare, and must not be confounded with the organic diseases to which continuous gastric succorrhea may be secondary and upon which it is dependent. Schreiber, Boas, and others believe that this is almost always a symptom of gastric atony or gastric ulcer. The occurrence of hematemesis or melena would favor ulcer.

Leube has described a neurosis in which there is a constant **subacidity of the secretion.**

Achylia Gastrica (*Einhorn*).—The suspension of the gastric secretions may result either from atrophy of the stomach glandules or from a nervous derangement of secretion. The condition has been mistaken for malignant disease of the stomach. Lactic acid, however, is not present in excess. Achylia gastrica may cause chronic lienteric diarrhea (A. A. Jones).

The **prognosis** in the foregoing affections is not bad as to life, and not infrequently a cure, even, can be effected.

Treatment.—The dietary of the neuroses of secretion embraces principally nitrogenous articles of food. Einhorn advises three large and two small meals daily. The two smaller meals should consist of a glass of milk with bread and butter, or like fare. Acids and spirits—substances that excite the glands of the stomach—must be excluded. The medicinal treatment should, in addition to meeting the general neurotic condition, consist of full doses of sodium bicarbonate. In some cases more active alkalies than sodium bicarbonate may be needful—*e.g.* magnesium and sodium salicylate, either separately or in combination. Lavage daily, before the chief meal, is also sometimes beneficial. Lemoine advises hydrotherapy and rest to strengthen the nervous system.

NEUROSES OF MOTILITY.

INCREASED PERISTALSIS OF THE STOMACH.

Gastric peristalsis is increased in various conditions, which will be considered *seriatim*, though briefly.

(*a*) **Belching and Eructations.**—These may be of *nervous origin* and are met with generally in *hysteric* subjects, and less frequently in *neurasthenics*. The air is swallowed, and then expelled with more or less noise, owing to an increased contractility of the stomach. The gas is *odorless*, and differs in this point from the gases of fermentative dyspepsia. Epigastric distress and distention often arise, and certain nervous phenomena, as anxiety or palpitation, may coexist. It must not be forgotten, moreover, that in hysteric subjects the belching may be from the esophagus alone.

(*b*) **Pyrosis** means regurgitation of the acid contents of the stomach.

(*c*) **Rumination** (*Merycism*).—A rare affection in which the food is regurgitated into the mouth, the cud chewed, and again swallowed after the fashion of ruminants.

(*d*) **Nervous Vomiting.**—This is a *reflex neurosis* that may affect persons of any age, though most frequently it is seen in adult females with an hysteric tendency. Without previous nausea, and independently of the character of the food taken, the contents of the stomach are readily expelled or, more correctly speaking, regurgitated into the mouth, and then expectorated. Though this usually takes place after meals, it may occur without reference to meal-time—a feature that indicates its nervous origin. The *attacks* of vomiting are separated by longer or shorter intervals of excellent health. Periodic vomiting may also occur independently of hysteria or other nervous affections, as pointed out by Leube. The *course* is rarely unfavorable.

(*e*) **Peristaltic unrest** (Kussmaul). or **spasm of the stomach,** has been referred to under Nervous Dyspepsia. It has also been observed in compensatory hypertrophy of the stomach-wall following pyloric stricture. In a case of gastric carcinoma in my own care the supermotility of the stomach caused an almost immediate expulsion of the gastric contents, and even of the rigid test-meal at certain times.

(*f*) **Cardiospasm.**—By this term is meant a painful cramp of the cardia. Two forms are distinguished: (*a*) acute cramp (of brief duration); (*b*) chronic cramp (exceedingly rare). Among causes are neurasthenia, hysteria, and local irritation (thermal, mechanical). Chronic spasm may lead to complete atresia of the cardia, and is a distressing affection. In acute cardiospasm the attacks often tend to recur.

(*g*) **Pylorospasm.**—Cramp of the ring-musculature of the pyloris may be *primary* or *secondary*. The latter is due to intense local irritation (superacidity, hypersecretion, excess of organic acids). The painful spasm in the pyloric region induces stagnation of the ingesta, followed by atony of the stomach and consequent dilation.

Treatment.—To the regimenal management, including a hygienic mode of living, the attention of the physician should be primarily directed. The medicinal treatment is to be aimed at the causal or primary nervous affection. The valerianates and the bromids often do good service. For the cramp of the cardia and pylorus belladonnæ or codeine are efficient.

DIMINISHED PERISTALSIS OF THE STOMACH.

(*Atony.*)

(*a*) **Pyloric Relaxation or Incompetency.**—This is a rare neurosis that allows the partially digested gastric contents to pass the portals of the stomach prematurely. It likewise permits the regurgitation of the contents of the duodenum into the stomach. Its recognition is possible upon inflating the stomach, when gas may be seen to pass into the intestines, and also (even with greater certainty) upon the regurgitation of intestinal contents into the stomach.

(*b*) **Insufficiency of the Cardia.**—This condition leads to eructations and regurgitations, and when these are of aggravated form they impair the general nutrition. Ordinarily no ill-effects follow.

(*c*) **Atonic Dyspepsia** (*Atony*).—This may occur as a neurosis, though oftener it is secondary to chronic gastritis. It implies *hypomotility* or insufficiency. The *chyme* is retained in the stomach beyond the natural time-limit. There is an *epigastric oppression* with a distention of the organ during digestion that tends to become permanent. There are eructations of gas, an impaired appetite, and often constipation. The stomach is found empty in the morning, and six or seven hours after Leube's test-meal it contains some chyme. In the absence of pyloric stricture the hypomotility may be shown by the administration of salol.

Treatment.—The diet is to be regulated as in chronic gastritis with dilation. It is rarely necessary to restrict the solids to any marked extent, but the quantity of fluids should be lessened. The patient must be taught to eat slowly and masticate thoroughly. His hygienic standard of living must be high, and he must not be allowed to over-use his mental faculties. Exercise in the open air and cold baths, properly regulated, are potent for good. Of medicines, strychnin stands first, and I have found the following formula of great service:

℞. Tr. nus vomicæ, f ʒiiss (10.0);
Inf. cascarillæ, q. s. ad f ℥iv (128.0).—M.
Sig. ʒij (8.0) three times daily.

Electricity is also indicated, and it is in these cases that intragastric faradization has given excellent results. The constipation is to be overcome by an appropriate dietary (green vegetables, Graham bread, an abundance of fruit). Lavage deserves a prudent trial.

NEUROSES OF SENSATION.

CARDIALGIA.

(*Gastralgia; Gastrodynia.*)

Definition.—Severe paroxysmal pain in the epigastrium in the absence of gastric lesions. There are two other forms of this disease that are clinically identical with gastralgia, the one occurring in ulcer and carcinoma of the stomach, and the other in certain chronic nervous diseases, forming the so-called gastric crises, which have been considered elsewhere.

Etiology.—The subjects are often hereditarily predisposed to neuroses of other types. Such conditions as anemia, exhaustion from repeated hemorrhages, and syphilis exert a *predisposing* influence. The female sex is more liable than the male, and in the former it appears to be dependent upon disturbances of the menstrual function or quite frequently upon hysteric conditions. It is sometimes excited by reflex irritation, by deep grief, worry, and great anxiety. Hypochondriasis and hyperacidity are also among its frequent causes.

Symptoms.—These are *sudden in their onset* as a rule, and quite characteristic. Occasionally the attack is preceded by anorexia, or it may begin with a sense of oppression and distention in the epigastrium, lasting for a few minutes. In any event, the onset of the attack proper is marked by *agonizing pains* in the epigastrium, that dart through to the back, and at times also pass around the lower ribs. The seizure lasts from a few minutes to an hour or two, and terminates with *eructations of gas*, or, less frequently, with vomiting. From the nature of the causative factors it is obvious that the gastralgic seizures are in no wise dependent upon the character of the food taken; hence the fact that they occur more frequently when the stomach is empty need occasion no surprise. Firm pressure over the epigastrium relieves the pain. *Nervous phenomena*, varying with the etiology of individual cases, are constant attendants, but cannot be detailed here. A distinct clinical variety is found associated with that form of nervous dyspepsia in which an excess of HCl is secreted (*vide* Hyperacidity); this occurs at varying intervals. Many purely functional nervous disturbances are thus subject to the law of periodicity. I believe that a very small percentage of cases are caused by malaria, since I have met with two such cases in a malarial district, both of which yielded readily to quinin. The disease took on a desultory, periodic character, and was associated with other malarial symptoms.

Diagnosis.—The history, the absence of any local causes, together with the characteristic gastralgic attacks and their time of occurrence, will render a positive diagnosis easy in most instances. The gastric crises that occur in locomotor ataxia must be excluded. Cholelithiasis may simulate gastralgia (*vide* p. 859). To discriminate this condition from *gastric ulcer* is sometimes difficult, but stress has been laid upon the differential points in the description of the latter disease (*vide* p. 772).

Prognosis.—This depends entirely upon the causal condition. The disease itself has no intrinsic fatal tendency.

Treatment.—This is to be subdivided into (*a*) the treatment of the attack; (*b*) the management of the intervals between the seizures. The pain is, as a rule, sufficiently intense to demand morphin, which is best administered hypodermically in combination with atropin. This should not, however, be repeated unless urgently needed. In mild attacks the constant or the faradic current often affords prompt relief. Under these circumstances counter-irritation, together with the internal use of Hoffman's anodyne or chloroform in small doses, may relieve the pain.

(*b*) *The Management of the Intervals.*—Here the physician's efforts should be directed to the detection of the causes and their removal by appropriate means. In hysteric females I have obtained good results from the prolonged use of the valerianates, combining with them iron and arsenic, thus:

℞. Zinci valerianat.,	gr. xviij (1.16);
Quininæ valerianat.,	gr. xxvij (1.74);
Ferri arseniat.,	gr. ij (0.129).

M. et ft. pil. No. xviij.
Sig. One after each meal.

A change of air is often highly serviceable, and should be advised whenever financial considerations permit. These patients are constantly in a more or less exhausted, anemic, and run-down condition, and a tonic plan of treatment is always indicated. In the intervals between the attacks digestion, as before stated, proceeds normally, and the stomach, therefore, requires no treatment. Constipation, if present is a condition demanding relief, not, however, by the use of purgatives, but by such means as massage, a suitable diet, enemata, or laxative suppositories. The physician must carefully regulate the sanitary particulars of the patient's daily life.

HYPERESTHESIA OF THE STOMACH.

This is met with in functional and organic diseases, as well as in chronic gastric catarrh and other affections of the stomach. Again, it may occur as a *neurosis*, most frequently in chlorotic girls and women. There is an increased gastric sensibility, so that the mildest irritant produces *painful sensations* that may be either gnawing or burning in character. A feeling of fulness and nausea are among the common features of the complaint. Food and certain articles that are not easily digestible may afford relief, and, oppositely, fasting or restriction of diet may aggravate the condition. The complaint, however, is often aggravated during digestion, particularly after excessive indulgence in certain kinds of food (crabs, lobsters, oysters, strawberries). Cutaneous symptoms, as erythema and urticaria, may appear. *Hypochondriasis*, neurasthenia, and hysteria are often associated. The above sympto [illegible] endent upon an individual idiosyncrasy.

Treatment.—At first a restriction of the diet to soft and liquid articles should be tried, and later a cautious return to solid food is to be made. Of medicaments, the bromids, given for a period of two or three months, have given the best results in my own hands. For the chlorotic type iron in the form of Blaud's pill, in ascending doses, is the best treatment.

ANOREXIA.

This consists merely in a loss of appetite, and occurs in many organic gastric disorders. It may also be a primary gastric *neurosis*, the latter being often associated with gastric hyperesthesia. Anorexia sometimes leads to a repugnance to food and a degree of abstinence that may induce grave nutritional disturbance. Among exciting causes mental shock of any sort ranks first. In other instances the patient may experience hunger, but on attempting to eat *anorexia* quickly develops. The recognition of anorexia as a primary neurosis of the stomach is difficult in the extreme after the general nutrition has become seriously impaired. Chronic dyspepsia, phthisis, and other diseases associated with emaciation and debility must be excluded before the diagnosis is established.

HYPERORBXIA.

(*Excessive Appetite.*)

This may either be symptomatic of other affections (*e. g.* diabetes mellitus) or it may be a gastric neurosis. It may also be paroxysmal (*bulimia*). The patient complains of burning sensations in the epigastric region and of an insatiable hunger. The symptoms of neurasthenia and hysteria are often in association. The local and general symptoms are relieved by food. It may also accompany other nervous disorders, as affections of the brain, exophthalmos, and migraine.

In bulimia the abnormal sensation of hunger may come on at any hour, even immediately after abundant food has been taken. When the morbid sensation of hunger develops more gradually and some time after meals it is spoken of as *polyphagia.*

Pica is the term applied to the craving for substances not used as food (slate-pencils, dirt, chalk).

Malacia represents the desire for highly spiced dishes (mustard, salads, pickles, fruits).

The above conditions are met with in neurasthenia, chronic gastric affections, and chlorosis.

VIII. DISEASES OF THE INTESTINES.

METHODS OF DIAGNOSIS.

Examination of the Feces.—Although the results are in most cases unsatisfactory, an examination of the feces should not be neglected, especially in the more serious affections of the intestine. This embraces—(*a*) a macroscopic; (*b*) a microscopic; (*c*) a chemical; and (*d*) a bacteriologic examination.

(*a*) The *macroscopic* appearances often suffice. A thorough inspection of the stools, a matter too often omitted, furnishes valuable points in regard to the presence or absence of coarse parasites, fragments of tumor, foreign bodies, concretions, blood, bile, pigment, fat, pus, mucus, undigested meat, and the like.

The shape, color, and consistence of the stools must be noted, and it is to be remembered that in these particulars, as well as regards their frequency, they exhibit a considerable range of normal variations, according to individual peculiarities, the character of food taken, and so on. It is to be recollected that normal stools contain fat in varying amounts, for the reason that only a limited quantity can be emulsified and taken up from the intestine. The naked eye may, at times, detect its presence from the "peculiar silvery appearance" of the feces. Fat in the stools (*steatorrhea*) is often pathologic, and the separate affections in which it is met with will be considered hereafter. The dejecta present a shining, tallowy appearance, either throughout or in circumscribed spots. Again, the fat may occur in the form of oil floating on the surface of liquid stools. Mucus is also visible, either as slimy or jelly-like masses, or as shreds and granules (sago-grains). Diarrheal stools should be examined macroscopically with great care for gross admixtures (flakes of casein, bits of meat, etc.). Constipational dejections often assume a rounded form (*sheep's dung*) on account of their delay in the large bowel. They may attain to the size of an orange, and may be, though rarely, enveloped in mucus or blood-streaked. Their color is dark. On the other hand, the stools may be colorless in cases in which the bile-ducts are occluded; these usually contain a large proportion of fat, though not invariably. The effect of certain drugs upon the color of the stools is to be borne in mind. When blood is intimately mingled with the feces, they have a reddish, dark- or blackish-brown (tarry) color, according to the quantity and the time allowed for decomposition in the intestine. Blood, either clotted or fluid, may also be passed in a pure state. Its source is usually the lower bowel, though when peristalsis is augmented, it may come from the small intestine, as in typhoid fever. Pus may occasionally be recognized macroscopically. From a diagnostic point of view, it is most important to examine for biliary concretions in doubtful abdominal colic. "For the detection of small concretions the stools should be passed through a sieve; large concretions are easily recognizable" (Ewald).

(*b*) *Microscopic Examination.*—Diarrheal stools can be examined as discharged, but to solid and mushy dejections a solution of common salt (½ per cent.) should be added and all hard masses thoroughly broken up. Different portions of the stools are to be selected for microscopic examination. *Microscopically* we are enabled to detect the eggs of parasites, pus, blood, protozoa, mucus in the form of shining, vitreous, homogeneous, or whitish masses; and in the interior of the latter certain pathogenic bacteria, various crystals, and intestinal epithelium may be seen. Remnants of vegetable food may stimulate mucous islets, but the former strike a blue color on the application of the potassium-iodid test. Microscopically, diarrheal stools show undigested muscle-fibers, fat-crystals, vegetable cells, starchy granules, and innumerable bacteria. Undissolved starch in even moderate quantity points to catarrhal enteritis of the small intestine. On microscopic examination of the dejections in constipation we find "a copious detritus of brown or black color, usually numerous colorless or slightly tinged triple phosphates (phosphate of ammonium and magnesium crystallizing in the form of a coffin-lid), or, more sparse, crystals of neutral phosphate of lime." Seldom do we meet with the rhomboid plates of cholesterin, which are recognized in that they are colored from a reddish-brown to violet by dilute sulphuric acid (1 : 5), and become blue or green on the further addition of a solution of iodin. Needle-shaped crystals of fat, single and also in the forms of tufts, are frequently met in obstruction of the biliary ducts. Bile-pigment cannot be detected. Remnants of food are sparsely present in normal feces. Epithelium from the mucous membrane, pus-cells, and blood-corpsucles, unless they come from the passage of the fecal mass through the anus (in which case they are simply adherent to the external surface of the scybala and are but little changed), are greatly altered; they are fatty, degenerated, shrunken, and hardly recognizable. Rhomboid crystals of hematoidin may be at times observed. The microscopic examination for animal parasites will be referred to in appropriate sections of this work.

(*c*) *Chemical Examination.*—The presence of bile-pigment is easily detected by the Gmelin reaction. The stools must, if needful, be rendered fluid by the addition of water, then filtered, and the filtrate allowed to dry. At the margin of the drop the characteristic green color will appear. Urobilin strikes a red color. The stools in diarrhea may contain ferments capable of digesting albuminoids. The fatty acids are distinguished from fatty soaps by the solubility of the former in ether.

To detect a very small amount of blood that may be intimately mixed with the feces the test of Fr. Müller and Weber gives the most reliable results. Solid or mushy stools are first rubbed up with water and filtered. A portion of the filtrate of liquid feces is added to 5 cubic centimeters of glacial acetic acid and ether, and the whole well shaken. The ether generally settles clear, but if it does not a few drops of absolute alchohol are to be added. The presence of blood (hematin acetate) is shown by a reddish-brown tint given to the layer of ether. The indol-reaction may be increased, pointing to increased intestinal putrefaction.

(*d*) *A bacterial examination* of the intestinal contents, and particularly of any mucus or muco-pus that may be discharged, may decide the diagnosis of certain intestinal disorders (tuberculosis, amebic dysentery). For the method of carrying on these investigations the reader is referred to special works on diagnosis and bacteriology.

Physical or External Examination.—*Inspection.*—This should be made with the patient in the dorsal position and with proper illumination. Localized prominences are to be noted, though the fact should be remembered that these may be simulated by localized contractions of the various abdominal muscles. The influence of respiration on these circumscribed bulgings is also to be observed. In the absence of unusual distention of the abdominal walls it is of great value to inflate the large intestine with air *per rectum*, and to note the progressive distention of the intestinal coils as a means of detecting obstructing lesions in the bowel; the position and mobility of a tumor should also be noted. It is often of marked aid to inspect the mucosa of the rectum by the use of approved specula. The volume of the abdomen may be diminished or even "scaphoid." Abnormal peristalsis may rarely be noted (important if associated with distention).

Palpation.—This is of first importance. The patient should occupy the dorsal decubitus, with the head raised, the thighs drawn up, and the mouth open, so as to relax the abdominal muscles. Something may be gained in this direction by distracting the patient's attention. I have found that placing the patient in the lateral decubitus, with the thighs flexed on the abdomen, to be the most satisfactory way of determining the degree of mobility of certain tumors. The examiner should not fail to remember the knee-elbow position in cases in which it is desired to palpate the parts occupying the bottom of the pelvic cavity and all deep-seated, movable growths. In certain cases relaxation of the abdominal muscles is only obtainable by anesthetizing the patient, and I do not hesitate to do this in cases in which the diagnosis is important. In palpating the abdomen for abnormal conditions we must keep in mind steadily the relations of the different parts of the intestines, and also that the latter may vary considerably in position—a fact particularly true of the transverse colon (*vide* Enteroptosis). In this connection Ewald's statement "that abnormally situated organs or neoplasms of parts other than the intestines will, under the pressure of the intestines filled with air or water, return to the position that the organ normally occupies," should be emphasized. New growths of the pancreas, of the spinal column, or of the pelvis, and retroperitoneal tumors will remain fixed. Palpation may detect pathologic peristalsis, and increased resistance if the coats are thickened (as in chronic stenosis). Tenderness, localized or diffuse, is to be noted, if present, as well as peritoneal friction. The rectum may be palpated if the symptoms point to disease of that organ.

The palpation of pathologic conditions of the intestines will be considered in connection with the separate intestinal affections.

Percussion detects a fluid effusion either in the general peritoneal cavity, the position varying with the position of the body, or in circumscribed localities; the latter must not be confounded with areas of dulness that are occasioned by splenic and hepatic enlargements, solid new-growths, or abscesses. Air in the peritoneal cavity (*meteorismus peritonei*) generally gives a pure tympanitic note, though if the tension be very strong, a non-tympanitic tone may be elicited. These sounds are general, even extending up to the fifth or fourth rib, and hence they cover the regions of the spleen and liver. The best results when the abdomen is not tense, however, are obtained after inflation of the large intestine with air. The pitch of the tympanitic note becomes elevated with increase in the tension of the gut; it falls with relaxation of the bowel. Hence the large cannot always be told from the small intestine by the percussion-note.

Auscultation.—Noises are often audible either at a distance or by means of a stethoscope applied to the abdomen. They are sometimes occasioned by the natural peristaltic movements or by certain voluntary or involuntary spasms of the abdominal muscle. I have repeatedly confirmed the observation of Ewald, who frequently found in those suffering with chronic intestinal indigestion a swashing or splashing noise, sounding as though air and water were being forced through a narrow space in the ileo-cecal region. These sounds may rarely be found in healthy persons. Similar noises sometimes have their seat in the descending colon, particularly if the bowel is unnaturally dilated by air or fluid. They are often audible prior to an evacuation in cases of colitis. Noises may also originate in the transverse colon, and to discriminate these it is necessary to empty the stomach if we would avoid confusion with identical gastric sounds. Direct auscultation of the intestines renders audible the peristaltic movements, and the absence of the latter indicates paralysis of the intestine, which may be local or general. Friction-sounds may be audible when inflammatory exudates are present. When obstruction of the large intestine is suspected, auscultation should be practised while air is being forced into the rectum, inasmuch as the degree of permeability can be thus determined. Metallic tinkling and amphoric noises may be audible, particularly on making auscultatory pecussion, but these are without real diagnostic value.

ENTEROPTOSIS.

Definition.—The descent of the intestines from their normal position. The condition occurs coincidently with gastroptosis, nephroptosis, and prolapse of other viscera, constituting splanchnoptosis (Glénard's disease).

Etiology.—It is linked with gastroptosis and other forms of ptosis by common etiologic influences, such as sex (being most common in females), tight-lacing, traumatism, muscular strain, numerous pregnancies, rapid emaciation, and probably the wrong use of cathartics. Either the small intestine alone or the large, or both, may be involved. Prolapse of the colon (coloptosis) is the more common, and, according to C. Meinert, is even more frequent than gastroptosis. Lying immediately above the symphysis pubis, it is sometimes elongated and tortuous—"S- or M-shaped."

Symptoms.—The condition, even when pronounced, may exist without symptoms. On the other hand, in the majority of instances the intestinal, gastric, and other bodily functions are disturbed, and yet enteroptosis is usually overlooked. Chief among the intestinal symptoms is *excessive flatulence;* not rarely, also, there is membranous enteritis, the latter probably being due to the flexures that produce an arrest of fecal masses, and this in turn causing inflammation (Boas). *Constipation* generally prevails, and sometimes alternates with diarrhea. The symptoms of gastroptosis and nephroptosis are often associated; they are loss of flesh and nervous symptoms, and the latter may simulate those of neurasthenia or hysteria.

The **diagnosis** is made upon the afore-mentioned points and upon the results of a careful physical examination. The position of the colon may be determined by inflation with air or gas. Again, after the injection of water (f℥viss–ixss—200–300 c.cm.) a splashing sound is audible; this is double the amount of water required in the normal condition. Glénard has pointed out that a transverse cord (which he believes to be the colon) can be felt in the upper part of the abdomen. Boas and Ziemssen assert that this cord is the pancreas, rendered palpable by the sinking of the stomach.

Treatment.—The bowels must be moved regularly, the tonicity of the abdominal walls must be increased by electricity, massage, and hydrotherapy, and in strongly nervous cases the treatment of neurasthenia, including the Weir Mitchell rest-cure, must be instituted. Supporting bandages have been found serviceable. The medicinal treatment aims at meeting certain symptomatic indications, such as flatulence and fermentation.

INTESTINAL CATARRH.

(*Catarrhal Enteritis; Muco-enteritis.*)

Definition.—A catarrhal inflammation of the mucous membrane of the whole or of any anatomic division of the intestinal tract. It may be either acute or chronic, primary or secondary. The chronic variety occurs less frequently than its counterpart, chronic gastritis, particularly in adult life.

Pathology.—The morbid lesions of the acute variety do not differ essentially from those met with in catarrhal inflammation of any other mucous membrane. The first stage is characterized by swelling and dryness of the mucosa; this is soon followed by a copious exudation of mucus, and more rarely of pus, which bathes the membrane more or less completely. After an abundant secretion is poured out the membrane appears rather pale, though the tips of the valvulæ conniventes in the small intestines may appear reddened. The solitary and agminated glands, as well as Peyer's patches, may stand out prominently, owing to their corrugated condition (*follicular enteritis*). The apices of the solitary glands often undergo a necrotic change, thus forming follicular ulcers. The remainder of the mucosa may also be the seat of rather extensive areas of superficial erosion, though this must not be confounded with postmortem softening of the epithelium. In some cases the desquamation of epithelium is more pronounced than the abnormal mucous secretion. In *chronic* intestinal catarrh the mucosa presents a slaty hue, with a more or less dark pigmentation of the villi and follicles; it is in most instances thickened, owing to an increase in its connective-tissue elements. In a smaller number of cases it is thinned, particularly in the intestinal catarrh of children, on account of atrophic changes affecting chiefly the glandular and muscular layers. Roughening of the inner surface of the bowel, due to projecting glands, is frequent in those forms of chronic intestinal catarrh that are attended with thickening of the coats. Polypoid cysts may develop in long-standing cases.

Etiology.—The **primary** form is produced by (*a*) local irritants, either mechanical or toxemic, that find their way into the intestinal canal. The chief source of these excitants is an unsuitable dietary, and especially is this the case in children. It is readily seen from this fact why the stomach and the intestines are often simultaneously involved in a catarrhal process. (*b*) Over-eating may be productive of the disease, though this often excites diarrhea by merely increasing intestinal peristalsis. (*c*) Idiosyncrasy has a positive influence, the ingestion of certain substances not difficult of digestion being invariably followed by this affection in individuals thus predisposed. (*d*) Toxic substances, whether in the form of tainted food-stuffs (spoiled meats, ice-cream, beer) or inorganic poisons (mineral acids, caustic alkalies, mercury, arsenic) or irritating cathartics, often produce intestinal catarrh. (*e*) Impure water, or water to which individuals are unaccustomed. (*f*) Atmospheric changes, particularly a prolonged high or a sudden fall of temperature, the latter being especially apt to cause it in children. (*g*) An excess or a lack of biliary secretion. Two functions of the bile (its antiseptic properties and its power to stimulate peristalsis) must not be forgotten: the one explains how a paucity of this secretion favors the abnormal processes of fermentation that are capable of exciting catarrh, and the other makes plain the possibility of a bilious diarrhea being due to an excessive hepatic secretion. It is not clear, however, that the latter condition is attended with an actual catarrhal process. The same is true of diarrhea due to fright, excitement, or other nervous influence. (*h*) Bacteria are, doubtless, among the *excitants*—*e. g.* the normal colon-bacillus. under conditions favorable to its growth and development, assumes pathologic virulence exciting irritation.

Secondary or **complicating** forms are caused—(*a*) By direct extension from adjacent organs (ulcers, gastritis, peritonitis, hernia, and invagination); (*b*) By general infectious processes (septicemia, pyemia, typhoid fever, dysentery, cholera, tuberculosis, pneumonia).

The *chronic forms* are met with—(*a*) In certain cachectic states (carcinoma, chronic malaria, chronic Bright's disease, Addison's disease, and profound anemia); (*b*) In connection with disturbances of the circulation, particularly such as produce stasis in the terminal branches of the portal system of vessels: among the chief diseases that tend to prevent the return of venous blood from the intestines are chronic heart-affections, diseases of the liver (especially cirrhosis), and emphysema; (*c*) Severe cases of chronic diarrhea, probably due to the *protozoon balantidium*, have been reported recently.

Among predisposing causes is the *age*, children being particularly liable to the disease. Unfavorable hygienic surroundings, especially when a high temperature prevails, and epidemic and endemic conditions also strongly predispose to the affection.

Clinical History.—From a clinical standpoint we recognize not only acute and chronic forms of enteritis, but also special varieties (*vide infra*).

The **simple acute form** of general catarrh of the intestines (muco-enteritis) has for its two most characteristic symptoms slight *griping* or *colicky pains* in the abdomen (sometimes absent), that are followed soon by *diarrheal stools*. The *discharges* consist, at first, of feculent masses, and later of a watery, highly irritating fluid. Diarrhea is due partly to increased peristalsis and partly to the abnormal irritability of the intestinal mucous membrane. Active peristalsis of the intestines may (*vide ante*) be of purely nervous origin, and produce a diarrhea that is to be distinguished from that due to catarrh. Again steatorrhœa may be present in cases in which the pancreatic secretion is absent. The causes that produce the catarrh also produce the undue peristaltic movements. If it be true, as physiology teaches, that the stools, owing to the absorption of the watery portions of the food, are normally formed in the large intestines, then catarrh of the small intestines alone does not excite diarrhea, though both large and small are involved in the majority of the cases. On the other hand, in *acute colitis diarrhea is conspicuous*, and forms the most important clinical symptom. The vigorous peristalsis also accounts for the gurgling and rumbling sounds (*borborygmi*) that are often felt and heard by the patient himself. These peculiar noises, if pronounced, point to isolated catarrh of the small intestines. The *stools* vary in number from two to ten or more, being increased in frequency after taking food; gases are also formed, causing tympanites. The thin or mushy stools either present a bright-yellow or a yellowish-brown color and emit offensive odors. Occasionally they are greenish in color from the presence of considerable quantities of bile-pigment or from bacterial action. In advanced cases of considerable severity there is painful tenesmus; the stools are often small and contain mucus and blood, becoming dysenteric in character, especially when the colon is chiefly affected.

A *microscopic examination* reveals large masses of epithelium and mucus, as well as countless microörganisms and isolated leukocytes, crystals of calcium phosphate, oxalates, remnants of food (starch-granules, fat, vegetable and muscular fibers). Flakes of yellowish-brown mucus, large pieces of epithelium, and large grayish-white masses of fat may often be seen *macroscopically*. The stools give an alkaline reaction as a rule.

The *physical examination* reveals on *inspection* slight tympanitic distention as a rule. *Palpation* elicits considerable sensitiveness in the majority of cases, though during the colicky pains pressure with the palm of the hand often affords relief. Fluctuation may be detected if the intestines contain much fluid. *Percussion* gives an exaggerated tympanitic resonance, varying, however, with the tension of the bowel. Nausea may be present, and the *appetite* is often greatly impaired. There is marked thirst and the tongue is dry and furred.

The *general symptoms* are often entirely wanting, save for a slight feeling of weakness due to the diarrheal discharges. Severe forms of infectious origin often disturb the general health considerably. The patient is languid, and prostration is prominent; he suffers much from headache, and pyrexia is common, the temperature often reaching 100°–103° F. (37.7°–39.4° C.). The higher temperatures are seen among children. Additional evidences of a systemic affection are sometimes observed, such as painful enlargements of certain joints and severe muscular pains.

Complications.—The symptoms of gastric catarrh (vomiting, nausea, and pain immediately after feeding) are often associated with those of enteric catarrh; the combination is then spoken of as *gastro-enteritis*.

Special Forms.—Though the anatomic limits in the more or less local forms of intestinal catarrh cannot be made out definitely, yet the different clinical pictures observed often ena [illegible] of the disease with considerable accuracy; it is important, moreover, from the standpoint of the treatment, to accomplish this whenever possible. The following may be briefly described:

(*a*) *Duodenal catarrh* (*duodenitis*), in which form constipation, often obstinate, is present in the place of diarrhea, the colon not being affected; merely local pain, tenderness on palpation, and uneasiness are complained of. These symptoms may frequently be overshadowed by those referable to the stomach when gastric catarrh coexists (*gastro-duodenitis*). Without the presence of *jaundice*, due to the occlusion of the common bile-duct in consequence of the swelling of the duodenal mucous membrane, the diagnosis of this affection must remain highly doubtful; but, fortunately, this symptom is frequently observed.

(*b*) Localized *catarrh of the jejunum and ileum* cannot, always be diagnosticated correctly. The condition is often found to be a more or less prominent feature in general enteric catarrh, in which complaint diarrhea is a prominent symptom. The existence of this special variety may be safely inferred when certain enteric symptoms are combined with marked gastric disturbance. Under these circumstances the symptoms indicative of inflammation of the small intestines are rumbling noises (borborygmi), colicky pain, swelling, and slight tenderness over the abdomen in the vicinity of the umbilicus or over other regions occupied by the small intestines. Finally, an *examination of the stools* furnishes valuable points for differential diagnosis. It must be kept in remembrance that in catarrh of the small intestines the stools may be quite solid, despite the increased peristalsis caused by the catarrhal process (*vide ante*). More frequently, when the ileum is the seat of catarrh the colon is also implicated, this combination being attended with diarrhea, even if it be of minor severity. The thin stools "contain food-remnants, that point indubitably to implication of the small intestine." As the result of increased peristalsis of the small intestines their contents are passed into the large bowel with undue rapidity; hence the latter contains undigested food-constituents and other substances that are normally found in the small intestines. These pass from the rectum unchanged. They are mainly starch, fat, and masses of meat-fiber, the latter of which may be of sufficient size to be seen by the naked eye. This would be pathognomonic evidence of the form of catarrh in question if it were not true that increased peristalsis of the small intestines, due to other conditions, as anemia, extreme nervousness, and fever-conditions, that are not seen in ileo-jejunal catarrh, causes the same fecal peculiarities. *Microscopically* the stools show hyaline particles of mucus, giving rise to a speckled appearance.

In health the contents of the small intestines give the characteristic color-reaction for bile-pigment, whilst the contents of the large bowel and the stools do not. There is quite often a large admixture of undecomposed bile-pigment (Strümpell) that responds to Gmelin's test,[1] a fact of considerable value in diagnosis. Nothnagel has called forcible attention to the fact that *bile-stained* stools and small *pigmented* masses of mucus are met with, and are highly characteristic of the diarrhea that marks catarrh of the small intestines.

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[1] This consists in bringing a few drops of nitric acid in contact with the intestinal contents, when the characteristic play of colors appears. (See also Methods of Diagnosis, pp. 792-796.)

(*c*) *Colitis.*—The joint appearance of abdominal pain and diarrhea is almost pathognomonic of this condition. These symptoms, in the absence of the more prominent and above-mentioned clinical features that have special reference to inflammation of the small intestines, point to the fact that the large intestines are the chief seat of the disease.

Physical examination is only partially confirmatory of the rational symptoms. The chief sign is tenderness on palpation over the track of the colon. An *ocular examination of the stools* furnishes important practical results. They may contain blood and mucus, and the latter often in masses large enough to be readily visible to the naked eye; it is not intimately mixed with the feces, as in catarrh of the small intestines, but forms separate masses. The feces are often of the consistence of soup. "If the catarrh affects the lower portion of the large intestine chiefly, it may be that the intestinal contents are already formed" in firm lumps, which may sometimes be wholly or partly enclosed in a layer of mucus (Strümpell).

Such *general symptoms* as loss of flesh, weakness, and sallowness of the skin are often observed. Simple diarrhea, lasting but a few days, as a rule, is to be classed with catarrh of the large intestines, since these affections imply increased peristalsis of the large bowel. It is not always easy, however, to discriminate diarrhea due to purely functional influences or to catarrh of the rest of the intestinal tract.

(*d*) *Proctitis*, or inflammation of the rectum, is characterized by painful tenesmus and by the presence of large quantities of mucus and pus, particularly in the dejections. The disease may be primary, though more often it is secondary to morbid lesions either in organs that are adjacent to or in the rectum itself.

Chronic intestinal catarrh may, comparatively rarely, be a *primary disease*, developing gradually. It may also be *secondary* (*vide* Pathology) at times to one or more attacks of acute intestinal catarrh. Generally there are no other local symptoms to call attention to the condition than *chronic diarrhea.* More rarely there are in addition colicky pain and tenderness over the abdomen. The diarrhea often alternates with constipation, and this is most apt to be the case when the disease is of idiopathic origin and affects only the large intestine (Nothnagel). *Constipation* is constant in those cases in which *atrophic alterations* occur in the glandular and muscular coats, as well as in those in which the lesions are in the small intestines. When constipation is not present the stools are thin, pale, sometimes fermented, emitting offensive odors, and vary greatly in number and quantity. There is commonly present visible mucus. When the small bowels are also implicated, food-remnants are found in the dejections (*lienteric diarrhea*). *Microscopically*, the picture does not differ from that of the acute form. That form of diarrhea occurring in *organic diseases of the heart, liver, and lungs* demands brief special mention. Here the serum of the blood is made to exude into the intestines, owing to mechanical obstruction to the return of the venous blood, and this results in a liquefaction of the feces. The *stools* are apt to be most copious and numerous during the morning hours. Sometimes an irresistible desire to evacuate the bowels seizes the patient as soon as his feet strike the floor on rising in the morning; two or more serous discharges follow each other at short intervals. Subsequently, all discharges cease until the following morning, when the same symptoms are repeated. The *general nutrition* suffers visibly in chronic enteritis, and emaciation eventually becomes pronounced. I have also noticed slight pyrexia, especially in the evening hours.

Differential Diagnosis.—Among the diseases likely to be confounded with acute catarrh of the intestines are *typhoid fever*, *dysentery* (diseases in which diarrhea is a cardinal symptom), *peritonitis*, and *colic*. The chief differential features between simple colic and enteric catarrh may be contrasted thus:

ENTERIC CATARRH.	COLIC.
Diarrhea is generally present.	Constipation is present.
Fever may be slight or marked.	No fever.
Pain is griping, and followed by diarrheal stools.	Pain is colicky, more severe, and is not followed by diarrheal discharges.
Tenderness in the intervals between pains.	No sensitiveness on palpation.

From *peritonitis* we may readily distinguish catarrh of the intestines by the more intense pain and tenderness, by the constipation, the greater tympany, the constitutional disturbance, and more especially by the anxious face, thoracic respiration, and immobility of the patient, all of which characterize the former disease. When the characteristic symptoms of *typhoid fever* (the typical temperature-curve, swelling of the spleen, eruption, Widal-test) and of *dysentery* (scanty, frequent stools, tenesmus) are present, they are easily separable from enteric catarrh. In children, however, the diagnosis between typhoid fever and simple catarrh of the bowels offers considerable difficulty; but the temperature-record, if carefully kept, the enlargement of the spleen, the characteristic eruption, and the Widal-reaction, taken unitedly, will warrant the diagnosis of typhoid fever and exclude acute enteritis.

In diagnosticating chronic intestinal catarrh we may have difficulty in eliminating *lardaceous disease of the bowels* and *ulcerations*. The latter condition will be excluded hereafter. *Amyloid degeneration* is a general disease, affecting primarily other organs than the bowel, and hence lardaceous diarrhea is always preceded by the clinical indications of the disease (enlarged viscera, albuminuria) elsewhere. The condition also gives a definite *etiology* as a rule.

Prognosis.—The prognosis in uncomplicated cases is favorable, though the possibility of a merging into the chronic form must be borne in mind. Occurring in weakly subjects, especially at the extremes of life, and in the course of debilitating affections, *acute catarrh* of the intestines may endanger life. Its duration varies much—from three to ten days or more—according as the type of the individual case is mild or severe.

The prognosis in the *chronic forms* is moderately good as to life, though as to cure it is not so, the disease often enduring for many years together, or as long as the chronic conditions producing it remain unremoved. It sometimes exhausts the system of those suffering from serious causal affections of a chronic nature, and occasionally it ultimately proves fatal. The prognosis will depend largely upon the character of the etiologic affection, but intestinal catarrh invariably renders the prospects of life more gloomy.

Treatment.—Respecting the treatment of this affection the views of the profession have undergone many changes, even within recent years; hence it may be reasonably inferred that our present therapeutic methods are by no means satisfactory.

Hygienic and Dietetic Management.—If the cause be some error of diet, all injurious articles must be rigidly prohibited. In the milder cases due to this cause a mild purgative, followed by proper *dietetic treatment*, is all that is required. Albuminous food in liquid form, such as skimmed milk, weak broths, and even semi-animal articles of diet, as eggs, oysters, sweet milk with seltzer, are usually well borne. In the severe forms predigested liquid foods only should be allowed. When the chief seat of the disease is in the large intestine, we may allow easily digested starches and certain green vegetables (arrow-root, sago, lettuce, water-cress); the coarser vegetables, all fats, and most fruits should be withdrawn absolutely. *Rest* in bed is especially beneficial in that it serves to keep the abdomen warm and mitigates the pain and diarrhea, and, in short, cures the disease. Sinapisms should be applied at the outset until the skin is reddened, succeeded by light linseed poultices until the local sensitiveness has, in a great measure, subsided; after this a flannel band may be applied. The local abstraction of blood by a few leeches, applied to the abdomen or anus, is beneficial in the early stages in severe types of enteric catarrh, provided the patient's strength is good.

Medicinal Treatment.—It is sound practice to prescribe a mild cathartic (castor oil, calomel, or rhubarb, followed by a saline) with a view to getting rid of decomposable intestinal contents. Combined gastric lavage and high intestinal irrigation has recently yielded excellent results in my hands; it is an appropriate method of overcoming the fermentative processes that tend to excite and maintain the condition.

If the chief tenderness be localized in the right iliac fossa, corresponding to the course of the colon, a simple enema, slowly given, will stimulate the bowel sufficiently and cleanse it more effectually than a cathartic. Subsequently, chief reliance is to be placed on intestinal antiseptics and astringents, though it must be recollected that the selection of internal remedies must, in part, be influenced by the etiologic indications. For instance, if the cause has been exposure to cold or wet, besides the efforts directed at the local condition diaphoretics and febrifuge mixtures are serviceable. I have found the following combination to be of benefit in controlling the inflammatory action:

℞. Salol,	ʒss (2.0);
Creasoti,	♏x (0.666);
Bismuthi salicylat.,	ʒj (4.0).

M. et ft. capsulæ No. xx.
Sig. One every three hours.

If pain be troublesome, opium or phenacetin may be combined with the above formula.

In many instances the secretions of the intestinal tube are decreased for a considerable period after the most active symptoms have been subdued. Here we must supplement the natural juices of the bowel, as follows:

℞. Pancreatin,	ʒj (4.0);
Sodii bicarb.,	ʒij (8.0).

M. et ft. chart. No. xij.
Sig. One an hour after meals.

In cases in which the large intestine is chiefly affected, and when the condition does not yield to internal medicines, treatment per rectum should be employed. If colicky pain be severe, morphin (gr. $\frac{1}{8}$—0.008) should be given hypodermically in addition to the measures before suggested. If the diarrhea shows no tendency to abate after forty-eight hours of the general treatment above outlined, large doses of bismuth (gr. xxx to lx—2.0 to 4.0) every three or four hours should be tried. In my own hands lead acetate (gr. ij—0.129), with the extract of opium (gr. $\frac{1}{8}$—0.008) in pill-form, has proved a most efficient combination. When there is reason to suspect that the main lesion is in the large bowel, small enemas of starch-water (℥ij—64.0), with laudanum (♏ xx–xxx—1.33–2.0), every four to six hours, are efficacious. The thirst is best relieved by chipped ice in small quantities or by carbonic acid and Apollinaris waters. For distressing flatulence we may prescribe the alkaline carbonates, or spirits of ammonia, and some carminative. The oil of cajeput is a most valuable drug in the treatment of excessive fermentation (Murrell).

In *chronic catarrh* of the intestines the local treatment is of paramount importance. Daily irrigation of the bowel with a weak solution of some antiseptic agent, as salicylic acid (gr. v–℥j—0.324–32.0), boracic acid (gr. x–℥j—0.648–32.0), creolin (♏ v–℥j—0.324–32.0), or with some such astringent as tannin (gr. v–℥j—0.324—32.0), or finally with an alterative, such as silver nitrate (gr. $\frac{1}{4}$–℥j—0.016–32.0), will be found to be beneficial. The latter solution is a most excellent remedy, but sometimes excites pain if used in excessive doses. To obviate this, I have often used a mild antiseptic or astringent with the foregoing, giving them on alternate days, and thus obtained most happy results. The only appliance needful is a fountain syringe with a soft-rubber end-piece, which should be gently introduced for a considerable distance into the bowel. The fluid used should be warmed to 90° F. (32.2°C.), and the quantity administered at each sitting should be not less than 2 to 3 pints (1–1.5 liters); this should be allowed to flow in slowly. The patient should, as a rule, assume the dorsal decubitus, though if the fluid is to be carried as high up as possible, the knee-elbow position may be assumed or the patient may be placed on the left side with the hips elevated.

The same careful attention must be paid to ***hygienic details***, and especially to the diet, as is directed in the acute form. In addition, flannel should be worn next the skin both in winter and summer. If the strength will admit of it, cold baths are useful.

A stay at a suitable spa (Saratoga, Bedford, Virginia Springs, Carlsbad, Kissingen) often produces most satisfactory results.

Among internal agents, zinc oxid (gr. v to x—0.324–0.648—t. i. d.), silver nitrate, lead acetate, and alum, given with tonics, such as strychnia, arsenic, and iron, are especially to be recommended.

The management of this troublesome malady depends upon the indications furnished by the causative affections. No method of treatment can succeed, however, that is not carried out patiently, systematically, and over long periods of time.

DIARRHEAS OF CHILDREN.

ACUTE GASTRO-INTESTINAL CATARRH.

(*Acute Gastro-enteric Infection; Summer Diarrhea; Gastro-enteritis; Cholera Infantum; Mycotic Diarrhea.*)

Definition.—This is the usual intestinal trouble that prevails during the warm summer months. It usually takes the form of an epidemic, and its course is manifested by a sudden onset, high fever, irritability of the stomach, frequent watery evacuations, symptoms of nerve-involvement. This form of diarrhea usually follows an attack of acute indigestion, in which it very frequently has its origin (*acute dyspeptic diarrhea*). Acute gastro-intestinal catarrh (*cholera infantum*) stands midway between acute indigestion and ileo-colitis.

Etiology.—Two important conditions seem to be necessary to influence the disease—*temperature* and *diet*. A general and well-recognized belief associates special danger with the second summer of children. Out of nearly 2000 fatal cases collected by Holt, only 3 per cent. were exclusively breast-fed. Generally speaking, the disease has its origin in some irregularities in artificial feeding. Heat is an important element in the continuation of the disorder when once commenced.

Season exerts a most potent predisposing effect. It is peculiarly a disease of summer (though instances are not uncommon in May and September), the greatest prevalence occurring in July. The pauper element of large cities furnishes most instances, although it may be met among the children of the most provident.

Baginsky inclines to the belief that the decomposition-products formed by the different varieties of microörganisms found in the intestines are the toxic substances that give rise to the disease.

The *proteus* class of bacteria are most frequent, and are most likely to possess pathogenic properties, according to Booker, who has made a complete study of the subject. With him are in accord the opinions of Jeffris and Baginsky. Escherich has shown that in milk-fed children the normal feces constantly contain two organisms—the bacterium coli commune and the bacterium lactis aërogenes. Later investigations enabled him to distinguish between these normal bacteria and those present in pathologic conditions. Escherich maintains the specificity of the streptococcus (violet cocci grouped in pairs or small chains), because it displaces the normal bacteria of the intestines during the disease and disappears as the disease subsides. Moreover, it is found in the mucous and serous elements of the discharges; also during life in the blood and urine, and finally, after death, in all the organs.

Pathology.—A catarrhal swelling of the mucosa of the large and small bowel is present; the mucosa itself is pink in color from capillary congestion. Peyer's patches are enlarged. The whole intestinal tube shows an early stage of inflammation (ileo-colitis). In addition there is most likely some involvement of the sympathetic nerves, leading to dilatation of the capillaries and transudation of serum into the intestine, and to alterations of the pulse, temperature, and respiration. Its nature is paralytic, and closely resembles in its results experimental sections of the sympathetic nerves. The changes in the other organs are slight. Broncho-pneumonia frequently occurs. The spleen is often swollen, the brain is anemic, and the kidneys are congested.

Symptoms.—Clinically, we recognize three forms of acute enteric infection: (1) acute dyspeptic diarrhea; (2) cholera infantum; and (3) ileo-colitis.

(1) *Acute Dyspeptic Diarrhea.*—There may be merely an increase in the number of stools, with or without fever; restlessness is usual at night. This condition may continue for two or three days, when the stools become more frequent and offensive, containing undigested food and curds. The *odor* by this time is very pronounced. Frequently the disease has a sudden onset, with vomiting, griping pains, and fever, which may quickly rise to 104°, 105°, or 106° F. (40°–41° C.). *Convulsions* may be the commencement of the attack. The abdomen is sensitive and swollen, and the child lies with its legs flexed on the stomach. The *stools* consist of grayish or greenish-yellow feces (mixed with gas, curds, portions of undigested food) and some fluid. In children two years of age and older the stools may contain unripe fruit or large curds from excessive drinking of milk. Relapses are frequent, and during hot weather the frequency of the attacks may lead to the commencement of a severe entero-colitis.

In delicate children a severe attack, especially if it is accompanied by convulsions, may prove fatal. From the fact that the general symptoms may be few, the case is often allowed to go on for several days, under the impression that the child is "only teething."

(2) *Cholera Infantum.*—The *initial* symptoms are sudden. The child voids immense stools, at first fecal, if no preceding diarrhea have been present. Soon they become watery, light yellow or greenish in color; frequently they are so thin and colorless as to pass through the napkin without leaving a stain. At times they contain a few yellow or greenish flocculi or a mass of mucus, and in all cases they are odorless. Very often the stools are brown and liquid, with a small quantity of fecal matter, having a peculiar musty odor that clings to the napkin and child for days. The number of stools per diem may vary from six to thirty, and a most remarkable feature is the fact that they are evacuated with considerable force.

The *stomach* becomes irritable, refusing everything; even ice is rejected as soon as swallowed. The *appetite* is, of course, entirely lost; intense thirst prevails, the little patient drinking at every chance and following the receding glass with eager eyes. The *tongue*, moist at first, soon becomes dry and pasty; the abdomen is collapsed. The *temperature* is always high—105° or even 108° F. (40.5°–42.2° C.); and the *pulse* small and very frequent—130 to 180 beats per minute. The *breathing* is shallow and irregular, and the *eyes* anxious and staring, but soon becoming dull. The urine diminishes in quantity daily

With this array of symptoms there is a striking and appalling change in the child's general appearance. Within a few hours smiling, perhaps plump and rosy, it can now scarcely be recognized; the face has become pale and pinched, the eyes and cheeks sunken, the eyelids and lips wide apart from loss of muscular control, the muscles flabby, the bones prominent, and the skin greenish or cadaverous, hanging in loose folds from the wasted frame, all the fat having melted from the body.

Collapse comes on soon: the hands, feet, nose, and breath become cool, the respirations more unequal, and there are drowsiness and utter apathy. When life is near its close, vomiting stops, the whole surface becoming cool and clammy as the patient sinks into a state of coma, with injected eyes and contracted pupils. At last the end is reached quickly, preceded perhaps by a slight convulsion. The *duration* of the disease is short; it may prove fatal in from one to four days.

(3) *Ileo-colitis.*—This may follow acute dyspeptic diarrhea, or complicate the acute infections of childhood. The symptoms develop acutely. At the outset there may be vomiting, but it is not persistent, and the stools are greenish, feculent, often showing masses of casein. Later the discharges are increased in frequency, are small, and contain also blood and mucus. In severe cases pain and straining are distressing features. The abdomen is prominent and there is tenderness along the course of the colon.

The *course* is variable. It may be acute, terminating either in convalescence or death due to exhaustion. In other instances the acute symptoms subside, particularly the fever, while moderate diarrhea continues and is attended with marked wasting, debility, and sometimes hydrocephalus. Gradual recovery may ensue, though more commonly relapses occur and death follows from broncho-pneumonia or an intercurrent acute attack.

Treatment.—The treatment of *acute gastro-intestinal catarrh* divides itself into hygienic, dietetic, and medicinal measures. If a child is attacked in the city during the summer and does not yield to treatment in two or three days, it should be sent to the country or seashore. In the case of a child under two years this is absolutely imperative. Fresh air is important in all diarrheal disorders in summer both in country and city, and all cases should be kept out of doors as much of the time as possible. Children should be kept quiet—not permitted to walk, even if able. Bathing is soothing and beneficial in that it ensures cleanliness and, what is very important, reduces the temperature.

Dietetic treatment is of great importance. It should be remembered that digestion is arrested in the early stage, hence to give food at this stage is to do harm. Thirst may be controlled by ice- or albumin-water, toast-water, or gum-water, with a little brandy.

Medicinal Treatment.—The first step is directed against the acute indigestion and the active putrefaction going on in the intestinal tube. The indication, therefore, is to empty thoroughly the whole alimentary tract as soon as possible, and no other treatment must be thought of until this end has been accomplished. Whenever vomiting persists the stomach should be washed out; usually one washing is sufficient. In older children emetics will favor complete emptying of the stomach, but are never to be given to infants under two years. For the intestine calomel and soda may be used; for the colon we may use, in addition, irrigation: this is advisable in all cases, as it hastens the effect of the cathartic and removes at once much irritating and offensive material. Opium should not be used until the whole intestinal tube is clean, and then cautiously. Spirits of chloroform, or camphor, is a better remedy for the pain than opium in any form. In older children the hypodermic injection of morphin and atropin in appropriate doses most frequently controls the whole train of symptoms. Bowles has used lactic acid in the maximum dose of $1\frac{1}{4}$ grains every hour in 60 cases of summer diarrhea, and found it to control the symptoms in every case in from 24 to 48 hours.

Treatment of Cholera Infantum.—In this form of infection of the intestinal tract we are likely to forget that we are called upon to treat a case of acute poisoning. The toxic material acts both powerfully and quickly as a cardiac and systemic depressant. It also acts toxically upon the nerve-centers, and paralyzes the vaso-motor nerves. According to Holt, the leading indications are—(*a*) to empty the stomach and intestines; (*b*) to supply the body with fluid to offset the great loss by vomiting and purging; (*c*) to counteract the effect of the poison on the heart and the nervous system; (*d*) to reduce temperature; and (*e*) to treat the symptoms as they arise. In the first condition thorough stomach and intestinal cleansing is absolutely necessary. Moreover, we cannot depend on emetics or purgatives to arrest pain and to limit the effect of the poison on the nervous system; a hypodermic injection of atropin and morphin is essential. Morphin must be given with discrimination to young children, especially when the vomiting and purging are slight; it is especially contraindicated when stupor or collapse seems near. Small doses repeated are better than larger single doses. Holt gives gr. $\frac{1}{100}$ (0.0006) of morphin, with gr. $\frac{1}{800}$ (0.00008) of atropin, as the first dose in a child one year old. In supplying fluid to the exhausted tissues it is useless to attempt to give them by the mouth, or even by the rectum, as by both avenues it would be rejected. An injection into the cellular tissues of the buttocks, back, or thighs of a saline solution (40 grains—2.59—of common salt to a pint of sterilized water) is the best way to meet the drain. One pint (half liter) may be used every twenty-four hours, and larger quantities may often be used with advantage. Baths must be given to control temperature, and ice-bags should be placed to the head. Ice-water injections will aid in the control of temperature, and ice-suppositories act efficiently when the water is not retained. Stimulants may be given hypodermically. During the active stage nothing should be allowed by the mouth except iced brandy or champagne.

The dietetic management and internal treatment of ***ileo-colitis*** are similar to that of the preceding variety. A dose of castor oil or of calomel is to be promptly administered and followed in a few hours by copious irrigations of the colon, preferably with tepid saline solution (strength 7 : 1000). Later a small quantity of a thin starch solution, to which ♏j to iij of laudanum has been added, may be gently thrown into the rectum, to be repeated once or twice daily. After the acute stage is over a weak silver nitrate solution may be employed.

CELIAC DISEASE.

(*Diarrhœa Alba; Diarrhœa Chylosa.*)

Definition.—A form of intestinal catarrh marked by copious fetid and frothy discharges resembling gruel.

Pathology.—Although ulcers have been noted in the intestine, the pathology of the disease is not known. Says Osler: This affection resembles somewhat the disease in adults known as "hill diarrhea" or the "white flux" of India.

Etiology.—The disease is limited chiefly to children from one to five years old. The filaria sanguinis hominis has been found in the feces in cases of diarrhœa chylosa.

Symptoms.—The disease is of slow development, and the characteristic feature consists of copious diarrheal (though not watery) stools, resembling gruel or oatmeal-porridge. These are also frothy (*frog-spawn*) and horribly fetid. The physical signs consist of a moderate distention of the abdomen and a boggy sensation that is imparted to the palpating finger. The general features may be summated in gradually increasing emaciation, debility, and pallor. The disease terminates fatally as a rule.

The **treatment** is purely symptomatic, unless the presence of parasites be suspected, when large antiseptic enemata should be given.

PHLEGMONOUS ENTERITIS.

THIS is a suppurative inflammation of the submucous layer of the intestines. It is among the rarest of grave maladies, especially as an irrelative disease. It may be diffuse or take the form of a circumscribed abscess. Rarely it occurs as a complicating condition in septico-pyemia and in malignant types of the exanthemata, resulting in the formation of abscesses that have their seat usually in the duodenum. Phlegmonous enteritis may be secondary to strangulated hernia or intussusception.

Symptoms.—The *local signs* simulate closely those of peritonitis. Among the symptoms *vomiting* is prominent, though not diagnostic; it is always severe, and may become stercoraceous. *Pain* and *tenesmus*, when due to obstruction, are intense. Rigors more or less severe have been observed. The *temperature* is high, and its curve is somewhat typical of the fever of suppuration. The disease is very fatal, the patient passing from a condition of extreme prostration to one of utter collapse.

Treatment.—The physician's task is confined to an attempt to support the powers of the patient and to relieve his inordinate suffering. The surgeon's aid should be invoked early in cases of obstruction.

CROUPOUS OR DIPHTHERITIC ENTERITIS.

Definition.—An intense inflammation of the intestinal mucosa, accompanied by a croupous exudate; it occurs in connection with widely various conditions. If from any cause the epithelial covering is destroyed, agents that set up local inflammation may be productive of croup.

Pathology.—There are two sets of morbid lesions to be distinguished: (1) The first and most important class exhibits a croupous deposit varying greatly in thickness and in superficial area. Its color is variable, being sometimes of a *grayish* or *grayish-white hue*, though more frequently, perhaps, *grayish-yellow*. I have almost invariably seen these lesions in the colon. (2) In the second group the solitary follicles alone are inflamed, and the diphtheritic deposit is merely coextensive with their mouths.

The *etiologic* factors may be (*a*) mechanical irritants (impacted feces, enteroliths, gall-stones); (*b*) chemical irritants (ammonia, acids, mercury, arsenic); (*c*) the condition may be secondary to acute infectious diseases and certain chronic complaints (Bright's disease, pyemia, carcinoma).

Symptoms.—When mechanical irritants give rise to symptoms, they do not differ from those due to stercoral ulcers, and there is no way of recognizing the croupous deposits unless they be discharged *per rectum* and are detected in the stools. In cases that arise from the action of irritant poisons vomiting and purging are well marked and the dejections contain blood-stained mucus. We cannot be certain about the presence of croupous deposits in toxic cases unless they be found in the discharges. When phlegmonous enteritis occurs as a complicating condition in infectious diseases, the symptoms are almost completely veiled. The symptomatology of the follicular variety cannot be separated clinically from that of follicular ulceration.

The **treatment** is that of the causal conditions or affections.

SPRUE.

(*Psilosis.*)

THIS has been defined as "an insidious, chronic, remitting inflammation of the whole or part of the mucous membrane of the alimentary canal, occurring principally in Europeans who are residing or have resided in tropical or subtropical climates" (Manson).

The principal **morbid changes** consist in patchy or general destruction of "the surface of the mucosa in all degrees, from slight erosions to complete disintegration of the villi, glands, and follicles." Congestive, catarrhal, ulcerative, and cirrhotic changes may be all combined in one and the same case.

The **etiology** is unknown, although the disease is probably of microorganismal nature. Residence in hot climates and previous affections of the alimentary tract are the main predisposing causes.

The leading **symptoms** are, according to Manson, irregular action of the bowels, and the passage of copious, pale, drab-colored, yeasty-looking, sickly-smelling stools. The oro-cavity is inflamed and the seat of erosions, cracks, and superficial ulcerations. Brunton has pointed out that Indian Hill diarrhea differs from sprue in that soreness of the mouth and anus is absent in the former.

Early appropriate **treatment,** which is principally dietetic (milk-diet) and hygienic, checks the progress of the disease.

CHOLERA MORBUS.

(*Cholera Nostras; Sporadic Cholera.*)

Definition.—A self-limiting disease, characterized by serous vomiting and purging, colicky pains, and often muscular cramps.

Pathology.—No constant anatomic changes have been noted. They are analogous to those seen in acute gastro-enteritis, though cases have terminated fatally in which no morbid lesions were found *postmortem.*

Etiology.—Among predisposing causes, the *age* and the *season* exert the most prominent influence. The condition may appear in subjects under two years, when the term "cholera infantum" is employed, though it is more often met with in older children and adults. It is almost invariably seen during the heated term in temperate zones, from the latter part of June to September, and it is especially prevalent during the months of July and August. Bad hygienic environment, foul air in particular, has a noticeable effect, and, though not as yet absolutely proved, it may be safely inferred from the clinical history and the usual course of the affection that it is of microbic origin. Among other factors are improper food, particularly unripe fruit, cucumbers, egg-plant, and exposure to cold and wet. Various *organisms* (especially the Finkler and Prior spirillum) have been found present. No one variety, however, has been definitely found to be the cause of the condition. Virulent specimens of the bacillus coli commune, and even of the streptococcus, have been noted.

Clinical History.—The *onset* is often sudden, and is marked by abdominal pain, vomiting, and diarrhea. At first the *vomitus* consists of food, and later of bile and mucus. The *dejections* are fecal in character at the onset; though they soon become watery, and may resemble the rice-water stools of Asiatic cholera.

Physical examination reveals only tenderness on pressure over the abdomen, particularly the epigastric region.

General symptoms are not wanting. Cramps in the calves are common. The thermometer may register a high temperature, though it varies greatly, ranging from 100° to 106° F. (37.7° to 41.1° C.). The skin-surface, however, and more particularly that of the extremities, feels cool, and owing to this fact the rectal temperature should be recorded. The pulse, as the case progresses, becomes rapid and feeble. The face is pale or even cyanotic, the features looking pinched. The extremities lose their plumpness, and the patient usually appears prostrated and mentally dull. The urine is apt to be scant, high-colored, and sometimes albuminous, and thirst is extreme. There is a group of cases that develop subacutely and the symptoms tend to persist.

Differential Diagnosis.—The symptoms of cholera morbus resemble so closely those of *Asiatic cholera* as to preclude the possibility of a differential diagnosis from the symptoms. A bacteriologic examination of the stools alone, however, permits a certain discrimination. During a cholera epidemic the distinction between them is made without difficulty. The effects of certain direct irritants, as in poisoning by ptomaïns and toxic doses of arsenic, must be excluded by the history.

Prognosis and Duration.—The duration of the disease varies from three to four hours to two days. It is rarely fatal, though in persons suffering from such chronic affections as Bright's or cardiac disease, and also in the aged, the prognosis is only guardedly favorable. A pronounced algid state should not be looked upon as free from danger. An element of danger is profound collapse. In two of my cases a condition of marked neurasthenia, indigestion, and functional heart-disturbance formed a series of sequelæ that lasted for several months. Nearly all cases, however, recover without sequelæ.

Treatment.—The *diet* must be rigorously restricted, and predigested milk and animal broths are to be prepared as lightly as possible until convalescence has been fairly established. The comfort of the patient, as well as the cure of the disease, is much enhanced by keeping the patient at absolute rest. *Local measures* are useful in combating pain and vomiting. A large mustard-paste applied to the stomach and abdomen, followed by linseed-poultices that are to be worn constantly, has a strong influence in accomplishing the relief of the symptoms before mentioned. If indigestible substances have been taken prior to the attack, prompt though mild laxatives are to be given at the beginning of the treatment. For the excessive thirst chipped ice, over which a little brandy has been sprinkled, is effective. For controlling the morbid sensitiveness, on which the pain, nausea, and the diarrhea depend, we have a remedy *par excellence* in the hypodermic administration of morphin. The dose should vary (gr. ¼ to ½—0.016 to 0.032) according to the severity of the symptoms, and I have rarely found it necessary to give a second dose. Not only are the pain and diarrhea subdued, but the peripheral circulation is also re-established. It has also been recommended to administer opium by the mouth for these symptoms in the form of the solid extract or laudanum, but the results are infinitely more brilliant when the drug is employed subcutaneously. The other points in the treatment of this affection are identical with those discussed under the treatment of Gastric and Enteric Catarrh.

INTESTINAL INFARCTION.

A FEW instances of occlusion of the superior mesenteric artery by an embolus have been recorded recently. The condition produces hemorrhagic infarction of the small intestines, and is marked by grave and usually fatal symptoms. Its *causes* are sometimes obscure. The cases that have come to autopsy have shown intense congestion, with a swollen, blood-infiltrated state of the jejunum and ileum. Osler has seen three instances: in one there were numerous vegetations on the mitral valves from which the embolus was probably derived; in another the superior mesentery was plugged at its orifice; and in the third the artery was blocked by a portion of the fibrous clot of an aneurysm of the aorta near the diaphragm. The *symptoms* are urgent. Quite often diarrhea is present from the first, the dejections sometimes becoming blood-tinged. Soon, however the characteristically grave symptoms of intestinal obstruction supervene—viz. *great pain*, *vomiting*, and *constipation* (less commonly diarrhea), with *tympanitic distention* of the abdomen (generally). The condition cannot be recognized from the symptoms and physical signs on account of their close resemblance to the various forms of obstruction, yet its probable existence may be inferred from the presence of the known causes.

INTESTINAL ULCERS.

DUODENAL ULCER.

Definition.—A small, round perforating ulcer of the duodenum (*vide* p. 767). It may be primary or secondary.

Pathology.—The morbid characteristics are so nearly identical in appearance and nature with those of peptic ulcer of the stomach that they scarcely demand a separate presentation. The *seat* of the ulcer is with few exceptions above the orifice of the common bile-duct. When these ulcers heal the resulting cicatrix produces stenosis, which in turn leads to dilatation of that portion of the duodenum back of it, and finally of the stomach also. Progressive cicatricial contraction may completely close the *ductus communis*, and in like manner the pancreatic duct or the portal vein may be occluded. Protective adhesive inflammation between the duodenum and the adjacent parts (pancreas, gall-bladder, liver) often prevents complete perforation of the duodenal wall: when perforation does occur, however, the peritoneal cavity may be opened, causing peritonitis, or a fistulous communication may be established with the gall-bladder, liver, or pancreas.

Etiology.—Though the duodenal ulcer has, as a rule, the same mode of origin as the gastric ulcer, the fact should be prominently mentioned here that extensive burns of the skin-surface of the body are quite prone to be followed by a perforating ulcer of the duodenum, while gastric ulcers are seldom caused in this manner. To explain this form of ulceration of the duodenum is difficult. It is quite probable, however, as in other forms of duodenal and gastric ulcers, that the circulation is arrested by an embolus (from decomposing masses of blood) at some point in the mucous membrane, the acid gastric juices subsequently digesting the part that is thus deprived of its blood-supply. Ulcerations of the duodenum have recently been met with in cases of chronic Bright's disease, and also found associated with gall-stones.

The influence of *sex* and *age* as causal factors is notable and in striking contrast with their import in gastric ulcer. In the latter disease most instances occur among young females, while in duodenal ulceration they occur, as a rule, in males between the twentieth and fortieth years. Of 64 cases collected by Kraus, only 6 sufferers were females. In view of the fact that the pathology of gastric and duodenal ulcers is the same, these differences respecting their etiology are inexplicable. The ratio of cases of gastric and duodenal ulcers, however, is about as 30 to 1 in favor of the former.

Clinical History.—Perhaps no real distinction between the symptoms of gastric ulcer and those of its homologue affecting the duodenum can be said to exist in most instances. A probable diagnosis of ulceration of the duodenum has, however, been repeatedly made, and sometimes verified by the subsequent autopsy. If duodenal ulcer be classed with gastric ulcer, there is great danger that the true nature of many cases will be overlooked. The difference in the symptomatology in the two forms of ulceration is owing solely to the difference in locality, implying a difference in nervous and blood supply.

The distinctive features of this disease may be shown by presenting its leading symptoms by the side of those characteristic of gastric ulcer:

Duodenal Ulcer.	Gastric Ulcer.
Usually occurs between 20 and 40 years, except when due to external burns.	May occur at any age after childhood.
Males are more frequent sufferers than females, in the proportion of 10 to 1.	Females are the chief sufferers.
Onset marked by intestinal hemorrhage, which may recur at intervals of varying duration.	Gastric hemorrhage often occurs, preceded by other gastric symptoms, as a rule.
The melena may be preceded by or accompanied by hematemesis, though not generally.	Blood may appear in the stools, usually after hematemesis.
Blood in the discharges often is bright red, profuse, sometimes dark, and tarry from the action of acid chyme when slight, though less marked than when from the stomach.	The blood in the dejections is dark and tarry from the action of the gastric juices.
Pain may come on late, two to four hours after meals; more often it is absent. It is localized in the right hypochondriac region.	Pain paroxysmal, greatly influenced by taking food. Pain sharply localized in the epigastric region, about two inches below the ensiform cartilage.
Gastric crises of great violence occur without reference to time of taking food.	Gastric crises come on soon after taking food.
Vomiting inconstant, without relation to ingestion of food, and affords no relief.	Vomiting more common (during painful crisis) and affords relief.
Jaundice occasionally present from occlusion of bile-duct.	Jaundice absent.
Less marked improvement after diet has been regulated.	Usually a marked improvement follows regulation of diet.
Dorsal pain-point absent.	Painful point (between the tenth and twelfth dorsal vertebræ on left side) usually present.

Finally, "whenever a young man in apparently good health is attacked by melena, with or without hematemesis, it is probable that the ulcer is located in the first part of the duodenum rather than in the stomach, the converse being the case in young women" (Fenwick).

Of the symptoms mentioned under Duodenal Ulcer, the *intestinal bleedings* and *violent crises* (in which the pain is referred to the right hypochondrium, and comes on from two to four hours after meals) are the most diagnostic. While hemorrhage is the leading single symptom in this complaint, we must not, in attempting to estimate its significance in any case, neglect to eliminate *hemorrhoids*, *carcinoma*, *tuberculosis*, *dysentery*, and finally the *hemorrhagic diathesis*—all conditions in which melena occurs as a cardinal symptom. Recently many cases have been reported in which there was an entire absence of symptoms until perforation occurred, followed by rapidly fatal suppurative peritonitis (*latent duodenal ulcer*). In regard to these accidents we may refer to what is said in the description of the latter disease (*infra*).

The signs of dilatation of the stomach, for reasons before stated, sometimes follow the healing of these ulcers, associated usually with chronic gastro-duodenal catarrh, the latter being due to mechanical causes. Rarely, stenosis of the *ductus communis* takes place as the result of duodenal ulcer; more frequently tumors either compress or occlude the lumen of the bowel below the mouth of the duct. The symptoms presented differ widely from those due to stenosis above the duct, the most characteristic being the continual backward flow of bile into the stomach, sometimes attended by constant vomiting of biliary secretions. As in the case of gastric ulcer, in the duodenal form there is at times so much thickening about the base of the ulcer as to give rise to the signs of tumor. This is especially true of those instances in which the base of the ulcer becomes attached to adjacent organs; in such cases the resemblance to malignant disease may be striking.

Prognosis.—The risk to life is greater than in gastric ulcer, since there is less tendency to cicatrization.

Treatment.—The suggestions made in the treatment of gastric ulcer are entirely applicable to the duodenal form also.

Follicular ulcers have already been described under Catarrhal Enteritis (*vide* p. 797), and they have a similar pathology and etiology. When present in goodly numbers they give rise to a symptom peculiarly their own, and hence may be dignified by a separate though brief mention. The symptoms of the condition arising in the course of chronic enteritis often escape observation for a long time. The most characteristic manifestation is the appearance in the stools of conical-shaped masses of mucus resembling "boiled sago." Marked weakness and emaciation rapidly ensue. Among children the disease is common and assumes an aggravated form, the little sufferers quite frequently reaching their end as the result of inanition. An unfavorable termination may be due to perforation followed by suppurative peritonitis. The *treatment* coincides with that of chronic enteritis.

Stercoral ulcers are the result of the mechanical effect of hard fecal scybala (often enteroliths, due to a deposit of lime-salts) upon the intestinal mucous membrane. They occupy the sides or tops of the normal folds in the colon.

Symptoms.—There is, as a rule, a clear history of *chronic constipation*, though the physician may, notwithstanding, be called on account of the presence of *diarrhea;* this is caused by the retained hardened feces working their way into the rectum. A *digital exploration* will now clear up the diagnosis. There are *tenesmus* and *colicky pain* in the abdomen, the latter symptom being also complained of when no diarrhea is present. The pain often occurs in severe paroxysms that may be attended with the discharge of thready or flaky mucus, pus, and sometimes blood.

Physical Examination.—Palpation may in rare instances reveal the presence of a sausage-shaped tumor and sharply localized tenderness over the seats of ulcers.

Enteroliths may lie in the intestines for years together, or they may finally be discharged with the stools. The ulceration that is thus caused often passes unrecognized.

The **prognosis** is good if the condition be not overlooked.

The **treatment** consists in thoroughly evacuating the bowels by salines and simple enemata, persistently used. Subsequently these cases are to be managed in the same manner as other non-specific ulcers of the bowels.

Simple ulcerative colitis is a not uncommon complaint, and one that is frequently associated with chronic intestinal catarrh. The ulcers may be quite extensive, removing the greater portion of the mucous membrane, though in several instances I have observed cases at the Episcopal Hospital that were superficial; these were confined almost solely to the mucosa. The muscular layer of the gut was greatly hypertrophied and its lumen increased in every instance. The non-ulcerated portions of the mucosa looked, in part, quite pale, and in part quite dark. Polypoid growths have been observed situated between the ulcers.

The **etiology** is obscure. The disease is met with most frequently in persons past middle life, and it is quite probable that chronic enteritis sustains a causal relation. Those whose constitutions have been enfeebled by previous disease or an improvident hygienic environment are the chief sufferers.

Symptoms.—The clinical features are ill defined at the onset, and are often erroneously ascribed to indigestion. *Diarrhea* (lienteric in character) is its most prominent symptom, and with it constipation may alternate. Pus and blood are absent with the rarest exceptions. The general health soon suffers greatly, the patient becoming weak and emaciated.

The **course** of the disease is subacute, tending to become chronic.

The **diagnosis**, apart from a consideration of the symptoms above mentioned, requires the elimination of *dysentery*—an easy task as a rule. The disease resembles most closely the amebic form of dysentery, hence in dubious cases a microscopic examination of the feces should not be neglected.

Prognosis.—This is unfavorable during the earlier stages in the aged. The tendency to chronicity of the disease must be considered.

The **treatment** embraces (*a*) a careful regulation of the diet, consisting in a restriction of the patient to liquids and semi-solids during the acute stage; (*b*) the administration of a gentle laxative, followed by antiseptics and astringents (bismuth gr. xxx—2.0—combined with salol gr. v—0.324—every four hours); (*c*) the more serviceable local measures in the form of enemata, among the best being silver nitrate (gr. ¼ ad ℥j —0.016 to 32.0) or creolin (2 per cent.).

Solitary Ulcers.—"Two instances of ulcer of the cecum, both with perforation, have come under my observation, and in one instance a simple ulcer of the colon perforated and led to fatal peritonitis" (Osler).

The **diffuse catarrhal ulcer** is inseparable from acute enteritis; the **cancerous ulcer** is alluded to under the latter head.

APPENDICITIS.

Definition.—A catarrhal, ulcerative, or interstitial inflammation of the appendix vermiformis. It must be confessed that, according to our present views, appendicitis is a surgical rather than a medical affection, particularly from the standpoint of treatment. Knowing from personal experience and observation, however, that general practitioners are constantly meeting with cases of appendicitis, its prompt clinical recognition by the latter is not only a matter of interest, but also of great practical importance for two reasons: First, in order that surgical intervention can be instituted at the proper moment; and secondly, because appendicitis is the leading serious disease of the intestinal tract.

The term "appendicitis" includes the affections *typhlitis* (inflammation of the cecum) and *perityphlitis* (a similar involvment of the connective tissue behind the cecum) by reason of the fact that with few exceptions when the symptoms of the latter affection are presented the appendix vermiformis is the part primarily affected. To the physicians and surgeons of America belongs the credit of having first established the truly important rank of appendicitis.[1]

Anatomic.—Without any known function the human appendix vermiformis represents the remains of the enormous cecum of inferior animals, especially rodents and herbivora. Clado asserts that the vermiform appendix is kept in position by two folds of peritoneum, a meso-appendix, which is attached to the iliac fossa, and a second fold, perpendicular to the first, which is attached to the posterior portion of the small intestine.[2] A lymphatic gland generally occupies the angle formed by the appendix, cecum, and the small gut; this receives all the lymphatic vessels of the appendix. The size of the latter varies greatly. Ferguson,[3] after measuring 200 appendices, gave as the average length 4½ inches (11.4 cm.), and as the diameter, that of a No. 9 English sound—about a quarter of an inch (0.62 cm.). Berry's studies, which are partly based upon personal examination of 100 bodies, and partly upon comparison of his own results with those obtained by other investigators, gives the average length in all the observations as 9.2 centimeters (3.6 inches). The caliber is ordinarily of the size of a goose-quill. Very exceptionally, as in a case reported by Swan, there is a congenital absence of the appendix. Its two fibro-muscular coats (external longitudinal and internal circular) are thick; its mucous membrane contains lymphoid elements in abundance. The blood-supply is derived from the ileo-colic artery at the valve, a single branch running to the end of the appendix. Shortly after middle life the cavity of the appendix becomes obliterated. Its blind extremity points most frequently toward the spleen. The appendix may lie behind the cecum, and sometimes partly to its inner side, its tip almost touching the liver or the gall-bladder. In not a few instances it dips downward, passing over the brim of the pelvis. There is no adjacent organ to which it may not become adherent, and in rare instances it is twisted like a loop around the small gut, causing constriction or even strangulation. Osler mentions one case in which the appendix, with the cecum, entered the inguinal canal, curved upon itself, re-entered the abdomen, and was adherent to the wall of an abscess-cavity just to the right of the promontory of the sacrum.

Pathology.—Three pathologic varieties are recognized:

(1) **Catarrhal or Obliterative Appendicitis.**—This may be acute or chronic. The term "catarrhal inflammation" is still retained, though scarcely applicable, since, as a rule, appendicular inflammation tends to spread quickly to all the coats, including the serosa. Obliterative ap-

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[1] The following names will long be connected with this disease: Pepper, Fitz, McBurney, Porter, Willard Parker, Weir, Sand, Bull, Warren, Keen, Morton, Price, J. William White, Deaver, Senn, and many others.

[2] *Sajous' Annual*, vol. i., 1893.

[3] "Some Points regarding the Appendix Vermiformis," *American Journal of Medical Sciences*, Jan., 1891.

pendicitis is descriptive and in every way preferable. The ***mechanism*** of the inflammation is briefly as follows: The mesentery being too short, the exit is too small, and in consequence of swelling of the coats (especially the mucous) the venous return is greatly impeded, then the arterial, followed often by abscess-formation. In the female a branch is supposed to be furnished by the ovarian artery, making a more perfect blood-supply. The appearances are, in the beginning, identical with those of catarrhal inflammations elsewhere in the bowel. Within twenty-four hours all the layers are swollen, with marked cellular infiltration, causing the appendix to become firm and often rigid. The mucosa may be denuded of its epithelium and present a granular surface. The external coat (serosa) is usually hyperemic, and not uncommonly the seat of fresh or old adhesions. The tube may become completely obliterated by pressure, resulting in a union between the granular surfaces, in this manner rendering subsequent attacks impossible (Hawkins). It is in cases in which this fortunate result is not reached, however, that acute appendicitis leads to the chronic form with relapses. Two additional terminations may be observed: First, an obliteration of the lumen may occur near the valve, in which case the appendix becomes dilated, and sometimes enormously so (cystic). The contained liquid may be either serous or purulent. Second, obliterative appendicitis may lead directly to ulceration of the mucous membrane, and often in the absence of a fecal concretion or foreign body. Again, the cystic appendix may ulcerate, with or without perforation. Obviously, the more marked the stenosis of the appendix the less favorable the conditions for natural drainage, and the greater the liability to recurrences of attacks of appendicitis. This variety then may end in resolution, complete obliteration, stenosis, or ulceration, and the latter sometimes in perforation.

(2) **Ulcerative Inflammation.**—Like the preceding, this variety may be acute or chronic. It may be a sequel of the obliterative form, and often accompanies chronic obliterative appendicitis. More commonly, however, it is seen in connection with concretions, and sometimes with foreign bodies also. By no means invariably, however, does the presence of these substances excite ulceration of the appendix. Micro-organisms play an important rôle in this variety (*vide* Etiology). The submucosa or muscularis usually forms the base of the ulcer. The termination may be in healing, with tendency to stricture. When obliteration is complete, dilatation beyond the seat of the latter may ensue. Again, the ulcer may extend in depth until perforation occurs.

(3) **Interstitial or Parietal Inflammation.**—This may be preceded by the obliterative or the ulcerative form, which may be followed by anemic necrosis and sloughing. Concretions or foreign bodies are often found, though specific bacteria are of greater etiologic importance. The gravest, most common, and hence the most important lesions are the gangrenous, which are usually limited to a circumscribed part of the tube. Interstitial inflammation has a single termination—perforation—and leads to appendicular peritonitis of a virulent and infectious type.

It may be that neither necrosis nor gangrene may supervene. When perforation occurs, one or more openings, ranging in size from one to several millimeters, may be observed, while the remainder of the appendix may present no abnormalities; more often, however, it is blood-injected and swollen. The appendix may slough *en masse*. The histopathologic changes may be characterized by intense cellular exudation, necrosis, or purulent inflammation. The muscular coat is hypertrophied, and chronic thickening of the appendix may result.

Consequences of Perforation.—A common result of all forms of appendicitis is a localized peritonitis, and this is a constant effect of the severer forms, either leading to (*a*) circumscribed peritonitis or to an (*b*) acute diffuse peritonitis.

(*a*) *Circumscribed Peritonitis.*—At first the surface of the peritoneum is opaque and velvety. Soon a fibrinous exudation covers the appendicular peritoneum, and quickly establishes adhesions between the appendix and the adjacent parts (abdominal wall, intestinal coils). The process may not proceed any further. Generally, however, it is soon followed by a serous or sero-fibrinous exudation, which becomes sero- or fibrino-purulent, and often forms the so-called perityphlitic abscess. The seat of the abscess is always near the tube, and is as varying as the position of the appendix; its size is also extremely variable, as it sometimes contains enormous amounts of pus. Among the most common locations are—McBurney's point, the vicinity of the cecum, the coils of the small intestines (near the umbilicus), and, more rarely, in the pelvis below. The pus contained in the abscess is rarely thick, grayish-yellow in color, and emits a fecal odor; more commonly it is thin, turbid, dark-gray or greenish in color, and has an extremely fetid or even gangrenous odor. The process of gangrenous sphacelation *en masse* is often completed after the limiting wall of adhesion has formed, when the entire appendix is found free in the pus-cavity.

The abscess may be *subperitoneal*, as when perforation occurs into the retro-cecal connective tissue, and the term "iliac abscess" was formerly applied to these extra-peritoneal purulent collections. They are rare, however, since the early operation has been employed. Their situation and dimensions depend upon the direction taken by the appendix. The latter may pass downward, and the pus is then apt to accumulate in the lower part of the iliac fossa, and may point and finally burst in the neighborhood of Poupart's ligament, with subsequent recovery. Occasionally under these circumstances a fistula remains for an indefinite period of time. The appendix may touch various abdominal structures, and the pus in following the line of least resistance may cause spontaneous rupture into the rectum, bladder, or the vagina when it points inward; and into the perinephric region or into the pleural cavity (through the diaphragm) when it points upward; or even into the cecum or colon. The contents of the abscess may also find their way through the abdominal wall in the vicinity of the umbilicus. The psoas muscle may conduct the abscess downward, and it may then point at the hip-joint or gain the gluteal regions or the scrotum, producing the so-called "scrotal appendicitis." The appendix has also been found in a hernial sac. Among the rare lesions to be noted are erosion of one of the arteries of the iliac region (causing fatal hemorrhage) and pylephlebitis. From the thrombi in the mesenteric veins in the latter condition infectious emboli may be conveyed to the liver, giving rise to hepatic abscess; this occurred in a case of my own at the Episcopal Hospital, Philadelphia. The abscess may also be due to an extension of the thrombo-phlebitis of the mesenteric veins that lead from the appendix to the portal vein. Thrombosis of the iliac veins with edema of the corresponding leg may also arise, and these veins may, during the process of healing, become compressed, with a resulting edema of the leg, as I have witnessed in two cases. It rarely happens that suppurative processes are both extra- and intraperitoneal.

(*b*) *Acute Diffuse Peritonitis.*—This follows perforation when previous adhesions have not taken place or when, having formed, they yield. Generalized peritonitis may also follow the circumscribed form, the lesions being propagated to the entire membrane by direct extension. The morbid changes are those mentioned in the description of Acute Peritonitis. Since the early operation has been employed peritonitis has been the result, usually, of direct perforation before a limiting wall of adhesion has been formed.

Etiology.—**Predisposing Causes.**—(*a*) Doubtless certain ***congenital structural defects*** aid in the production of appendicitis. Among them are unnatural length, location, and arrangement of the organ; also the shape of the meso-appendix and Gerlach's valve. These factors tend to obliterate the lumen of the canal by producing kinks and twists, thus favoring the collection of material within the appendix. (*b*) ***Strictures***, particularly near the cecal end of the tube, and adhesions due to old inflammation, especially peritonitis, operate in the same manner as the preceding, only with greater power. (*c*) ***Fecal concretions*** are the main cause in nearly one half, while ***foreign bodies*** play a small rôle, having been present in 7 per cent. only of 1400 cases (J. F. Mitchell). The calculi form in the appendix itself (Rochaz). The foreign bodies are very various, and consist of seeds, worms, gall-stones, pills, bristles, and, more rarely, pointed bodies, as fish-bones or pins. The presence of fecal concretions and foreign bodies is often tolerated by the appendix without symptoms or local pathologic changes; hence they are looked upon rather as a predisposing than as an exciting cause of appendicitis. (*d*) ***Ulcers*** (tuberculous, typhoid, and, rarely, actinomycotic) may also produce this affection. (*e*) ***Straining Efforts and Traumatism.***—Not uncommonly excessive muscular exertion, traumatism, or jarring of the body as in jumping, act as favoring causes. (*f*) *Age.*—The disease is especially frequent in young adults between the fifteenth and thirtieth years. It is not very infrequent in childhood, however, after the third year, and it has even been seen in persons over seventy years of age. (*g*) *Sex.*—Appendicitis attacks males oftener than females; this fact has been explained (*vide supra*). In the female it is rarely of adnexal origin. Adhesions between the tube and ovary and the appendix may occur, the morbid process then extending to the latter. (*h*) *Gastro-intestinal Disturbance.*—Indiscretions in the diet may precede a primary attack, and are of paramount etiologic importance in the recurrent forms of the malady. (*i*) *Heredity.*—That this plays no mean rôle in many cases of appendicitis I have long felt convinced. This serves as the explanation of those cases in which rheumatism and uric-acidemia seem to act as causal agents. (*j*) Evidence to show that influenza and other affections may cause appendicitis is not wanting. (*k*) It is not improbable that *poor blood-supply* and retrogression of the organ, plus torsion and the like, are the leading predisposing factors.

Bacteriology.—The combined results of several experimentalists tend to show that no special organism plays an exclusive *rôle* in this disease, but the studies of Hodenpyl indicate that the *bacillus coli communis* is the bacterium most generally present: it is well known, moreover, that this bacillus becomes pathogenic when it escapes into tissues in which it does not naturally belong. A. O. J. Kelly found this organism present alone in 73.4 per cent. in 94 instances of acute appendicitis; alone in 89.71 per cent. of 107 cases of chronic appendicitis. Barbacci emphasizes the etiologic importance of the passage of the intestinal contents into the peritoneal cavity—*i. e.* the chemical factor. Of other specific bacteria, those of *typhoid* and *tuberculosis* are not uncommonly found to be present. The *streptococcus pyogenes* may also be found to produce the most virulent infection, and the *staphylococcus pyogenes aureus*, the *proteus*, and other specific organisms have been found. The great frequency of appendicitis is rendered appreciable by the numerous favoring factors (including the congenital conditions) acting upon the appendix, which naturally has an exceedingly low vitality; also by the constant presence of one or more organisms that are known to become pathogenic in the presence of a slight lesion.

Clinical History.—Doubtless many cases are overlooked because of the extreme mildness of the symptoms. These are often attributed to intestinal indigestion or to a "cold," to which the patient pays little attention unless he displays unusual susceptibility.

The *onset* of acute appendicitis may be slow and gradual, but oftener it is *quite sudden.* A clear history of some obvious cause (an error in diet or muscular effort) may be obtainable. Again, preceding the onset of the definite symptoms and extending over a day or two, there may have been *certain prodromes*, as impaired appetite, nausea, constipation, or diarrhea. In slow cases the local and general symptoms are at first slight, but gradually increase in severity as the different stages of the disease are evolved. Indeed, in the latter class the patient may go about his customary duties during the attack with ill-defined rational symptoms, while in reality suffering from periappendicular abscess. These patients run two serious dangers—first, spontaneous rupture of the abscess into the peritoneal cavity may occur; and secondly, the slow septic absorption may suddenly overwhelm the system. As a rule, the sudden cases develop in seeming perfect health, and are sometimes heralded by a rigor or chilliness.

The characteristic features of the invasion are *abdominal pain, fever, tenderness over McBurney's point, circumscribed resistance, gastric disturbances*, and, as a rule, constipation. The *pain* varies in intensity from a mere feeling of soreness to that of the most agonizing suffering. It may be paroxysmal, though oftener it is constant, with moderate exacerbations. Severe pain points to an involvement of the peritoneum and signalizes a danger of perforation. At first the pain may be referred to any point in the abdomen; later within forty-eight hours it becomes more distinctly localized in the ileo-cecal region.

Elevation of Temperature.—The exacerbations may at first touch

102°, 103°, or even 105° F. (38.8°–40.5° C.), and particularly in children; more commonly they range from 100° to 102° F. (37.7°–38.8° C.). The degree of fever is unreliable, however, as a criterion of the severity of the case, since the worst cases may have a subnormal temperature throughout.

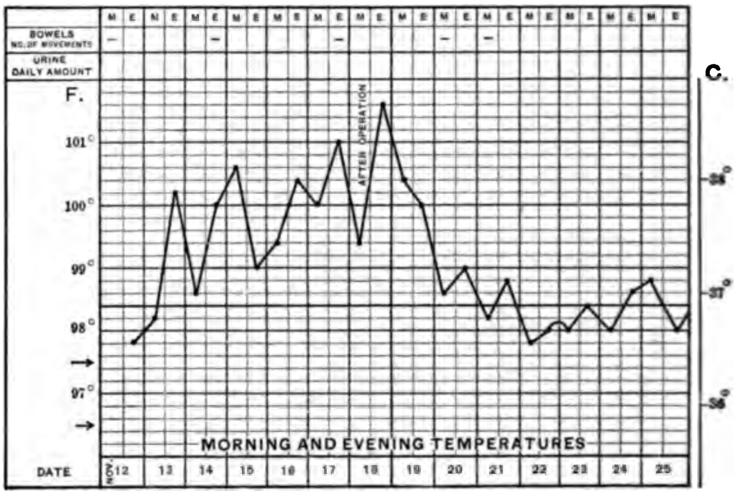

FIG. 56.—Temperature-chart of a case of appendicitis. M. M——, aged thirty-five years; motorman. Laparotomy, by Prof. E. Laplace, disclosed catarrhal appendicitis with adhesions.

An elevation of temperature, however trivial, is most significant, pointing as it does to inflammation as the cause of the local symptoms. The pulse-rate is somewhat higher than the elevation of temperature would lead one to expect, and in bad cases the pulse is usually much quickened. Sometimes, however, it remains at 80 to 90 per minute, and may be full and soft, even though the patient be practically moribund.

Fixed *tenderness* is practically constant on pressure over a limited area, midway on a line between the anterior superior iliac spine and the umbilicus (*McBurney's point*), and is a most valuable sign. The seat of the tenderness may rarely be found at other points, depending upon the location of the appendix. I have twice observed it in the lumbar, once in the right hypochondriac region, and once far below the usual point, in the right iliac fossa. It has been found in the umbilical and left iliac regions, in the pelvis, and in the groin. In several instances, although I have found it elsewhere in the early stage, it has shifted to McBurney's point later. On the other hand, it may move from the usual position in cases that are allowed to drag on. When the sensitive area is at McBurney's point, as is the rule, the gentlest pressure often suffices to elicit exquisite tenderness, but when it is situated elsewhere firmer pressure with the finger-tips is usually required. Deep pressure always reveals localized tenderness at some point in the abdomen if the case is one of appendicitis. ***Palpation*** also detects an abnormal tenseness of the right rectus abdominis muscle. On or about the second day a *circumscribed induration* manifests itself, followed soon by a fulness and swelling tending to obliterate the depressions above and in front of the anterior iliac spine. The position of the indurated area varies according to the location of the appendix, but is usually found at or in the vicinity of McBurney's point. Sometimes a resistant mass of the shape and size of an enlarged appendix is palpable. In such cases peritoneal exudation has not as yet occurred to any great extent. In some cases the induration is diffuse at first, but assumes the usual circumscribed form later; it may, moreover, be so deeply seated as not to be appreciable. The degree of tenseness of the two recti muscles—right and left—should be compared, though an absence of tension of the right rectus does not, I feel certain, eliminate the possibility of appendicitis. The results of *percussion* furnish no certain guide. As a rule, the note on light percussion differs from that on the opposite side; on deep percussion a dull tympany or a circumscribed area of dulness can be outlined. This deadness may be due in great part to the presence of fecal matter in the adjacent coils of intestine. While at the start the abdomen may be flattened or even retracted, tympanitic distention afterward appears, particularly in the cecal region, giving rise to exaggerated tympany on percussion.

Less characteristic, though still of diagnostic worth, are certain other symptoms. At the beginning *vomiting* usually occurs, unless there be diarrhea, and is attended by more or less nausea; it may continue throughout the course of the attack. In most cases, however, after a few fits of vomiting the symptom disappears, though it may recur if errors in diet be committed or if peritonitis supervene. During the attack constipation is the rule, though diarrhea, which sometimes precedes appendicitis, may also occur at a late stage as a septic symptom. There is anorexia, and the tongue is coated. The *decubitus* is dorsal, with the right leg flexed. Frequent micturition (early) and retention of urine (later) are not uncommon, the urine having a deep color-tint, and sometimes containing albumin.

The case may follow a mild *course*, terminating in resolution with recovery; or it may be of a severe type and develop perforation, with the formation of abscess or diffuse peritonitis. As graphically stated by Fitz, it is impossible to obtain statistical evidence on a large scale of the relative frequency of these alternatives, and hence the frequency of treatment of appendicitis by abdominal section. From all available data, however, it would appear that in more than one-half of the cases the course is light and favorable.

If not operated upon early, the fever may continue for three to five days, and then subside, with simultaneous abatement of the severe local and general symptoms and with the establishment of convalescence. The same amelioration of the symptoms may be brought about by early free purgation, either as the result of salines or, rarely, spontaneously. In these instances resolution takes place even after invasion of the peritoneum. Small abscesses may be absorbed, and usually in cases terminating in resolution perforation has not occurred. Infection of the peritoneal membrane directly through the appendix is not uncommon.

In severe attacks perforation may occur, with the development of localized peritoneal abscess or generalized peritonitis (*vide* Pathology), and it must be remembered that cases that begin gradually may also show a tendency toward perforation. When this event occurs early in the course of a severe attack or after a protracted mild appendicitis the symptoms of *local* or *general peritonitis* are superadded. If early, the symptoms pointing to peritonitis are intense; the abdomen swells quickly, and is exquisitely tender, though the physical signs of a tumor are absent. The temperature often falls, and the characteristic vomiting and circulatory collapse appear. Often the generalization of the peritonitis is marked by less violent symptoms. Starting from the seat of circumscribed inflammation, the pain and tenderness propagate themselves noticeably from day to day until every portion of the peritoneum has been invaded. Besides progressive augmentation in the local features, including the pain, there is a gradual failure in cardiac power, as shown by the condition of the pulse; vomiting also returns, and at last becomes fecal. Death results from asthenia, and sometimes suddenly when unanticipated. If perforation occurs later, sufficient time has been allowed usually for the inflammation to become circumscribed, in which case the localized abscess is generally intra-peritoneal; it may, however, rarely be extra-peritoneal. The local symptoms intensify, the pain becomes excruciating, and the spot of tenderness may rapidly extend itself in all directions, particularly downward. Vomiting sets in, and may become troublesome, and constipation is absolute, not even gas escaping from the rectum. Retention of urine is common.

Physical Signs of Localized Abscess.—*Inspection* shows distention of the belly, the affected area being prominent, with an obliteration of the natural depression in the right iliac region. *Palpation* discovers induration and great tension that soon yield to pressure (doughy), and edema of the skin. If the abscess is superficially seated, fluctuation may be appreciated on bimanual palpation. Deep-seated tumors are not uncommon, and then fluctuation is detected with difficulty or not at all. An examination *per rectum*, with a view to determining whether the abscess has gained the pelvis, is highly important. Counter-pressure above with the free hand aids materially. In doubtful cases bimanual pelvic examination should not be neglected. *Percussion* reveals dulness if the abscess be superficial. A tympanitic note, however, is often elicited, due either to an intervening coil of intestine or to the gas contained in the sac of the abscess.

If active peritonitis and septicemia do not develop, the constitutional as well as the local symptoms may abate, and the patient leave his bed, carrying with him, however, the abscess. The latter may point somewhere in the right lower quadrant of the abdomen or in the lumbar region. There is also a strong tendency toward spontaneous rupture into the rectum, bladder, vagina, or cecum. Often, preceding the discharge of pus into these organs, the latter display marked irritability, particularly the rectum and bladder. There is always the danger that the contents of the abscess may find their way into the general peritoneal cavity. The symptoms of hepatic abscess may develop at an advanced stage. The pus may traverse the abdomen in the upward direction until it touches the diaphragm, when the symptoms of subphrenic abscess may be manifested. Extension through the diaphragm may now occur, causing pleurisy or pericarditis, and a pleuro-fecal fistula may thus be established.

The general symptoms undergo a modification, due to the *suppurative* *process.* Rigors or a decided chilliness may occur. Diarrhea often succeeds to previous constipation, and drenching sweats to a dry skin. Improvement and even spontaneous cure may ensue if spontaneous rupture into one of the outlets of the body should occur. The fever (Fig. 57) may be either remittent or intermittent, and if the localized inflammatory process be active, the usual pronounced features of septicemia are predominant in the clinical picture. The latter specially grave condition often drifts into an extreme typhoid state with a hopeless course.

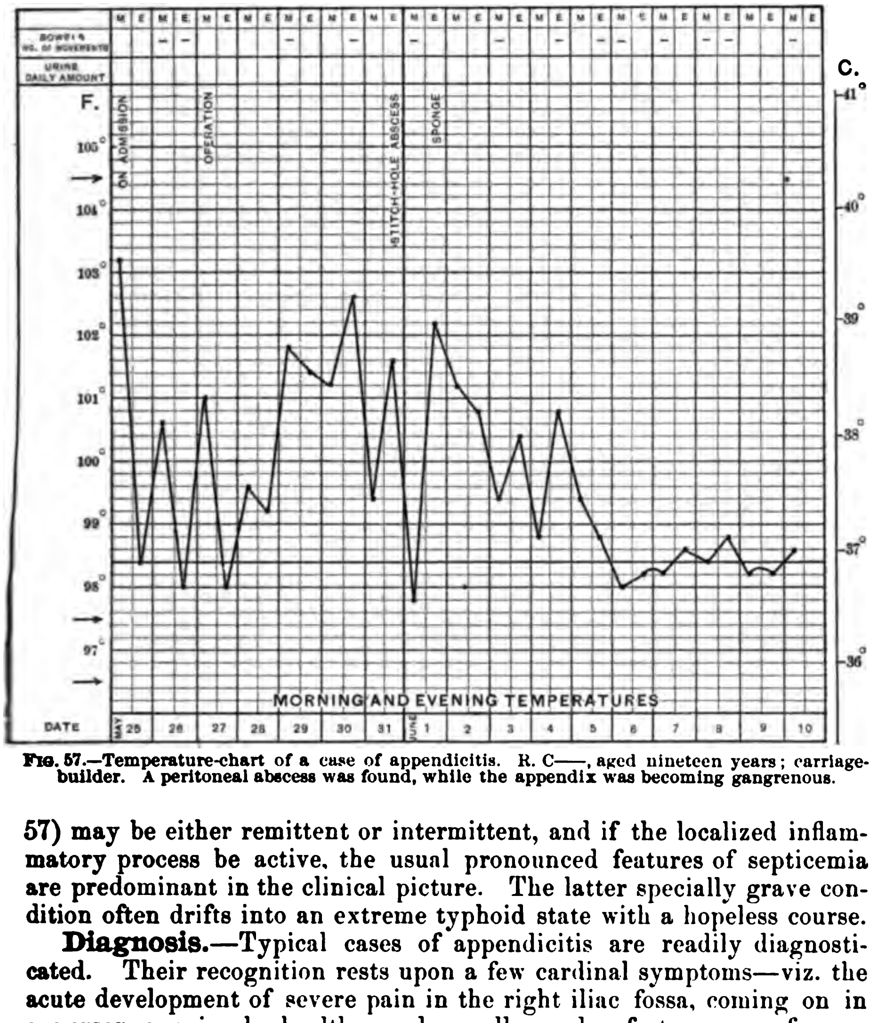

FIG. 57.—Temperature-chart of a case of appendicitis. R. C——, aged nineteen years; carriage-builder. A peritoneal abscess was found, while the appendix was becoming gangrenous.

Diagnosis.—Typical cases of appendicitis are readily diagnosticated. Their recognition rests upon a few cardinal symptoms—viz. the acute development of severe pain in the right iliac fossa, coming on in a person previously healthy and usually under forty years of age; appendicular tenderness, unilateral induration, fever, vomiting, and constipation, or, more rarely, diarrhea. Atypical cases, however, may offer difficulty, although Morris affirms that errors in diagnosis are less frequent than in almost any other disease. Often the pain is, for a time, referred to a circumscribed area far removed from the usual site of the appendix, and rarely it continues without a change of situation throughout the attack. In the latter case the local lesions may occupy the usual, though oftener they have an unusual, position. Thus, when the pain is referred "due east," or to the left iliac fossa, with bilateral induration, the appendix will be found in the pelvis (Deaver). In such instances a rectal and a bimanual vaginal examination are imperative. It should be an unvarying rule in all cases of severe abdominal pain to palpate with the finger-tip every square inch of the abdomen if necessary, to find the localized tenderness when it is not found at McBurney's point. The degree of tenderness sustains a close relationship to the severity of the local inflammation as long as the condition remains strictly localized, but this relationship is lost when generalization occurs. With the appearance of a circumscribed induration and of the intense local tenderness and pain it is reasonably sure that perforation either has occurred or is impending. Perforation, however, may occur without local induration, and even after subsidence of the acute pain and excessive tenderness. Gangrenous appendicitis is *most deceptive*. The very acute symptoms, including the fever, may disappear, and unless the physician be upon his guard the patient will be considered convalescent and be allowed to go about. Rupture of the abscess now occurs unexpectedly into the peritoneal cavity, intestines, or some other direction, or a large-sized abscess develops with the usual signs and symptoms.

Differential Diagnosis.—*Typhlitis, and especially the Massing of Feces in the Cecum.*—These are truly rare conditions. According to McBurney, 99 per cent. of all typhlitic abscesses are of appendicular origin, and of 400 autopsies by Einhorn 91 per cent. had this origin. Ball and others have performed laparotomy for ulcerative cecitis, but this condition cannot be recognized during life. Stercoral typhlitis is discriminated from true appendicitis by the precedent constipation, which may become absolute, by the dragging character of the pain, the late-appearing fever, and chiefly by the physical signs, which indicate the presence of a superficial, sausage-shaped tumor that is often doughy and extends vertically from a point near the right costal border "southward" through the ileo-cecal region. Percussion elicits dulness over the seat of the tumor. The localized tenderness and circumscribed resistance of acute appendicitis are wanting, and a thorough emptying of the large intestine usually cures stercoral typhlitis.[1]

Renal Colic.—The absence of fever and of a localized spot of tenderness and induration, and the presence of hematuria are points that distinguish this affection from appendicitis.

Indigestion.—Digestive disturbances, and particularly pain and vomiting, accompany appendicitis. When they occur independently of appendicitis, however, they can be relieved, and the appendicular region remains free from fixed pain, tenderness, or tumor.

Cholecystitis with Distention.—This gives rise to a superficial, mobile, pear-shaped tumor, with or without jaundice—features not met with in appendicitis. Osler, however, mentions a case of this sort in which the diagnosis was undetermined until laparotomy was performed.

Perinephric Abscess.—Without a history indicative of chronic renal disease or of nephro-lithiasis the differentiation cannot be made except by exploratory incision.

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[1] It is highly probable that the term "stercoral typhlitis" is synonymous with chronic appendicitis with retained feces in the cecum.

Acute Peritonitis, due to Ovarian or Tubal Disease.—When the appendix occupies, not its usual seat in the iliac region, but the pelvic fossa, then the distinctions between salpingitis and appendicitis are not easily drawn. Right ovaritis, owing to the presence of pain, tenderness in the right iliac fossa, and fever, often closely simulates appendicitis. In the former tenderness is less pronounced, and the organs of utero-gestation manifest certain disturbanees of function. A clear history, coupled with a careful pelvic examination, will usually complete the clinical separation of these two conditions.

Extra-uterine Pregnancy.—In this condition the menstrual history furnishes important information. There is, in addition, profound collapse, due to hemorrhage, when rupture of the adhesions occurs. Elevation of temperature is absent. The localized tenderness and increased resistance are lower in the pelvis than in appendicitis.

Acute Tuberculous Peritonitis.—As in appendicitis, so in tuberculous peritonitis, pain, tenderness, and fever are present, but in the latter the onset is more gradual, and the signs of tumor and increased resistance in the ileo-cecal region are absent. Movable dulness may be present in the tuberculous affection, but not in appendicitis until the peritonitis is generalized. The lungs generally show lesions in tuberculous peritonitis.

Acute Intestinal Obstruction.—When this is due to intussusception there may be signs of a tumor, but not at McBurney's point; the tenderness over the site of the mass is less intense, while the frequent bloody discharges that are seen in this condition, accompanied by tenesmus, do not characterize appendicitis. When obstruction is caused by strangulation stercoraceous vomiting is apt to occur, and is absent in appendicitis. Pain, local tenderness, and, not uncommonly, signs of a tumor appear, but elsewhere than at McBurney's point. Some of these instances, however, remain obscure till celiotomy is performed.

Acute Hemorrhagic Pancreatitis.—This affection simulates appendicitis with generalized peritonitis. But the deep-seated epigastric pain, followed by circumscribed resistance in the same region (a grouping absent in appendicitis), should arouse strong suspicion of pancreatitis.

Hip-joint Disease.—In both hip-joint disease and appendicitis the dorsal decubitus with flexed leg is noted. If the patient be anesthetized, however, full extension of the leg and a normal condition of the hip-joint are easily demonstrable in appendicitis.

Typhoid Fever.—Mild cases of appendicitis with accompanying diarrhea bear a close superficial resemblance to typhoid fever. In typhoid fever, however, the onset is more gradual and the fever-type more continuous than in appendicitis. In typhoid the stools are somewhat peculiar, the spleen is swollen, there is dulness of intellect, bronchitis and the characteristic eruption attend,—all features that are absent in appendicitis. The diazo-reaction, if present, would strengthen the diagnosis of typhoid, and a response to Widal's test would be conclusive. In appendicitis the local features, and in typhoid the general, are predominant.

Dietl's Crises.—In a case of movable kidney which I saw recently all the symptoms pointed to appendicitis. An operation was about to be performed when a sudden subsidence in the abdominal swelling and local induration occurred. The kidney was subsequently detected in an abnormal location (*vide* Mobility of the Kidney).

CHRONIC APPENDICITIS.

(*Relapsing Appendicitis.*)

Relapses occur in nearly one-half the total number of persons who have suffered from a primary attack of appendicitis. In most of these cases there is constantly present a slight local discomfort during the interval; in a small percentage, however, there is an entire freedom from uneasiness. The *local symptoms* in those having had an antecedent peritonitis are more pronounced than in the first attack, but after a number of recurrences the symptoms are likely to be less severe with each new attack. The most constant symptom between attacks is a subacute form of *pain* that is liable to manifest exacerbating periods with slight fever. Physical fatigue, a strain, and errors in diet causing gastro-intestinal disorder are very likely to induce a relapsing appendicitis. Chronic appendicitis strongly favors the retention of fecal matter in the cecum, thus forming so-called *stercoral typhlitis.* This association was formerly mistaken for primary typhlitis. The characteristics on which the diagnosis is based during the attack are similar to those detailed under Acute Appendicitis; the course is, however, somewhat more condensed than that of the acute form.

In the intervals between the attacks the appendix can be readily appreciated on *palpation*, the method employed by Edebohls being preferable: "The patient lies upon his back with the examiner at his side; the latter places his right hand upon the patient's abdomen over the right rectus muscle, opposite the anterior superior spine of the ilium, and presses the left hand upon the right, so that no force is used by the right hand and the tactile sense of its fingers is left undisturbed. The hands are drawn slowly outward, allowing the contents of the abdomen to slip from underneath them. The coils of intestine can be felt as they escape from under the hand as it presses against the posterior abdominal wall."[1] In this way the appendix may be felt as an elongated tumor of the size and shape of the little finger. If there be only a slight exudation present, the appendix often appears to be immediately beneath the abdominal wall. It may, however, be deep-seated, even though the exudation with adhesions be absent. Both pain and tenderness are pronounced, and particularly if pus be present.

The results of chronic appendicitis upon the general health and nutrition of the patient are quite noticeable, and tend to augment as time passes, if the attacks be frequent or the intervals between them grow shorter. The chief symptoms are those of a nervous type; emaciation and debility are also observed. The associated nervous symptoms are those of neurasthenia. These patients often become introspective and exceedingly irritable, the mental condition being accounted for, to a great extent, by the consciousness that there is ever present the overhanging danger of a fresh attack with serious possibilities.

Differential Diagnosis.—*Carcinoma of the Cecum.*—This presents many points of similarity to chronic appendicitis. I have under my care at present a lady aged sixty years suffering from chronic appendicitis, whose case had been diagnosticated as carcinoma of the cecum, and for a considerable time my own view coincided with that of my predecessor.

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[1] B. Farquhar Curtis: *Twentieth Century Practice of Medicine,* vol. viii.

The occurrence from time to time, however, of relapses, during which the feces were massed in the cecum and fever arose, soon indicated the correct diagnosis. Besides the absence of periodic attacks of fever, the general features—loss of flesh and strength, anemia—are more steadily and rapidly progressive in carcinoma of the cecum. The history of the mode of onset also aids in the distinction. Pain, tenderness, and a resistant tumor are common to both affections.

Hypochondriasis and Hysteria.—Hypochondriasis and hysteria may lead to the manifestation of morbid feelings simulating those of appendicitis. Such cases may show merely a greatly exaggerated uneasiness, or such an increase of sensibility as to cause the patient to complain of pain in the right iliac fossa. In addition, there may be localized tenderness. I recently witnessed the removal of the normal appendix from an hysteric female in whose family two genuine cases of appendicitis had occurred not long previously. Hypochondriasis and hysteria distinguish themselves by the antecedent history and by the absence of a tumor-mass and of increased resistance; there is also an absence of localized tenderness if the patient's attention be withdrawn. In such subjects oxaluria is not infrequent, and it is possible that irritation of the right ureter by the passage of crystals of calcic oxalate, as mentioned by Cabot, may explain the localizing of the discomfort (Wood and Fitz[1]). I recently saw a case of this sort in a neurasthenic medical student.

RECURRENT APPENDICITIS.

When successive attacks occur in the same individual at intervals varying from several months to a year or more, each new attack is spoken of as a *recurrent appendicitis*. Severe attacks may succeed light ones, or, conversely, mild recurrent may follow severe preceding attacks. I recall several cases in which rudimentary appendicitis (indicated merely by colicky pain) occurred, and lasted from a few hours to a day or two. Often the illness is too trivial to lead the patient to consult a doctor. An absolute diagnosis demands, besides the subjective symptom, pain, the presence of localized tenderness (with or without induration), and elevation of the temperature. In several subjects of recurrent appendicitis formerly under my care the last attack occurred three or four years ago. That each new attack may be the last is always to be remembered.

Prognosis.—In forming the prognosis in a given case of appendicitis the same rules may be followed as in the case of acute infectious diseases. To estimate the severity of the type of infection, however, is not a simple matter. Unlike many of the acute infectious diseases, the height of the temperature and, to a lesser degree, the rate of the pulse are unreliable guides in appendicitis. Broadly speaking, however, in the severer forms the local process exhibits a strong tendency to spread; the temperature and pulse are relatively high, and there is an intense appendicular intoxication. These are the cases that suppurate or result in perforative peritonitis (often rapidly spreading), and in pericecal abscesses. They are among the gravest of known conditions. Of this fatal group of cases not less than 68 per cent. die before the eighth day.

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[1] *The Practice of Medicine*, p. 886.

The development of *fulminant peritonitis* or of a peritoneal abscess after perforation is attended by a falling temperature, though subsequently the latter may mount high or become markedly irregular.

On the other hand, in the mild forms that are included in the name *catarrhal appendicitis* recovery is the unvarying rule. These lighter cases often lead to adhesive peritonitis—a circumstance that strengthens the view that they are of an infectious nature. The temperature is only moderately elevated as a rule, and the pulse-rate correspondingly quickened. Both pulse and temperature indicate marked improvement on the third or fourth day, while the pain and localized tenderness disappear. In this connection the deceptiveness of gangrenous cases must be recollected (*vide supra*, Diagnosis). The complications that are most likely to arise and other points of prognostic significance have been fully stated in the Clinical History. The general mortality of appendicitis is about 14 per cent. (Fitz). Improved methods, chiefly surgical, of dealing with the disease have, however, greatly reduced its death-rate. The prognosis in *chronic appendicitis* is most uncertain; after the patient has survived several attacks it is on the whole more favorable.

Treatment of Appendicitis.—Whether imminent danger of perforation exists or not, the physician who is called to a case of appendicitis should at once request the services of a competent surgeon. Few surgeons subscribe to the doctrine that all cases demand operation; but, since it may become necessary to perform celiotomy at any hour thereafter, the latter should help to settle the important question: "When is it necessary to operate in the case?" The physician who does not pursue the course above recommended falls short of his duty, both toward the patient and toward the surgeon on whose skill he relies to remove safely the source of danger. Surely, in a disease that so often baffles both physician and surgeon, suddenly developing, as it sometimes does, a fatal virulence without previous unfavorable symptoms, they should stand guard together from the moment the case is diagnosticated or appendicitis is strongly suspected. Unfortunately, both the medical and surgical treatment of appendicitis have each been recommended with great earnestness by their respective advocates.

With rare exceptions, prompt surgical intervention should be recommended. The indication for an immediate operation is undoubted in all cases of acute appendicitis, whether marked by sudden and severe or mild invasion-symptoms, if seen at the beginning of the attack, and free purgation at the earliest possible moment is not followed by decided relief. A waiting policy and medical treatment are also perilous in doubtful cases. Obviously, the conditions are less favorable for operation after a case has progressed to the beginning of abscess-formation—*i. e.* from the third to the fifth day of the illness. It is at this period that the peritoneal inflammation tends to circumscribe itself by the formation of adhesions. Hence, as Richardson states, it is "too late for an early operation, and too early for a safe late operation," since there is great risk of infecting the general peritoneal cavity. Whether it is wise to allow the appendix to remain after adhesions have been formed in some cases, and merely to drain, cleanse, and pack the cavity, cannot be discussed here. The mild attacks that develop in the course of chronic appendicitis after numerous previous seizures need not excite alarm. Under such circumstances operation should be undertaken between attacks, when the mortality is practically *nil.* On the other hand, in cases that have been allowed to drag on until general peritonitis has set in, treatment by operation is not advisable. Moreover, the most ardent advocate of immediate operative treatment is sometimes compelled to rest satisfied with medical measures. Such cases are those in which there are associated chronic affections (advanced diabetes, Bright's disease), not to speak of those in which the patient declines operation. Hence there is a medical treatment of appendicitis, but it should not be the treatment of election.

General Management.—The patient should be kept in bed in a quiet, well-ventilated apartment, and in no affection is the value of *absolute rest* in the treatment of inflammation greater than in appendicitis. The *diet* should be liquid and nutritious, consisting chiefly of pancreatized milk and concentrated broths. All articles of food that tend to undergo fermentative changes, and all carbonated drinks, should be prohibited, since they increase meteorism. The patient should be under- rather than over-fed. At the start, and particularly if a sausage-shaped tumor be present, intestinal irrigation, oft-repeated with a view to removing the fecal matter, must be carried forward assiduously. Saline laxatives (Rochelle salts, ʒij—8.0—every hour or two, preceded by a dose of castor oil or a few fractional doses of calomel) are to be administered until the evidence of their action upon the bowels has been definitely noted. There almost never exists a contraindication to the use of *saline aperients* at the onset of the attack, and they constitute the best known means of obviating, as well as limiting, the spread of peritonitis by depleting the portal system and emptying the bowels. If commenced early, they may be continued throughout in doses sufficient to produce two or more daily evacuations. In the event of a development of evidences of peritonitis with pus-formation, salines should be pushed vigorously, unless an operation can be promptly performed. I am aware that many authors advocate withholding purgatives when indications of suppuration appear, but I have yet to see a case in which perforation has followed an active saline treatment. I avoid the use of high enemata in progressive cases, since they are more apt than salines to induce rupture of the sac.

As regards the use of opium professional opinion is not united, though a general tendency toward the limitation of its use to the minimum amount necessary to alleviate pain is happily noticeable; unless demanded by excessive suffering it had better be omitted altogether. When necessary, it is best administered hypodermically in the form of morphin (gr. $\frac{1}{12}$–$\frac{1}{8}$—0.0054–0.0081). The greatest objection to the use of opium is its effect in veiling the symptoms that assist the physician in forming a judgment as to the prospects and progress of the case.

Local Measures.—The suspended ice-bag is an excellent means of combating the pain, and often obviates the necessity of an internal use of opium. Instead of the ice-bag, cloths wet in cold water may be applied and changed every few minutes. In the early stage a few leeches may be beneficial in their effect upon the local inflammation. Blisters, however, are rarely advisable, and are particularly objectionable should the patient afterward be submitted to an operation. Mild forms of counter-irritants (mustard-paste) are preferable, though these also render the skin and underlying tissues hard and leathery.

Management of Convalescence.—The patient should not be allowed to leave his bed for several days after the disappearance of all symptoms; even the mildest forms of exercise should not be undertaken for at least one week subsequent to getting out of bed. During convalescence the diet must be carefully guarded, and the bowels, at all hazards, kept in a soluble condition. It is questionable whether drugs will aid in the absorption of the exudate or assist in resolution.

INTESTINAL OBSTRUCTION.

(*Ileus.*)

Definition.—An acute or chronic, complete or partial, occlusion of the intestinal canal.

Pathology and Etiology.—The causes of intestinal obstruction may be divided, at once most simply and practically, into the (1) *acute* and (2) *chronic* forms. In the former variety the narrowing or closure develops very suddenly or rapidly, and usually in the small bowel; in the latter, the large bowel is commonly affected by pathologic conditions that develop gradually and narrow its lumen (usually in advanced years).

Acute.—(*a*) *Strangulation.*—In the order of frequency, this is first among the causes of acute intestinal obstruction. It is produced most often by bands of adhesion, the result of a former recent or remote peritonitis, and is most commonly situated in the right iliac fossa. Incarceration of the bowel from flexions and adhesions not rarely follows upon abdominal section for the treatment of pelvic disease in women.

The usually free end of Meckel's diverticulum is sometimes attached to the abdominal wall, and may thus cause constriction of a loop of bowel. This diverticulum is the remains of the fetal omphalo-mesenteric duct, and arises from the ileum about half a meter (1.64 ft.) from the ileo-cecal valve. A similar constricting band is formed by a cord representing one or more of the obliterated omphalo-mesenteric vessels. The adhesive attachment of the free end of the appendix vermiformis may also form an opening through which the bowel may be caught.

Internal strangulation (hernia) may be the result of forcing a portion of bowel through a slit in the omentum or mesentery, or into peritoneal diverticula and openings, such as the duodeno-jejunal fossa (*Freitz's retro-peritoneal hernia*) or the foramen of Winslow.

Meyer has described acute obstruction from the passage of the small intestine into a pocket of the peritoneum in the posterior abdominal wall.

Diaphragmatic herniæ are not of extreme rarity, and may be either of congenital or traumatic origin. Most cases of intestinal strangulation occur in males during early adult life.

(*b*) *Intussusception.*—*Invagination* is the descending "telescoping of one section of the bowel into another," probably caused by a circumscribed, irregular peristalsis of the intestine. The effect of the latter state in producing invagination may be either a thrusting forward of the receiving portion by a contraction of the longitudinal muscular coat (Nothnagel), or a thrusting inward and downward of the portion immediately above by means of an increased or spasmodic peristaltic action. Thus, a cylindric or sausage-shaped tumor results, varying from a half inch to over a foot (1.3–30 cm.) in length. The layers met with in intussusception are the outer or receiving, called the *intussuscipiens*, the middle or returning layer, and the inner, called the *intussusceptum*. The seat of invagination is most commonly at the ileo-cecal valve, though it is often found in either the ileum or colon alone. Sometimes the intussusception occurs and is detected in the rectum. A lateral or partial invagination, more or less chronic, may also occur, due to the attachment of a tumor within the bowel.

The intussuscepted portion of intestine is usually the seat of peritoneal adhesions and considerable tumefaction, so that in pronounced cases the parts are so firmly agglutinated that reduction is wellnigh impossible. The engorgement may pass into an intense local inflammation, with final necrosis and sloughing, and even the discharge *per rectum* of the invaginated portion; or a fatal termination may be ushered in by perforation of the bowel.

Intussusception occurs most frequently by far in children prior to ten years of age, in whom also the disease is more acute than in adults. Males are more subject to invagination than are females.

Invagination is asserted to be an occasional consequence of the operation of circular enterorrhaphy and of lateral anastomosis by plates (Robinson).[1]

(*c*) *Volvulus.*—Twists of the intestine are met with most commonly at the sigmoid flexure of the colon. An unusually long or relaxed mesentery predisposes to the condition, so that the axis of twisting may either consist of the mesentery itself or frequently of the bowel. Not rarely the pedicle of the volvulus contains both a twist and a sharp bend in the bowel, causing complete acute strangulation. The latter condition may be pronounced in such cases, or at least be hastened, by the accumulation of the intestinal gas and of masses of feces, or by bowel-adhesions to an adjacent stump of omentum (Nieberding). The passive reactive pressure of the coils of intestine and of the abdominal walls tends also to further confine the enormously dilated and twisted loop of bowel to its abdominal state. Knots may be formed by the association of loops of the ileum with each other or about the pedicle of a twisted cecum.

Here, again, males between forty and sixty years of age have been observed to be especially the subjects of volvulus.

Chronic.—(*a*) *Fecal Impaction.*—*Intestinal Concretions.*—Accumulation of feces (*coprostasis*) is a common cause of intestinal obstruction, the impaction taking place usually in the cecum or sigmoid flexure.

Though not infrequent in children, fecal obstruction is more common in adults (particularly in females), in the hysteric, the demented, and the hypochondriac. Congenital dilatation of the colon may predispose to coprostasis, and an acquired dilatation, which in some cases becomes enormous, is often the result of paresis of a portion of bowel caused by over-distention for a long period of time. The retained fecal masses may become hard, but for some time permit the passage of soft or liquid material through the interstices of the accumulation, until finally either complete obstruction takes place or the condition is relieved. So severe may the obstruction prove in some cases as to result in inflammation, ulceration, and even perforation of the bowel.

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[1] *Med. Record*, Aug. 13, 1892.

Among other causes of obstruction due to abnormal contents may be mentioned *enteroliths*. These are intestinal concretions formed of various nuclei, as gall-stones, hardened feces, phosphates of lime and magnesia, various foreign substances, and organic derivatives. Balls of tangled ascarides may mass sufficiently to cause obstruction.

Gall-stones not infrequently become impacted in the duodeno-jejunal or ileo-cecal regions after ulceration through the duct, except in the case of very small stones, which enlarge subsequently by accretion.

Foreign bodies, as pins, buttons, coins, fruit-stones, may also cause obstruction of the bowel. It is stated that even insoluble mineral medicines, as bismuth or magnesia, have caused obstruction by accumulation in the intestines.

(*b*) *Tumors.*—Tumors cause a form of chronic obstruction that may at any time develop suddenly into the acute type. They may do so either as—(1) *new growths* in the wall of the intestine itself, or by (2) *compression and traction from without.* Again, the intestinal neoplasms may be *malignant* or *benign* in nature. *Carcinoma* of the bowel is at once the most frequent and important of these. It may be either circumscribed and annular, causing a gradual narrowing of the bowel-lumen, or a diffused infiltration of the intestinal wall, commencing either in the mucosa or in its glands (cylindric epithelioma). Its most common seat of growth is the large bowel, about the sigmoid flexure. The mesenteric and retroperitoneal glands are usually secondarily affected. Ulceration of the bowel and catarrhal inflammation of the mucous membrane above the carcinoma may coexist late in life.

Sarcoma usually attacks the small bowel, starting beneath the mucosa, and is of the recurrent variety. Regional infection of the mesenteric and retroperitoneal glands (*Löbstein's cancer*) is also a usual consequence of sarcoma. It may occur in children or in young adults.

Benign tumors may be polypoid, adenomatous, fibromatous, and lipomatous. Intestinal obstruction due to compression or traction may be caused by tumors (omental) or by adhesions of the pelvic viscera.

(*c*) *Cicatricial strictures* cause chronic intestinal obstruction, as after the healing of various ulcers, the cicatrices of which slowly contract. Cicatricial stenosis of the colon is commonly due to the cicatrization of dysenteric ulcers. In the rectum the stenosis is usually a result of a syphilitic lesion. Tuberculous and, very rarely, typhoid ulceration may be followed by stricture of the small intestine.

(*d*) *Congenital stricture* is rare, and is more purely surgical than the preceding cases. It is often an occlusion or an imperforate condition of the anus (*atresia ani*), and is only mentionable in this connection.

(*e*) *Paresis of Peristalsis.*—This condition—called also *adynamic obstruction*—while it is a functional affection, is held to be either a circumscribed or diffuse paresis of the intestinal muscular coat. It is caused by some such inflammatory disturbance as enteritis or peritonitis, or even by the manipulations employed in prolonged abdominal sections. In such cases the obstruction is due to an accumulation of feces and gases in the paretic portion of the bowel, causing marked tympanites, vomiting, and constipation.

Special Pathology.—The pathologic changes that accompany nearly every form of intestinal obstruction are briefly stated as follows: Accumulative dilatation—with hypertrophy in chronic cases—of the intestine above the seat of disorder, and an emptiness, narrowing, and even atrophy below the obstruction. The affected walls of the bowel are inflamed, and there is a surrounding acute or chronic peritonitis. Catarrhal and sometimes diphtheritic inflammation of the mucosa may develop. Gangrene, ulceration, and perforation of the bowel, with resulting generalized peritonitis, may also ensue.

Symptoms.—**Acute Obstruction.**—There is a suddenly developed *abdominal pain* that may follow some abrupt or severe exertion. *Early vomiting* and *absolute constipation* are also conspicuous and important symptoms. If the obstruction is high in the small bowel, distressing hiccough and eructations may precede the vomiting. Except for the possible discharge of the intestinal contents below the seat of obstruction, the constipation is usually complete and obstinate. Accompanying the latter condition there is tympanites, which is most marked in obstruction of the colon. Intermittent and colicky at first (partial obstruction—Treves), the *pain* soon becomes agonizing and constant. *Vomiting*, also, alternating with *painful retching*, is more constant and severe after several hours. The *material* at first ejected is gastric and mucous; it then becomes bilious, and finally is characteristically stercoraceous, due, most probably, to the putrid decomposition of stagnated contents above the obstruction.

The *constitutional symptoms* develop early, are intensely threatening to life, and cause rapid and profound depression and collapse. The pinched and pallid features, cool and moist skin, Hippocratic expression, rapid and feeble pulse, the usually subnormal temperature, shallow and accelerated breathing, marked thirst, scanty urine, great anxiety and prostration,—all indicate the gravity and danger of the condition.

The *physical examination* will discover a swollen, extremely tender, and tympanitic belly. Exaggerated peristalsis of the intestine above the obstruction may be visible on the surface of the abdomen. Borborygmi, gurgling, and splashing may be heard on auscultation.

Chronic Obstruction.—The symptoms are more dependent upon the special causes operating than in acute obstruction. The fact that early in the case only partial obliteration of the intestinal lumen may be rightly inferred in many of the chronic forms of obstruction has given rise to the discriminating term of *intestinal constriction*. In general, the clinical history is one of *increasing* and *intractable constipation*, sometimes alternating with diarrhea, due to catarrhal inflammation of the mucosa above the obstruction. Paroxysms of colicky *pain* and, later, augmenting *tympanites*, *vomiting*, and *prostration*, attend. These symptoms may merge suddenly into those of the acute form of obstruction. The bowel-movements in chronic obstruction are irregular, infrequent, slight, and sometimes accompanied by pain and tenesmus. The *stools* consist often of small, hard, ribbon-like, or scybalous masses, and may contain blood and mucus. When the stenosis is in the small intestine the constipation is less apt to occur on account of the fluidity of the contents. Sometimes, and particularly in old people, the rectum becomes distended with hardened accumulations of feces; there is in such cases a constant feeling of fulness and a harassing desire to defecate, but the attempts thereat are ineffectual. The pain of fecal impaction may be due either to colitis or to peritonitis, and may be referred to the regions of the cecum or sigmoid flexure.

In *malignant* and in *cicatricial stenosis* there are a prolonged and variable history of constipation, occasional vomiting, localized pain, meteorism, and, in cancerous cases, the development of the characteristic cachexia and the progressive emaciation.

Physical Examination.—*Inspection* shows the abdomen to be distended from meteorism, the movements, and contour even, of the coils of intestine in active peristalsis above the seat of stricture being evident. A tumor or the throbbing aorta (excited, perhaps, by pressure of the distended bowel or growth) may be *palpated*. Tympany and borborygmous noises may also be noted.

Diagnosis.—**Locality of the Obstruction.**—Given the symptoms of a sudden, severe, and exacerbating pain in the abdomen; of marked, and later feculent, vomiting; of absolute constipation and of tympanites and profound, early, systemic depression,—a diagnosis of acute intestinal obstruction may be easily made. The determination of the seat of trouble, however, is often very difficult. First may be mentioned the differential diagnosis between obstruction occurring in the small and in the large intestine. It may be noted of the former that vomiting occurs early, is scanty, and later feculent, while in the latter there is less vomiting and the vomitus is seldom feculent. Again, in obstruction of the small gut the distention is both less marked and higher situated, while in that of the large gut tympanites is often quite marked, is more central, is associated with tenesmus, and sometimes with mucus and blood. If the cause of obstruction be a tumor or stricture, the locality may be successfully palpated or the lower limit of the active coils of hypertrophied intestine may be defined.

In stenosis of the duodenum or jejunum, owing to the stagnation and decomposition of albuminous substances, the products of which (indol and phenol) are absorbed and partly excreted by the urine, use may be made of the discovery of increased amounts of indican in the urine for diagnostic purposes. On the other hand, in stenosis of the large intestine the urinary test may be negative, since the albuminous elements of the intestinal contents are absorbed before they reach the stenosed portion of bowel, where stagnation and putrefaction can take place.

Examination *per rectum* with the finger or with the rectal tube, by means of liquid distention or gaseous inflation of the colon, may enable us to determine the seat of obstruction in certain cases. The detection of a deeply-seated incarcerated hernia (in the abdominal fossæ and pouches, diaphragm, or obturator foramen) is often made only *postmortem*.

Nature of the Obstruction.—This is even more difficult of discovery than the preceding. The following causes of obstruction with their differentiation may be referred to in attempting a diagnosis: *Strangulation* often affords a previous history of peritonitis or abdominal section or of recurrent attacks of abdominal pain, occurring mostly in young adults. Early fecaloid vomiting is common.

Intussusception usually gives a negative previous history. The suddenness of the attack, without appreciable cause, occurring in a child, and associated with colicky pain, tenesmus, and the presence of mucus and bloody stools, and of an elongated cylindric tumor in the right iliac or umbilical regions, however, render this condition easy of diagnosis in some instances. It is to be noted that absolute constipation and meteorism here are unusual. The intussusception may be felt in the rectum.

In *volvulus* it may be helpful to elicit a history of former constipation and flatulence, with evidences of atony of the bowel, in persons of advanced years, along with marked abdominal tympany, tenderness over a distended coil, which may perhaps be outlined (Wahl), a rigid abdomen, and sometimes dyspnea from great gaseous distention.

The history in cases of fecal obstruction is nearly always one of obstinate, habitual constipation, and occurs especially in females and neurotic subjects. The onset is gradual; pain is less acute; and tympany and fecal vomiting are less prominent and late in appearance. Fecal masses in the colon and rectum may be palpated, and even indented, particularly in the cecal and sigmoid flexures. Dulness is present on percussion, with slight tenderness over the tumor.

Obstruction due to *large enteroliths* or *foreign bodies* may be only surmised; especially is this true when symptoms of appendicitis arise.

Biliary calculi may give a history of previous attacks of hepatic colic and jaundice.

In the chronic obstructive form of *stricture* of the bowel due to cicatrices or neoplasmata the history of dysentery, tuberculosis, sarcoma, or carcinoma should be considered. The detection of an irregular tumor and the cancerous cachexia point to malignancy.

In obstruction caused by *intestinal paresis* there is generally a history of a previous enteritis, peritonitis, or celiotomy. The abdomen is smooth, though tympanitic throughout, and there is no perceptible peristalsis.

Not rarely it will be of therapeutic as well as of diagnostic importance to ascertain whether an attack of acute obstruction is primary, or whether it is the terminal exacerbation of a chronic condition, such as carcinoma of the bowel. Here a study of the past history of the patient, as well of the present signs of a probable nature, will afford considerable aid.

Differential Diagnosis.—Acute intestinal obstruction must be discriminated from *acute generalized peritonitis*.

ACUTE GENERALIZED PERITONITIS.	ACUTE INTESTINAL OBSTRUCTION.
Etiology.	
There is a history of causal conditions or diseases (ulcer, appendicitis, pelvic infection).	There is a history of previous chronic obstruction or hernia. (The age of the patient if it be intussusception.)
Symptoms.	
An early and considerable rise of temperature; later variable or may be absent.	No early rise (except in volvulus), but later with advent of peritonitis.
Pain more continuous and diffuse.	Pain in short paroxysms and localized.
Vomiting is characteristic, but not stercoraceous.	Vomiting becomes characteristically stercoraceous.
Collapse occurs later.	Earlier onset of collapse.
Slight increase of indican in the urine.	Excessive indicanuria, particularly when the small intestine is obstructed.

Physical Signs.

Distention of the abdomen is usually general and marked.	Less marked (sometimes partial), unless the obstruction be situated in the lower segment.
Visible peristaltic waves absent.	Present and pronounced when the seat of obstruction is low.
Tenderness general.	Tenderness localized.
Signs of effusion appear.	Less common, due to secondary peritonitis.
Auscultation negative.	Loud gurgling and splashing sounds audible on auscultation.
Prognosis almost hopeless.	Not so if operated upon early.

It must also be differentiated from *acute enteritis*, in which (particularly when due to toxic minerals) there is more apt to be diarrhea with considerable mucus and blood, an elevated temperature, intense gastric pain, associated with traces of the poison in the vomitus, as well as with its effects on the oral mucous membrane, and an absence of marked tympanites and fecal vomiting. There are also localized pain, tenderness, and tumor, or there may be collapse.

The various forms of *abdominal colic*, as enteralgia, hepatalgia, and nephralgia, should not be mistaken for acute intestinal obstruction after considering the history of the cases, the character and locality of the pain, and the absence of such symptoms as obstinate constipation, fecal vomiting, early collapse, intense local pain and tenderness.

Course, Complications, and Prognosis.—A case of acute obstruction usually terminates within from two to seven days. The chronic form may last weeks, and even months, with progressive emaciation and anemia, until the superaddition of more or less acute symptoms, lasting from ten to fourteen days. As a rule, the *prognosis* is wholly unfavorable, and especially in the acute cases. The chronic forms, due to fecal or other impaction, often recover with the discharge of the disturbing intestinal contents. Life may be prolonged by surgical interference in certain cases if they are taken in their inception.

Complications that may occur, as secondary peritonitis, gangrene, perforation, septico-pyemia, and enteritis, are all grave, and only tend to hasten the dissolution.

Treatment.—Whilst the treatment of intestinal obstruction is sooner or later essentially surgical, attention to the medical aspect is frequently of prime importance. The first indications for therapeutic interference in acute obstruction are presented by the *pain* and the *incessant vomiting*. The former is to be met by hypodermic injections of morphin, which at the same time tend to arrest the excessive peristalsis. For the vomiting no other measures are comparable to gastric lavage and starvation. It is well in most cases to withhold food for some hours to prevent retching and aggravation of the condition. The lavage is strongly advised by Kussmaul, who claims that both the tension above the seat of stricture and the inordinate peristalsis are thus greatly diminished and, exceptionally, cured. It may be repeated every six hours. A diagnosis of intestinal obstruction having been made without having learned the cause or character of the obstruction, cathartics should absolutely not be given. If it has been determined that fecal impaction is the trouble, it is still prudent to avoid purgatives until the main mass has been moved, as in many cases there are both paresis and inflammation at the seat of impaction, so that this class of agents would thus be useless, if not harmful. High rectal injections, copious, steady, and regularly repeated, are to be practised, using for this purpose preferably "a warm saline solution of olive oil" (particularly if scybala be present) administered while the patient is in an inverted position by means of a fountain syringe, so that the flow is readily controllable. The abdomen should be methodically kneaded (a valuable adjunct in the procedure) and the patient at times well shaken. This method of treatment, by hydrostatic pressure, can and must be carried forward without undue violence, and if it be unsuccessful, the intestines are to be inflated from a large india-rubber bag with air or hydrogen gas (Senn), of which two to three gallons may be cautiously introduced. Thorough manipulation of the abdomen from below upward, particularly if it be a case of intussusception, may be combined. In the latter condition inflation, early and perseveringly applied, cures the majority of instances. In cases of intussusception or strangulation of the bowels these efforts should be continued for twenty-four hours, when, if the condition is not relieved, immediate operation is to be encouraged and advised. Although the statistics of Fitz show the mortality in cases without operation to be lower (69 per cent.) than with operation (83 per cent.), I am convinced from personal observation that the less favorable results from abdominal section would not obtain if it were performed in due time. To relieve the excruciating tympanites the plunging of a fine trocar and cannula into the intensely distended bowel, as in case of volvulus, may be required.

In chronic obstruction the treatment of the underlying or etiologic conditions and various complications is to be conducted on general principles. Additionally, the patient's dietary is to be arranged with care, and the bowels moved with unfailing regularity, by the use of unirritating laxatives and enemata. During the periods of threatening complete occlusion, with pain, the methods advocated above for acute obstruction are appropriate. If total obstruction persist despite medical treatment, surgical treatment—enterectomy, enterotomy, or other operation, as the circumstances of individual cases may dictate—is required.

The after-treatment consists in keeping the bowels active and regular by habit, diet, and an aperient pill if needed. Massage and electricity to the abdomen are found useful at this time.

CARCINOMA OF THE INTESTINE.

(*Carcinoma Intestinalis.*)

Carcinoma of the intestine is perhaps the commonest cause of chronic intestinal obstruction. The stenosis is usually partial, and is due both to compression and to direct invasion of the lumen of the bowel by the growth. Primary intestinal carcinoma is rare in comparison with the occurrence of gastric carcinoma.

Pathology.—When carcinoma attacks the intestine it is usually in the form of a cylindric-celled epithelioma, although it may assume the various forms as found in carcinoma of the stomach—namely, scirrhous, medullary, and colloid. The growth may be annular or semipolypoid, or it may occur as a diffuse nodular infiltration of the bowel-walls. Ulceration of the surface of the carcinoma may take place, and the glandular structures of the abdominal cavity sometimes reveal metastatic growths. The most frequent seat of intestinal carcinoma is the rectum, and next in order of frequency are the sigmoid flexure, the transverse and descending colon, the *papilla duodenalis*, the ascending colon, and the lower and middle portions of the ileum. The bowel is dilated above the constriction, and is usually filled with an accumulation of fecal matter. The muscular coat is hypertrophied. Below the narrowing the intestine may be atrophied. Rectal adenoma may develop into carcinoma (adenosarcoma).

Etiology.—Heredity and advanced age are of chief importance as predisposing causes. Whether or not antecedent intestinal ulceration may afford a probable nidus for carcinomatous growths is doubtful.

Symptoms.—A description of the course of *rectal carcinoma* belongs more properly to surgical works. The chief symptoms are *progressively increasing distress* and *radiating pain* in the *rectum*; these occur, at first, during defecation only, but later almost constantly. There may be diarrhea alternating with constipation, and the feces often contain blood and mucus. Gradual *bodily wasting* and increasing mental anxiety are associated. Paralysis of the anal sphincter and consequent incontinence may ensue.

The symptoms of carcinoma of the bowel *above the rectum* are often vague, and vary according to the portion involved by the neoplasm. With or without an appreciable tumor in the abdomen the clinical history is usually that of *chronic obstipation* of the intestines. There are irregular attacks of *sharp, colicky pains*, especially a few hours after eating, distressing defecation, obstinate constipation, perhaps alternating with diarrhea, sometimes vomiting, which may be feculent, and not rarely slight meteorism. The special symptoms of carcinoma of the *papilla of Vater* are vomiting, jaundice, and colic. The progressive emaciation and debility of the patient are marked. In advanced cases of stenosis the *feces* are passed in small, compressed lumps resembling sheep's dung.

Physical Examination.—*Inspection* of the abdomen may show the presence of a tumor along the line of the sigmoid flexure or colon; peristalsis may be seen above the site of the carcinoma, communicating its movements to the abdominal walls. *Palpation* may be resorted to in order to confirm the above, and the growth is then frequently found to be nodulated. A sign which is practically diagnostic of stenosis is a sudden appearance of small coils of bowel which vanish very quickly and reappear again (Boas). *Percussion* may give either dulness or a muffled tympany over the tumor and for some distance above, on account of accumulated masses of feces. This area of impaired intestinal tympany may be sharply defined.

Diagnosis.—This may rest, in some cases, upon heredity, the age, the evidences of the cancerous cachexia, sharp, radiating abdominal pains, bloody stools, and the detection of a firm and nodular tumor.

Differential Diagnosis.—(*a*) Carcinoma of the bowel above the rectum needs to be discriminated from other abdominal tumors. The presence of the following may render the diagnosis of carcinoma during life well-nigh impossible: *sarcomata, fibromata, myomata, adenomata*, and *cys-*

tomata, all of which may produce symptoms of obstruction like those due to carcinomatous growths. The cancerous cachexia may be simulated by other conditions. The advanced age of the patient and the distressingly rapid and downward progress of the disease will, however, point toward malignancy. *Fecal tumors*, *enteroliths*, *foreign bodies*, and *peritonitic adhesions* may need to be excluded also. It is to be recollected that fecal masses may exist above and overshadow the presence of carcinoma of the intestine. When peritonitic adhesions are present, inquiry may elicit the previous existence of peritonitis (Eshner).

(*b*) *The portion of the bowel involved* by the neoplastic growth is also difficult of definite diagnosis, except when it occurs in the rectum, when the digital and visual examination of the parts, supplemented, it may be, by microscopy, are sufficient. The locality of the tumor as detected by palpation, associated with special symptoms, is of value in arriving at a diagnosis of the diseased portion of bowel. Heulin[1] has studied carefully primary cancer of the duodenum, and asserts that the comparative frequency of duodenal involvement is due to limited motion of the organ, being thus subject to injury. When it occurs above the papilla of Vater the symptoms greatly resemble those of *dilatation of the stomach*. An important point separating carcinoma above from that below the papilla is the presence or absence of bile in the vomit, being absent if situated above. When the carcinoma involves the papilla of Vater symptoms of biliary obstruction necessarily follow. A hard nodular mass may sometimes be felt in the lower epigastric region; this coupled with increasing gastric dilatation and marked persistent jaundice would indicate carcinoma of the duodenum. It is apparent, however, that *carcinoma of the pylorus*, of the left lobe of the liver, or of the omentum or mesenteric glands, or a thickened cecum might all be easily confounded with carcinoma of the bowel at various adjacent parts of its course. The injection of fluid into the bowel may be resorted to in locating the probable situation of the growth. Thus, if obstruction from carcinoma exists in the sigmoid flexure, liquid will be arrested there and the rectum distended; while, if the stenosis be high up in the large or small intestine, the colon will be found comparatively emptied of feces and will be distended with the injected liquid.

Course and Complications.—Carcinoma of the intestine sometimes runs a rapid course, and may last but a few months or even weeks; in the scirrhous variety, however, the disease may last two or three years.

Intestinal carcinoma may perforate the bowel and cause fatal purulent peritonitis, and carcinoma of the rectum may perforate and invade the vagina and bladder, causing purulent vaginitis and cystitis. Or, owing to extreme distention by fecal accumulation between a cancerous stricture of the sigmoid flexure, for instance, and the resistant ileo-cecal valve, rupture of the colon, followed by a terminal peritonitis, may result. Extension of the growth into surrounding tissues, with ulceration, may lead to cellulitis, phlebitis, and pyemia.

The **prognosis** is almost hopeless.

Treatment.—This, from a strictly medical standpoint, is simply palliative. The diet should be highly nourishing and easily assimilable, but when the symptoms of acute obstruction supervene the administration of food by the mouth is contraindicated. Attention to the state of the bowels by the use of enemata, or of the aloin, strychnin, and belladonna pill is necessary in most cases. Opium or cannabis indica for the pain, and stimulants for the depression, may also be serviceable.

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[1] *Gaz. hebdom. de Méd. et de Chir.*, Feb. 13, 1898; *Thèse de Paris*, 1897; Saunders' *Year-Book*, 1899, p. 194.

Lavage of the stomach gives decided relief when decomposing matters tend to cause regurgitation on account of the damming back of accumulated food-detritus.

Carcinoma of the bowel may be treated surgically by colotomy, excision, lateral anastomosis of the bowel, enterostomy, and, if the growth be situated in the rectum, by extirpation by means of sacral resection (*Kraske's operation*).

HABITUAL CONSTIPATION.

(*Costiveness.*)

Definition.—Chronic fecal retention, habitual infrequency, irregularity, difficulty, or insufficiency of the evacuations of the bowels.

Although constipation is a symptom, and although habitual constipation is frequently a symptom of chronic disease, the causal elements of the latter may be so indefinite and obscure that the former takes on all the individual importance of a functional affection. I describe habitual constipation, therefore, as a disease *sui generis* ("idiopathic").

Etiology.—In the majority of cases habitual constipation is the direct effect of a lack of expulsive or peristaltic power, and also of a deficiency of the hepatic and intestinal secretions. Two sets of causes operate to bring about these conditions of abnormal defecation:

General Causes.—(*a*) *Temperament:* it has been observed often that people of a nervous and "bilious" or motive temperament, of the dark type, are much troubled with constipation. Anemic brunets—persons having pale skin and dark hair combined—are particularly so affected, although alternating periods of diarrhea may supervene, owing to the hydremic state of the blood. "Torpid liver" and "sluggish bowels" are commonly held to be synonymous with these physical characteristics. (*b*) *Habit:* a sedentary life conduces to secretive inactivity. Thus, a lazy life, in which the calls of nature are irregularly attended to or habitually neglected, leads to frequent over-distention of the rectum and paresis, a common cause of chronic constipation. Again, the feminine false modesty (so called) that prompts a postponement and suppression of the desire to defecate in public places, as well as the habitual, hurried performance of the act in illy-kept, uncomfortable, and unsanitary closets,—all these tend to obtund the sensibility of the bowel to fecal masses in the rectum. The accumulation of these fecal masses causes paralytic over-distention, their hardening into scybala, and difficulty of expulsion. (*c*) *General bodily weakness, and diseases,* as neurasthenia, hysteria, anemic brain- and spinal-cord affections (causing inhibitory disturbances of the intestinal nerve-supply), acute fevers, hepatic disorders, especially the presence of jaundice, and the habitual dependence upon and use of purgatives. (*d*) *Diet:* the constant use of concentrated articles of food, as meats, in which little residual matter is left to stimulate the bowel to peristalsis. On the other hand, a very coarse diet may leave such an excess of residue as to cause fecal impaction. (*e*) A change of drinking-water, or water from chalky regions. (*f*) *Abundant and prolonged diuresis and diaphoresis*, by causing loss of fluids, also may induce chronic constipation.

Local Causes.—(*a*) *Atony of the abdominal muscles* from obesity, or, in females, as a result of many pregnancies. (*b*) *Atony of the large bowel* (the sigmoid flexure in particular) from chronic colitis. (*c*) *Pressure by tumors.* (*d*) The presence of *intestinal stenosis* from external or internal constriction. (*e*) *Congenital stricture* or *giant growth* of the colon, with coprostasis (as in Formad's case). (*f*) Tonic contraction of the muscular coat, as in basilar meningitis and lead-poisoning.

Symptoms.—In cases in which there is no adequate cause for habitual constipation other than a constitutional and perhaps an inherent peculiarity there may be the appearance of perfect health. Nothing is complained of save the fact that an evacuation of the bowels occurs too infrequently. It should be borne in mind here, however, that the term "constipation" is, individually speaking, almost wholly a relative one—*i. e.* one person may enjoy good health with but one evacuation every other day, another with two passages per diem, while still another must have one stool a day, *cæteris paribus*, to feel perfectly well. The last is usually considered an average normal state with most people. Persons such as are instanced above, in apparently good health, but observing that they have to defecate less often than many others, sometimes grow anxious, worried, and even hypochondriac, until assured that they are not truly constipated if enjoying perfect physical ease.

Symptoms of habitual constipation may be direct or reflex. *Direct* or *local* troubles are seen in the feeling of fulness, weight, and pressure in the perineum and abdomen. Flatulence, colicky pains, and alternating diarrhea occur not infrequently. The hurried and inattentive performance of defecation gives rise to the so-called "cumulative constipation," in which the accumulated feces are but partially evacuated with the movement, and the rectum consequently is not emptied. A sense of fulness then remains, and complete relief is not felt in these cases.

Reflex and *general* symptoms are malaise, languor, hebetude, irritability of temper, headache, facial flushing, palpitation, cold extremities, anorexia, vertiginous attacks, paresthesia, menstrual distress in women, sleeplessness, and bad dreams. Pressure on the sacral and visceral nerves may cause neuralgias. The tongue is coated. Palpation of the abdomen often shows the presence of doughy-like fecal tumors at the cecum or at the hepatic, splenic, and sigmoid flexures, or of bologna-like masses at intervening places. In marked cases attacks of nausea and vomiting, with diarrhea, may ensue; fever may also be present, and typhoid fever even may be simulated (Meigs).

Complications and Sequelæ.—Hemorrhoids, ulcerative colitis, perforation, and enteritis may be associated with chronic constipation. Not rarely do we have as results dilatation of the colon or sacculation, with the presence, in old people mainly, of *enteroliths* (calcified scybala); also intestinal obstruction and typhlitis, or cerebral hemorrhage or hernia from violent straining efforts.

Diagnosis.—Bearing in mind the relativity of constipation in different individuals, the diagnosis is read at sight. The detection of the causes is not difficult, though sometimes tedious. Hypochondriasis or melancholia should be carefully placed either as precedent to or consequent upon chronic constipation, the nervous condition often acting to produce the latter, and *vice versâ*.

The **prognosis** is usually favorable, but should be guarded.

Treatment.—**Hygienic.**—Causative factors must, of course, be removed, modified, or lessened. Systematic regularity as to time and frequency and sufficiency of movements of the bowels should be enjoined upon and practised by the patient. Exercise is of signal value, and particularly horseback riding or gymnastic motions that bring the abdominal muscles into play. Attention to the calls of nature should be esteemed a duty, and proper time and heed must always be given to the completeness of defecation. Young girls especially should be instructed in this regard. The wholesale swallowing of cathartics is to be vigorously combated. The dietetic regimen, if properly looked after, often avails much in relieving this affection, and foods calculated to be easily digestible, but leaving a moderate residue after digestion, are to be recommended. Such are bread made of unbolted flour, plenty of vegetables and fruits, butter, and such laxative articles as figs or honey. A glass of cold water taken regularly at bed-time and in the morning before breakfast is efficacious and a point of common knowledge.

Remedial.—The methods and means offered for the cure of chronic constipation number legion. From the little aperient pill or "peristaltic persuader" to the cannon-ball rolled externally along the course of the large bowel is made up such a list of drugs and measures as to leave untenable any plea of lack of resource that may be advanced. *Drugs occupy a subordinate part in the treatment of habitual constipation.* Indeed, their use should be restricted mainly to those periods when the bowels become unusually obstinate and when a more or less free movement is urgently needed. That the constant use of laxative and purgative drugs tends to a confirmation of the condition, and its ultimate resistance to the action of cathartics when circumstances will have required their use, is familiarly known.

I have found of value in lithemic and dyspeptic subjects the laxative bitter waters (Hunyadi Janos, Kissingen, Friedrichshall, Carlsbad).

Drugs employed to unload a filled bowel may at times be used sparingly and in the smallest adequate quantities; the mildest forms should be selected. Since the constipation is only temporarily relieved by catharsis, the frequent use of strong purgatives in large doses only tends to render the bowel accustomed to their use. Creosote in large doses has recently been highly recommended; it probably relieves the constipation by overcoming the intoxication.

Among those laxatives and cathartics most commonly used may be mentioned aloes, rhubarb, Rochelle and Epsom salts, compound licorice powder, castor oil, jalap, senna, mercury, colocynth, and podophyllin. Important adjuncts in combination with one or more of the above are the extract of nux vomica (or strychnin) and the extracts of belladonna, hyoscyamus, and physostigma. The much-used aloes, strychnin, and belladonna pill can be used for a considerable length of time in the hope of stimulating a normal intestinal and sphincteric activity, and thus inducing even a cure in some cases. The formula may be made up as follows:

℞. Aloin.,	gr. iij–v (0.194–0.324);
Strychninæ sulphat.,	gr. $\frac{1}{3}$–$\frac{1}{2}$ (0.0216–0.0324);
Extr. belladonnæ,	gr. ij–ijss (0.129–0162).

M. et div. in pil. No. xx.
Sig. One pill at bedtime.

Sulphur in confection, along with the official pill of aloes and iron, has been recommended for the habitual constipation of anemia. In senile atony of the bowel, with much flatulence, a laxative pill having in combination asafetida or capsicum is often beneficial.

The subjoined formulæ are also rationally and empirically serviceable in chronic constipation:

℞. Ext. cascar. sagrad.,	ʒss (2.0);
Ext. nucis vomicæ,	gr. iv (0.259);
Ext. physostigmat.,	gr. iij (0.194);
Ext. belladonnæ,	gr. ij (0.129).

M. et ft. in pil. No. xx.
Sig. One at night, or night and morning.

(Aloes, gr. j (0.0648), or podophyllin, gr. ij–iij (0.129–0.194), or ext. colocynth. comp., gr. ij–iij (0.129–0.144), may be substituted for cascara in the foregoing formula.)

The **mechanical** means of relieving habitual constipation, as by *enemata*, are injurious if long continued, by reason of their irritating effect on the rectal and colonic mucous membrane, as well as on account of their tendency to become incompetent. At times, when the stomach is weak or irritable, a loaded bowel may be relieved by an ordinary enema of soap and water or by one containing $\frac{1}{2}$ to 1 ounce (16.0–32.0) of castor oil, with 1 or 2 drams (4.0–8.0) of oil of turpentine if there be some flatulence. Glycerin enemata, containing from $\frac{1}{2}$ to 2 ounces (16.0–64.0) of the agent, may be used. *Suppositories* of soap, molasses candy, or glycerin are included in the armamentarium. *Massage* also claims an important part in the relief of habitual constipation. It acts by stimulating the peristalsis and the abdominal muscles, and should be employed at set times in the day preceding a desired evacuation of the bowels. The hand of the *masseur*, or that of the trained patient even, when systematically used in this way, may be effectual when all other means have failed. The regular rolling of a metal ball along the course of the greater gut may be mentioned for its novelty as well as for its undoubted efficacy. The application of the faradic current to the abdominal walls or galvanization of the lumbo-abdominal circuit deserves proper trial in many cases. Hydro-therapeutic measures, or cold sponging and baths, are nearly always useful adjuncts in the treatment of this often stubborn affection.

DILATATION OF THE COLON.

(*Ectasia of the Colon.*)

This is usually a chronic condition, though not rarely it is acute. It may also be general, but in the majority of cases it is confined to the colon, and particularly to the sigmoid flexure. The *postmortem* findings are those of hypertrophic dilatation of the bowel, and rarely ulcerative and catarrhal lesions of the intestinal mucosa are noted. The sigmoid flexure is prone to become dilated in subjects in whom it is congenitally elongated. Mya[1] believes that the condition is due to a faulty development, and not to fetal disease. Atony of the muscular coat is a leading causative element. The most distinctive features are *constipation*, which generally dates from infancy, and great *abdominal distention*. In the case reported by Rolleston and Hayward peristaltic waves were visible upon the surface. Cases of this kind have been mistaken for acute intestinal obstruction. The condition may fluctuate, constipation alternating with regular daily movements, and the distention changing to a normal softness of the abdominal parietes in some instances. I have recently seen a case of this kind in a male aged twenty-seven, in whom the affection had commenced in infancy. In the *treatment* of the constipation resulting from congenital ectasia of the colon, lavage of the intestine with a very long tube is superior to laxatives or purgatives. The principal aim of treatment, when possible, is to overcome or check the causative conditions. A *diet* calculated to prevent or relieve constipation is indicated (*vide* p. 844). Vegetable foods leaving a gross residue should be restricted or even prohibited. Massage, galvanism, and hydrotherapy are all capable of beneficial effects in suitable cases. Strychnin is a valuable remedy, and in cases attended with constriction, operation should be recommended or at least considered.

NEUROSES OF THE INTESTINE.

As in the case of the stomach, these embrace derangements of (*a*) secretion, (*b*) sensation, and (*c*) motion.

(*a*) SECRETORY DISTURBANCES.

Unquestionably the intestinal secretion may, through a purely nervous influence, be augmented. This manifests itself most frequently in the primary morbid secretion of mucus (*mucous colic*) and in membranous enteritis. Moreover, the fact that an actual catarrh of the intestinal mucosa may supervene as a secondary event is undeniable.

MEMBRANOUS ENTERITIS.

(*Enteritis Membranacea.*)

Definition.—A peculiar pathologic condition, chiefly of the large intestine, attended by a morbid secretion of mucus.

Pathology.—In the truly primary form there are no morbid lesions discoverable in the mucosa. Osler states that the membrane is due to a derangement of the functions of the mucous glands the nature of which is unknown. My own view is that membranous colitis is a secretory neurosis, and that the catarrhal process may develop as a secondary event.

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[1] *Lo Sperimentale*, 1894, fasc. iii. p. 215.

Etiology.—Sex has a decided influence, not less than 80 per cent. of all cases (W. A. Edwards) being noted in women. Hysteric and neurotic females are the most frequent victims of the disease, which is rare in children. W. H. Thomson[1] emphasizes direct mechanical irritation of the rectum (horseback-riding, bicycle-riding, uterine fibroids, hardened scybala).

Symptoms.—I have found the condition to be invariably associated with a decidedly constipated habit—a fact that may, in part, explain its occurrence, since time is thus allowed for the formation of the membrane. The most important clinical feature is the *passage*, at *varying intervals*, of *long, ribbon-like threads of mucus*, or of more or less *perfect casts of the gut*, the act being attended with *tenesmus* and severe *colicky pains*. The composition of the stools has been thoroughly investigated by M. Rothmann and O. Rothmann and C. Ruge. They "consist of a uniformly turbid ground-substance, which, on the addition of acetic acid, becomes opaque and striped. It is interspersed with a cellular detritus, consisting partly of strongly refractile granules and partly of cellular elements, desquamated epithelial cells, round cells, and peculiar glossy flakes. There are also found cholesterin-crystals, needles of fatty acids, triple phosphates, remnants of undigested food, pigment-granules, many bacteria, and occasional red and white corpuscles."

The individual paroxysms vary in *duration* from one to ten days or more. In one case observed by me the attacks lasted about two days, recurring regularly at the end of every three months. Ordinarily the recurrence is after a shorter interval.

Diagnosis.—It is important to make a microscopic examination of the pieces of membrane. If, when thus examined, mucus, cylindric-celled epithelium, a few round cells, and the other elements already mentioned are found present, the diagnosis of mucous enteritis is undoubted. It is to be recollected, however, that membranes are not passed with every attack.

Course and Prognosis.—The disease pursues a very chronic course and lasts for many years. The bodily nutrition suffers considerably if the attacks are frequent and severe, though, as a rule, this does not occur until a late stage in the affection. The risk to life is slight.

(*b*) SENSORY DISTURBANCES.

It may be noted here that the sensory nerves of the intestines, as well as the inhibitory and vaso-motor dilators, are traceable to the splanchnics. Increased sensibility of the sensory nerves produces—

ENTERALGIA.

(*Neuralgia of the Intestine.*)

Etiology.—This is commonly met with in hysteric, neurasthenic, and anemic subjects. It occurs as a reflex neurosis, as in the case of cold, gout, and irritative lesions of the pelvic organs (kidneys, liver). Enteralgia is symptomatic of many local affections and conditions that induce direct irritation of the sensory nerve-filaments of the intestine; among these are inflammation of the mucosa, foreign bodies, gall-stones, abnormal distention with gas, and enteroliths. Under these circumstances the condition is associated with increased activity of the motor nerves or heightened contraction of the muscularis, forming true intestinal colic. In lead colic it is probable that the lead acts directly upon the nerves or their ganglionic cells. I have repeatedly observed the action of certain exciting causes, and particularly of nervous shocks.

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[1] *Med. News*, June 2, 1900.

Symptoms.—Enteralgia may develop very *suddenly*, but oftener it sets in less abruptly, and is then attended with eructations of gas, expulsion of flatus, and the like. In the fully-developed attack the *pain* may attain to great violence, causing the patient to "bend double" or even faint, and its character is variously described as boring, tearing, or cutting. The pain may be confined to a circumscribed spot or may be diffuse. The attacks are sometimes brief, or they may be characterized by a sudden subsidence. At other times they last for days or perhaps weeks, and then subside gradually. *Recurrences* are common, but the intervals between the attacks vary extremely in duration.

Hypogastric neuralgia is a term applied to neuralgia affecting the sensory nerves lying in the most dependent segments of the intestine. Here the nerve-fibers entering into the hemorrhoïdal plexus are involved. It is caused chiefly by tabes, by hemorrhoids, and by the neurotic state so common to females. This form of neuralgia has its seat in the hypogastric region, and is accompanied by a distressing sensation of pressure in the rectum and bladder, and by an irresistible desire to go to stool; pains also radiate to the sacrum, thighs, and perineum.

Diagnosis.—The various organic diseases and conditions mentioned under Etiology, in the course of which colic is a common symptom, must be separated from the true neurotic enteralgia. The former are distinguished from the latter by a group of symptoms peculiar to themselves (fever, aggravation of the pain upon pressure, vomiting, constipation, or diarrhea), and by the usual definite causes furnished by the history.

Renal and *hepatic colic* bear a superficial similarity to enteralgia. The former conditions, however, are distinguished first by the seat and direction of the pain, and secondly by the appearance of jaundice in hepatic colic and of hematuria in renal colic. *Rheumatism* of the abdominal muscles is easily eliminated, since it is generally combined with rheumatism in other parts of the body; the pain is also greatly increased upon throwing the muscles into contraction, as in stooping or rising; finally, it vanishes in response to the action of the salicylates.

DIMINISHED INTESTINAL SENSIBILITY.

This implies diminished peristalsis or constipation. A greater or less degree of anesthesia of the bowel attends, with a loss of desire to go to stool and an accumulation of feces in the rectum. This is a usual concomitant in many diseases of the brain and cord, with which paralysis is associated. Motor innervation may remain intact, and when atony of the intestine is absent spontaneous movements of the bowels occur; when atony is present, however, to a marked degree (motor paralysis), the feces must be artificially removed.

(*c*) DISTURBANCES OF MOTILITY.

When the contractility of the muscularis is increased from purely nervous causes the result is—

NERVOUS DIARRHEA.

This condition presents no morbid lesions. The increased contractility results from an exaggerated irritability of the motor nerves of the bowels. It may also result from morbid processes in the central nervous system and in other organs of the body; in short, the condition may be a reflex one.

Examples of this sort are caused by tabes, by gastric disturbances, as after certain foods and drinks, by dentition, and the like. Most cases, however, are encountered in persons having an abnormally irritable nervous organization—*i. e.* the neurasthenic and hysteric classes. In such the effect of mental excitement, of fright, and similar psychic influences is to induce diarrheal evacuations.

Symptoms.—The *stools* vary in number from two or three to twenty-four or more daily. In rare instances they are soft—not truly diarrheal—and formed, yet they may be quite frequent. Blood and mucus, pus, and other morphologic elements are absent from the dejections. It is characteristic of nervous diarrhea that the stools follow one another in rapid succession, usually during the morning hours, and then discontinue for the greater part of the day. The bodily nutrition is often well preserved.

In the **diagnosis** organic affections of the bowel are to be carefully eliminated.

ENTEROSPASM.

(*Spasm of the Intestine.*)

By this term is meant a concurrent spasm of both the longitudinal and circular muscular fibers, usually inducing spasmodic constipation, and sometimes total, though temporary, occlusion of the bowel.

Its *causes* are similar to those of nervous diarrhea, and the condition is clinically related to enteralgia. Neither pain nor constipation, however, is a constant feature. The stools may assume the form of a ribbon or of large rounded masses (sheep's dung), but they are not pathognomonic. They may also be covered with mucus. Ewald distinguishes between an idiopathic and a secondary or symptomatic spasm, the latter being a concomitant of basilar meningitis and of chronic lead-poisoning (see also Constipation, p. 843). Another variety affects the rectum (*proctospasm*), and is generally secondary to some other rectal affection, as fissure of the anus; it may, however, occur as a neurosis in the hysteric and nervous class of subjects.

The *diagnosis* of true functional enterospasm can only be made after all organic causes that may produce spasm of the bowel have been excluded.

CONSTIPATION.

This is a common condition as a functional neurosis. It is due to an abnormity of function of the intestinal nerves that leads to a weakened peristaltic action, and is met with in hysteria, neurasthenia, and in those suffering from the various forms of psychoses. Central nervous affections often manifest atony of the intestine as a symptom; hence this form is not a disease *sui generis*. Cases of this class do not respond to any variety of cathartics (Ewald).

Paralysis of the external sphincters is a common concomitant in a great variety of local (catarrhal) and central nervous diseases. Under these circumstances the act of defecation may be purely *reflex*, owing to loss of control of the voluntary muscles; or it may be *voluntary*, except when the person affected is not upon his guard, or during mental excitement, micturition, sneezing, and like influences.

Treatment of Intestinal Neuroses.—A suitable change of environment, including an appropriate arrangement of the dietary, is of primary importance, and is uniformly applicable in this class of sufferers. Further, the treatment of special cases has peculiar reference to the character of the nervous derangement. After making an accurate diagnosis a search for the factors of the greatest etiologic importance should be made, and these must then be vigorously assailed.

In the *secretory neuroses* an associated membranous enteritis must be corrected, the digestion must be improved if faulty, and the obstinate constipation overcome. For the latter symptom enemata containing ox-gall, either alone or in combination with salines, are especially serviceable. Kussmaul and Fleiner have obtained the best results from regular large oil-enemàta administered once or twice daily. During the painful attacks copious enemata of normal saline solution to which has been added oil of peppermint (5 drops to the pint at a temperature of 100° F.) will sometimes bring speedy relief from the pain and other distressing colonic symptoms, and will assist nature's efforts at separating the adherent membrane. Pain must be at times relieved by morphin.

In the *sensory disturbances* in which the activity of the sensory nerves is increased (enteralgia and hypogastric neuralgia) the treatment may be considered under two headings: first, the relief of the neuralgic pains; and secondly, the correction of the causes or conditions on which the enteralgia depends. If the pain be severe, opium or morphin may be required. Especially good as an antispasmodic is codein, which may suffice in all save the severer cases. The object should be to give the minimum amount of the opiate that will meet the necessities of the case, with a view to obviating a resultant constipation. In hypogastric neuralgia I have found suppositories containing opium to be little short of magical in their effects.

In cases in which there is constipation due to diminished sensibility, with a loss of motor innervation (atony of the bowel), the feces must be artificially removed unless the underlying condition can be successfully overcome. It is especially important that the environment—physical and psychic—be so regulated as to bring about an improvement in the general condition of the patient. It may become necessary to employ tonic preparations of strychnin, iron, or arsenic.

The treatment of nervous diarrhea involves the same principles, so far as the indication presented by the peculiar nervous organization is concerned, as in the sensory and secretory neuroses. It is especially important to prevent the operation of the direct causes—fright, mental excitement. Astringents and intestinal antiseptics are not called for, unless the bodily nutrition be affected thereby. Enterospasm is to be met by the same remedies that are used to control enteralgia.

IX. DISEASES OF THE LIVER.

ANOMALIES IN SHAPE AND POSITION.

Altered Shape.—Occasionally **malformations** of the liver are met with that materially alter the shape of the organ, either primarily when the result of disease, or secondarily from pressure of adjacent structures. Of the latter class the most important cause is tight-lacing, met with almost exclusively in women and producing the so-called "corset liver." The lower part of the right lobe of the liver is usually the part affected; the hepatic parenchyma is atrophied, owing to continued compression, and shows deep grooves that correspond to the position of the lower ribs. The connective-tissue capsule and the peritoneal coat are both thickened at this point, the smaller blood-vessels often being entirely obliterated. In marked cases the right lower lobe may become converted into a dense fibrous band, with only a vestige of the former liver-structure remaining. Among other acquired causes of anomalies in the shape of the liver may be mentioned deformities of the vertebræ and ribs, or tumors of the ribs or adjacent structures (the pylorus, omentum) pressing against the liver.

Diagnosis.—Rarely, clinical symptoms are present. "A constant *sensation of pressure* and *pulling* is felt in the hepatic region, and sometimes, as a result of venous stasis, there is a temporary but decided swelling of the isolated portion, and, possibly, *violent pain* and indications of irritation of the peritoneum, such as *vomiting* and an approach to *collapse*. Jaundice is rare in consequence of this deformity" (Strümpell). The danger of this condition lies in a possible mistaking it for an *abdominal tumor* (Pepper), *amyloid disease*, *passive congestion*, or *new growths* of the organ (Strümpell).

Primary alterations in the shape of the organ may be due to active or passive congestion, hereditary syphilis, hypertrophic or atrophic cirrhosis, acute yellow atrophy, carcinoma, abscess, or hydatid cyst. The accompanying *symptoms* would, of course, be those of the disease causing the deformity.

Anomalies of position are not infrequently met with, the organ being displaced upward, downward, or laterally. The most common cause of lateral displacement is found in an abnormal lengthening of the suspensory ligament. The organ may occupy the epigastric region or be displaced into the lower part of the abdominal cavity, but a change in the posture of the patient or external pressure is often sufficient to replace the liver in its normal position. The *symptoms* (if present at all) consist of a dragging sensation, often amounting to pain that may be severe and referred to the right shoulder. On *physical examination* palpation may reveal a fissure between the right and left lobes, together with a movable tumor presenting the size and normal outlines of the liver, which by manipulation may be returned to the right hypochondriac region. Percussion gives tympany over the normal area of liver-dulness, which changes to flatness when the organ is pressed or falls into its natural position.

Displacement upward may result from gastric or intestinal distention, marked ascites, or an abdominal tumor; while downward displacement may be due to a mediastinal tumor, an emphysematous lung, or a pleural effusion.

Diagnosis.—Among the conditions likely to be confounded with movable liver may be mentioned *carcinoma of the omentum* or of the *pylorus*, *dermoid cysts*, *tumors of the ovary* and *uterus*, *hydro-* or *pyonephrosis*, *tumors of the kidney*, and *chronic proliferative peritonitis*. By a careful study of the symptomatology, and in the absence of the normal physical signs over the hepatic area, the differential diagnosis can usually be firmly established, although marked fatty degeneration or atrophic cirrhosis may coexist with any of the above conditions and cause marked diminution in the area of hepatic dulness.

The **treatment** of movable liver is merely palliative, and consists in the application of a suitable bandage for preventing the displacement.

JAUNDICE.

(*Icterus.*)

Definition.—A condition in which the tissues and secretions are stained with bile-pigments. Jaundice is not a disease, but a symptom. The doctrine of a hematogenous jaundice has been successfully overthrown by the investigations of Stadelmann, Hunter, and others. All forms are due to obstruction (hepatogenous); at all events the cases of hematogenous origin must be extremely exceptional.

Hepatogenous or *obstructive jaundice* is more commonly seen in—(1) Inflammatory swelling of the duodenum or of the lining membrane of the duct, which is by far the most common factor in its causation, and demands separate consideration (*vide infra*, Catarrhal Jaundice); (2) Foreign bodies within the ducts, as gall-stones or parasites; (3) Stricture or obliteration of the duct; (4) Tumors within the duct or obstructing its orifice; (5) Pressure on the duct from without, as by a tumor of the liver, stomach, pancreas, or omentum; also by fecal accumulations, displaced organs (*e. g.* floating kidney), a pregnant uterus, enlarged glands in the fissure of the liver, and, more rarely, by abdominal aneurysm; (6) Lowered blood-pressure in the vessels of the liver favoring resorption of bile, as in simple icterus of the new-born (Frerichs).

CATARRHAL JAUNDICE.

(*Hepatogenous Jaundice; Icterus Catarrhalis; Duodeno-cholangitis; Inflammation of the Common Bile-duct.*)

Definition.—A condition characterized by a discoloration of the tissues from retention and absorption of bile and resulting from a catarrhal inflammation of the lining membrane of the ducts, more especially the larger, and of the duodenum.

Pathology.—On examining a liver and gall-bladder *in situ* the former is usually found enlarged, lighter in color than normally, and of a distinct icteroid tint. On making a longitudinal section drops of bile can be collected on the edge of the section-knife.

The gall-bladder is found distended with bile, and on firm pressure a tough plug of mucus is usually expelled from the common duct into the duodenum, after which bile flows into the intestine freely. The mucosa lining the *ductus communis* is swollen and inflamed, and the catarrhal process may extend to the cystic, and in some cases to the hepatic, duct. As a rule, that portion of the common duct lying in the intestinal wall is more frequently and more deeply involved. If the disease becomes chronic, a formation of connective tissue occurs, owing to the irritation caused by the retained secretion, and atrophy of the liver-cells, with biliary cirrhosis, may result. Suppuration is rare.

Toxic (hematogenous) jaundice, so-called, has for its lesion extensive catarrh of the intra-hepatic bile-ducts from their origin. Here duodenal catarrh is not necessary for the production of jaundice. It was formerly assumed that the pigment (hemoglobin) was liberated in the blood; but Stadelmann and others have shown that the bile containing the poison, or its irritant products (toxins), excite inflammation of the finer ducts.

Etiology.—Simple catarrhal jaundice results in a majority of cases from extension of inflammation in gastro-duodenal catarrh, and the principal *predisposing causes* are as follows: (*a*) Exposure to cold and wet; (*b*) The use of improper foods, under which heading may also be comprised faulty cooking and improper mastication; (*c*) The excessive or prolonged use of irritants (tea, coffee, alcohol); (*d*) Prolonged anxiety and mental or physical overwork; (*e*) Certain acute diseases, as pneumonia, relapsing fever, typhoid fever, and malaria (toxic jaundice, *vide supra*); (*f*) Portal obstruction, occurring in chronic heart- or kidney-disease; (*g*) More rarely it has occurred in epidemic form.

Symptoms.—Preceding the development of the distinctive features by several days, dyspeptic symptoms are in evidence (*vide* Gastro-hepatic Symptoms). The principal symptoms in detail are: (*a*) *Icterus*, or tinting of the body surface may be the first symptom noticed in this condition, appearing usually on the forehead and neck and rapidly spreading over the entire body. The conjunctivæ also early become discolored, and the general hue, though variable, is commonly a bright lemon-yellow. In chronic cases the color is apt to change to a bronzed or deep-green tint.

(*b*) *Secretions and Excretions.*—The urine and sweat are often found to contain bile-pigment, the patient's linen frequently being discolored. In extreme cases the urine may be dark-green in color, while in those of average severity it is of a lighter or deeper greenish-yellow hue. The shaken specimen foams, and the froth has a yellow color-tint.

Often the presence of bile is detected before any noticeable coloring of the conjunctivæ occurs. In cases of intense or long-standing jaundice albumin and tube-casts may be present, and the latter may be bile-stained.[1] Hyaline casts are often found in cases of moderate intensity.

The bowels are constipated, and the stools are pale-drab or slate-colored; they are usually very fetid. Diarrhea, however, may be present, owing to the production of irritating substances and decomposition.

The tears, saliva, and milk are rarely stained with bile-pigment. The expectoration also is rarely tinted, unless pneumonia or some form of pulmonary infiltration coexists.

(*c*) *Circulation.*—The pulse, although not appreciably altered in volume or tension, is usually slow (often 30 or even 20 beats per minute), though this is not an unfavorable symptom.

(*d*) The *temperature* is usually normal, although slight elevations may occur (100°-101° F.—37.7°-38.3° C.).

(*e*) *Gastro-hepatic Symptoms.*—Dyspeptic symptoms—viz. anorexia, a sense of fulness after eating with flatulence, acid eructations, nausea and vomiting, accompanied by a dull, heavy pain over the hepatic area, with some tenderness on pressure—are present. These often develop insidiously; more rarely they occur suddenly with a severe rigor or chill, violent headache, and vomiting—*e. g.* at the onset in the epidemic form.

(*f*) *Cutaneous Phenomena.*—Pruritus or itching often becomes a troublesome symptom, being more common, however, in the chronic forms. Lichen, urticaria, furuncles, and sweatings (diffused and localized) may develop, the latter being often limited to the skin covering the abdomen and the palms of the hands.

A peculiar disease of the skin called *xanthelasma* or *bita higoidea* may also occur. It consists of bright-yellow spots, slightly elevated, appearing on the eyelids, and rarely on other parts of the body.

In the more severe forms spots of ecchymosis, and in some instances profuse hemorrhages, may occur into the skin and mucous membranes. These are usually associated with other symptoms of a grave type.

(*g*) *Nervous Symptoms.*—Headache and vertigo are common; irritability of temper, despondency, and wakefulness or mental dulness almost equally so. With the oncoming of darkness vision may grow indistinct (*hemeralopia*) or it may attain unnatural clearness (*nyctalopia*). Rarely, objects look yellow (*xanthopsia*). The nervous phenomena observed in catarrhal jaundice are attributable to the effects of the bile-acids. In certain cases, however, associated with destruction of the hepatic substance, as in acute yellow atrophy, carcinoma, cirrhosis, and fatty degeneration, grave cerebral symptoms (acute delirium, convulsions, and coma) may develop suddenly and prove fatal. This class of symptoms has been named *acholia*, *cholemia*, or *cholesteremia* (the latter owing to the mistaken supposition that cholesterin is the poisonous product). The true nature of the toxic agent in the blood is unknown. In some fatal terminations of this character death was due directly to a renal complication.

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[1] *Tests for Bile.*—*Gmelin's test*, or the play of colors, consists in bringing a few drops of urine in contact with the same quantity of commercial nitric acid on a plain white slab, whereupon various shades of yellow, green, red, and violet are produced.

Rosenbach's test is made by filtering the suspected urine and touching the filter-paper with a drop of nitric acid. If bile be present, a green circle will form at the point of contact. (See also Choluria, p. 952.)

The **physical signs** in a case of simple catarrhal jaundice show on palpation and percussion an increase in the hepatic area, the lower border of the liver projecting in some instances several fingers' breadths below the ribs. Rarely, the distended gall-bladder projects below the lower lobe of the liver, as when there is complete obstruction near or at the duodenum, and then it can be distinctly palpated.

Diagnosis.—The etiology (errors in hygiene and diet), a history of previously existing gastro-intestinal catarrh, the age of the patient (young adult life), and the appearance of the jaundice unaccompanied by pain or general emaciation, together with an absence of symptoms pointing to *cirrhosis*, *carcinoma*, or *acute yellow atrophy*, form a characteristic grouping of clinical indications.

Duration and Prognosis.—The *duration* of catarrhal jaundice varies from two to eight weeks. If the symptoms continue longer than two months, grave doubts may be entertained as to the case being one of simple jaundice. The prognosis is guardedly favorable. A rise of temperature usually indicates mischief (Pepper), while hemorrhages of the skin and mucous membranes always influence the issue unfavorably.

Treatment.—The *diet* and *hygiene* are the first considerations in the treatment. Rich, highly seasoned foods, rich pastries, fats, and sweets, are to be interdicted; starchy foods, lean meats, bread, soups (containing no fat), and green vegetables may, however, be used in moderation. Skimmed milk, butter-milk, and alkaline drinks (Vichy and Saratoga mineral waters) may be used freely, while sour wines, lemonades, and tamarind-water are allowable. Systematic bathing (Turkish or Russian baths, under supervision) and regulated hours of sleep exert a beneficial effect. The free use of pure water often does good by increasing the flow of bile and by dislodging plugs of mucus.

Gerhardt and Kraus have recommended the faradic current, applied over the region of the gall-bladder; manipulation has also been tried with a view to removing the obstruction, but without success.

The first *therapeutic indication* is to keep the bowels freely soluble by the use of saline aperients, as Hunyadi water or Carlsbad salts (½ to 1 teaspoonful in hot water before meals). The latter remedies tend to lessen the catarrhal inflammation by depleting the mucous membranes. In obstinate constipation calomel, rhubarb, the extract of colocynth, or castor oil may be employed.

Conspicuous among other remedies may be mentioned the alkalies, sodium bicarbonate, salicylate, and phosphate, which tend to increase the flow of bile and render it less thick; hydrochloric acid (which, according to Ewald, by aiding digestion prevents the formation and consequent absorption of toxic substances), in combination with the bitter tonics—gentian, quassia, and nux vomica; ammonium chlorid, which sometimes proves beneficial; and silver nitrate (gr. ⅛–¼—0.008–0.016, thrice daily).

Injections of cold water (60°–70° F.—15.5°–21.1° C.), daily, in quantities of 1 or 2 quarts (1–2 liters), are highly recommended as promoting the secretion of bile; while lavage, practised daily and over a protracted period of time (one to two months), has proved highly beneficial, especially when gastro-duodenal catarrh has existed.

Itching.—This troublesome symptom may often be relieved by the external application of a solution of borax or sodium bicarbonate (℥ss–Oj—16.0–512.0), or of menthol and alcohol (gr. x–℥j—0.648–32.0). Internally, large doses of the bromids or the continued use of pilocarpin, as recommended by Witkowski, are worthy of a trial.

Flatulence.—To this end it is important to regulate the diet, avoiding starches and sugars. Charcoal tablets, bismuth subnitrate or salicylate, and beta-naphtol are all useful. Irrigation of the colon with some efficient antiseptic in solution is often a factor of service.

Headache is caused by the circulation in the blood of some toxic principle. Of drugs, caffein citrate, camphor monobromate, and phenacetin, either singly or in combination, may be recommended.

When the obstruction is due to mechanical causes (biliary calculi, tumors pressing on the duct) the treatment is surgical.

OTHER FORMS OF JAUNDICE.

Modern experiments, as I have said (*vide* p. 852), tend to show that the so-called hematogenous jaundice is always hepatogenous—*i. e.* the blood-dyscrasia probably exerts a toxic influence on the liver-cells and intra-hepatic gall-ducts; and there may at the same time be a more rapid blood-destruction in the liver (Neumeyer, Stadelmann, *et al.*).

There are instances of jaundice in which active hemolysis is an element of importance (Stengel): (*a*) Severe infections, as septico-pyemia, yellow fever, acute yellow atrophy of the liver, and the jaundice of the new-born. (*b*) Grave forms of anemia, as pernicious anemia and chlorosis. (*c*) Certain poisons, as the venom of snakes, chloroform- and ether-poisoning; also in poisoning by phosphorus, arsenic, mercury, and other minerals.

Experiments conducted by Münzer, Starling, Hopkins, and others, tend to show that the liver-function is not suppressed by many of the conditions and affections mentioned above; but, on the other hand, that increased secretion (*polycholia*) and increased formation of bile-pigments (*polychromia*) may prevail. Again, the poisons or toxins may cause swelling of the cells and compression of the biliary capillaries; this would cause obstruction of the outflow of bile and the subsequent absorption. Lastly, circulatory disturbances (emotional jaundice) may lead to overproduction, or obstructive retention, of bile.

ACUTE INFECTIOUS CHOLECYSTITIS.

Definition.—An acute inflammation (infective) of the gall-bladder. It is rare.

Pathology.—Three pathologic varieties—catarrhal, suppurative, and phlegmonous—are recognized. The gall-bladder progressively enlarges and becomes filled with muco-purulent or purulent or (rarely) hemorrhagic material. The cystic duct is often occluded. In the suppurative form (the most prevalent) ulcers may coexist and perforation, followed by localized peritonitic abscess or acute diffuse peritonitis, may occur. The lesions of cholangitis, either catarrhal or suppurative, may be associated.

Etiology.—The *bacterial* excitants include the streptococci, staphylococci, the pneumococcus, the colon bacillus, and the typhoid bacillus.

Among predisposing conditions are many of the acute infections, as typhoid, typhus, malaria, sepsis, pneumonia, puerperal fever, and cholera.

DaCosta has collected 58 cases of typhoid cholecystitis. His figures show that it may occur at almost any *age*, and of 48 cases in which the sex was stated, 26 were males and 22 females. The etiologic importance of gall-stones has been greatly overrated in the past.

Symptoms.—The onset is abrupt, with pain (often paroxysmal) in right side of the abdomen or epigastrium. The region of the gall-bladder is acutely sensitive, and with the development of spreading peritonitis the tender area grows correspondingly. Rigidity of the right rectis may be observed. In many cases a *tumor* occupying the seat of the gall-bladder is present and has marked diagnostic importance. It is detected on palpation as a firm, pear-shaped tumor or as a "mere resisting mass below the costal margin." The latter is often due to peritonitic abscess following perforation.

Nausea and *vomiting*, which may be persistent, are usual symptoms at the outset. *Jaundice* occurred in 17 out of 58 cases (DaCosta). Among the *general symptoms* chills are conspicuously absent. *Fever* may be present, but by no means always; the pulse becomes rapid and feeble, the abdomen distended, and prostration profound. In the suppurative form a blood examination generally shows leukocytosis. This serious affection may be entirely latent.

Differential Diagnosis.—*Appendicitis* may be mistaken for cholecystitis, particularly if the appendix be situated abnormally high up. The discrimination would here rest upon the history (following typhoid or other infection), the presence of a tumor and marked sensitiveness in the region of the gall-bladder, corroborated by jaundice.

Acute intestinal obstruction may be closely simulated in cases in which adhesions between the gut and gall-bladder are present. In such cases exploratory celiotomy is to be advised or at least considered with a view to clearing the diagnosis. *Recurrent cholecystitis*, a not uncommon complaint, gives the history of recurring attacks of pain simulating cholelithiasis. In one of my cases Laplace operated and found the gall-bladder somewhat enlarged and the seat of catarrhal cholecystitis. Osler suggests that in some of these cases gall-stones may have been present and have passed before the operation (see also p. 856).

Prognosis.—This is dependent upon the special variety, although it is among the most fatal of diseases. A fatal result is the rule in purulent and phlegmonous cholecystitis. In the catarrhal form recovery is not infrequent (DaCosta). Pneumonia may be the immediate cause of death. Phlegmonous cholecystitis may end in gangrene.

Treatment.—This embraces absolute rest, rectal alimentation, the relief of pain by the judicious use of morphine, and of other symptoms as they arise. The circulation must be supported by stimulants as a rule. If the diagnosis of suppurative or phlegmonous cholecystitis can be established surgical intervention is imperatively demanded as a rule.

BILIARY CALCULI.

(*Gall-stones; Cholelithiasis.*)

Definition.—Concretions formed in the gall-bladder, due to an altered physiologic function or pathologic change; they often set up characteristic disturbances (*cholelithiasis*).

Etiology.—As a result of biliary retention increased consistency and a concentration of bile occurs, and certain constituents that were before held in solution are thrown down. Among common **predisposing causes** may be mentioned: (*a*) *Female sex*, especially between the ages of forty and sixty. Senac's statistics, out of a total of 311 individuals, give 227 women (Dujardin-Beaumetz). (*b*) *Irregular meals* and an *excessiue diet of starches and of fats*, combined with a sedentary life. Among minor causes are *constipation*, *tight-lacing*, *pregnancy*, *chronic obstruction* to the flow of bile (as from tumors, catarrh of the ducts, or heart-disease, as mitral stenosis), and, more rarely, the rachitic and lithic-acid diathesis. (*c*) It may occur during childhood. (*d*) Incidence. Brockbank found among 13,047 completed postmortem records, 7.4 per cent. were gall-stones. (*e*) Infectious cholecystitis.

Composition and Appearance.—Water comprises from 2 to 5 per cent. of the composition of gall-stones, the chief solid constituent being cholesterin, and the remainder being composed of bile-pigment and salts (lime, potash, soda, and perhaps traces of iron and copper). *Pigment-lime* may be, though rarely, the main constituent. In *size* they vary from the smallest particle of sand to that of a goose-egg. Fagge reports a calculus weighing, in a dry state, 462 grains (30.0). The *color* varies from white or light-yellow to that of a dark-green (as in pigment-lime calculi), and may present any variation between these two extremes. The *nucleus* often consists of cholesterin, the outer layer being usually the harder, and made up, for the most part, of lime-salts. The center of the nucleus generally consists of desquamated epithelium or dried mucus, and on cross-section concentric laminæ are usually developed. The cholesterin gall-stones cut like wax, are white, and the cut section presents a crystalline appearance. Other forms are apt to be brittle. The *surfaces* may be smooth, striated, or hollowed out, solitary calculi being usually round or ovoid, while multiple stones often present smooth facets, due to the massing together of the calculi (Dujardin-Beaumetz). They are usually olive-shaped, but may be pyramidal, cylindric, lenticular, pisiform, cubic, finger-shaped, or olivary. Their *seat* is usually the gall-bladder, but they may be found anywhere along the biliary passages.

Symptoms.—There may be no subjective symptoms. Indeed, in the majority of cases of cholelithiasis biliary colic does not occur. The passage of a calculus through the duct, if it sets up a "perialienitis" or inflammation of the structures surrounding it, will give rise to *hepatic colic*, whereas a permanent blocking of the duct will cause symptoms of chronic obstruction (*vide infra*).

Hepatic Colic.—When a gall-stone becomes impacted in a bile-duct the patient experiences *agonizing pain* (tearing, cutting, or lancinating in character) in the right hypochondriac region, radiating to the right shoulder, and accompanied often by profuse *sweating*, *vomiting*, and a *feeble*, *running pulse*. The most common *seat* of the pain is two to three inches to the right of the median line and about an equal distance below the ensiform cartilage. Less frequently it is in the region of the gall-bladder. This happens in cases in which the gall-stone is impacted in the cystic duct, and may be due to distention of the gall-bladder, or to associated cholecystitis. The pain is sometimes so severe as to produce *syncope*. Hepatic colic, however, may occur independently of the passage of biliary calculi, as from inflammation of the gall-bladder. On the other hand, large calculi have been found in the dejecta without having excited hepatic colic. I recently saw an instance of this kind in which the gall-stone was of the size of an English walnut. A *rigor* or *chill* often precedes the attack, which is usually accompanied by moderate *fever*, the temperature reaching 101°–102° F. (38.3°–38.8° C.). If the stone passes through the duct without becoming impacted, jaundice and pain may either be only slight or entirely absent. When, however, occlusion of the common duct occurs, the *jaundice* becomes intense. This symptom, however, may be present, though less marked, before the gall-stones reach the *ductus communis*. Jaundice occurs in about 50 per cent. of the cases (Fitz), and it sets in from eight to twenty-four hours after the onset of the attack of pain. *Physical examination* reveals on inspection a slight prominence in the hepatic area, and on palpation the edge of the liver can often be distinctly felt below the costal margin—at times as low as the umbilical level. The enlarged liver is sensitive on pressure, and particularly the gall-bladder, which can be often palpated. If the latter viscus contains many calculi, crepitation may be noticeable to the palpating fingers (rarely), and a friction-sound may be distinguished on auscultation. The swollen organ, after the cessation of the colic, quickly subsides. *Recurrences* of the painful attacks after varying intervals of time are common. Finally, the gall-stone is expelled and the colic ceases to return. Multiple stones, however, may be passed. In severe cases the symptoms of profound shock may develop.

Rupture of the duct, followed by fatal peritonitis, has been known to occur. Attacks of biliary colic are of variable *duration*, lasting from a few hours to a few days, and in some instances one or more weeks. Sudden cessation of the pain is usually followed by rapid disappearance of the jaundice (when present) and the discovery of the *stone in the feces*. Examination of the *urine* after the paroxysm reveals bile and an abundance of uric acid and urates. The *pulse* often becomes slowed. Exner has demonstrated the presence of about 0.4 per cent. of sugar in the urine in 39 out of 40 cases of gall-stones. On the other hand, Kausch has found glycosuria in only one of 85 cases of cholelithiasis.

The **prognosis** of biliary calculi as regards life is good, but as regards recovery only guardedly favorable. Cardiac distress with palpitation may occur during hepatic colic and form a serious complication. Fatal syncope has also been known to occur, and fatal intussusception has followed the impaction of gall-stones in the region of the ileo-cecal valve. If evidences of an infectious inflammation arise, the outlook is then more serious.

Diagnosis.—The diagnosis of gall-stones is sometimes extremely difficult on account of the obscure clinical symptoms and the entire absence of physical signs. When, however, the calculus becomes impacted in the duct, symptoms of biliary colic—intense pain in the epigastrium and right hypochondriac region, radiating to the back and right shoulder—usually appear. There are also fever, vomiting, and in one-half the instances jaundice and the finding of the stone in the dejecta.

Differential Diagnosis.—*Gastralgia* usually occurs in individuals with neurotic tendencies, and is characterized by severe paroxysmal pains in the epigastrium, extending to the back and base of the chest. It occurs often when the stomach is empty and is relieved by eating. Firm pressure over the epigastrium often alleviates the pain temporarily, and the absence of fever, jaundice, stones in the dejecta, and the negative urinalysis, together with the history of former attacks, would tend to differentiate it from hepatic colic.

Renal Colic.—The pain in this condition starts in the flank of the affected side and is transmitted down the ureter. The testicle and inner side of the thigh are very painful, the former being often retracted. Micturition is frequent and sometimes painful, and the urine is scanty in amount and often mixed with blood.

Intestinal Colic.—In this variety the pain is of a boring or twisting character, usually centering about the umbilicus. It is relieved by firm pressure. Abdominal distention is often present, and relief comes with the passing of flatus. Usually there is a history of an indiscretion in diet. When due to *lead-poisoning*, the history, the blue line on the gums, and the presence of wrist-drop would tend to confirm the diagnosis.

Reflex colic, due to uterine or ovarian disease, may also occur. The recurrence of the attacks, together with causes and symptoms pointing to pelvic disease, would establish the identity of the condition.

Pollatschek, Riedel, Stockton, and others hold that biliary colic may arise from cholecystitis (due to an infection) without the presence of any gall-stones. With the cholecystitis, localized peritonitis and angiocholitis are often associated.

CHRONIC OBSTRUCTION OF THE DUCTS BY GALL-STONES.

The obstruction may exist in the *ductus choledochus*, in the cystic duct, or in both.

1. **Obstruction of the Common Duct.**—**Pathology.**—The result of the irritation produced by the presence of the stone is a catarrhal process (*cholangitis*) that may either remain chronic or terminate in suppuration (*suppurative cholangitis*). In a case of simple obstruction the gall-bladder is often moderately enlarged, though rarely extending below the lower border of the liver. The common duct is greatly distended, the stone being usually located near its termination; it is distinctly felt just beneath the mucous membrane of the descending duodenum. Occasionally two or more calculi are present, completely obliterating the canal. The hepatic duct and its branches are greatly dilated, and often contain thin, colorless mucus, the membrane lining the ducts being smooth and clear. The liver in these cases is firmer in consistency than normal, showing some increase in the connective-tissue elements (biliary cirrhosis). Following moderate enlargement of the organ progressive atrophy may rarely occur. When *suppuration* has occurred the mucous membrane

is greatly swollen and reddened, and in some instances shows erosions or ulceration (Ulcerative Angiocholitis). The process often extends through the hepatic and cystic ducts into the liver and gall-bladder, giving rise to the localized abscesses in the former and to empyema of the latter. In some instances the gall-bladder has been perforated and abscesses have formed between the liver and stomach. *Diverticula* are sometimes found postmortem, containing biliary calculi.

While cholelithiasis is by far the most common cause of catarrhal, suppurative, and ulcerative angiocholitis, it not rarely complicates hydatid disease, carcinoma of the bile-ducts, and the acute infections, particularly typhoid fever (*vide* Acute Infectious Cholecystitis, p. 857). Rarely foreign bodies (fish-bones, lumbricoids) operate as excitants.

Symptoms.—Chronic obstruction by gall-stones, with coexisting *catarrhal inflammation* (catarrhal angiocholitis), is characterized by a distinctive group of symptoms, among the most prominent of which are—

Jaundice.—This may be constant and very intense, or intermittent and slight, depending upon the amount of obstruction present. In some cases it disappears entirely for several months, and then recurs with varying intensity (ball-valve action of the stone). *Itching* is, as a rule, a most distressing feature.

Pain, occurring in paroxysmal attacks and referred to the region of the liver. This is accompanied by fever that may reach a high degree (102°–103° F.—38.8°–39.4° C.), also by chills and sweating, resembling somewhat the paroxysms of malaria. Painful points in the right side posteriorly may be annoying; these are either constant or paroxysmal.

The ***chills*** are often intense, and may present a quotidian, tertian, or quartan form. The temperature of the intervals is normal. The peculiar exacerbations of temperature were first described by Charcot, and to them has been given the name of *Charcot's intermittent fever.* Concerning their nature Murchison writes: "These paroxysms may be more or less periodic, and may extend over several months, without necessarily indicating pyemic hepatitis, the patient ultimately recovering." He adds that they are probably analogous to febrile paroxysms produced in passing a catheter along the urethra. Charcot believes the etiologic factor to be a septic poison, bacterial in origin and the result of chemical changes in the bile. Various microörganisms have been detected in the bile in such cases (bacterium coli commune, streptococcus pyogenes, *et al*).

Gastric Disturbances.—These may excite alarm during the paroxysm. Intense pain is complained of in the epigastrium, accompanied often by persistent nausea and vomiting, which, however, usually subsides at the close of the paroxysm, while the jaundice at this time deepens. The attack may persist for years without progressive failure of health.

The symptoms of *suppurative cholangitis* are intense. The *paroxysms* of fever occur more frequently, the temperature merging into the remittent type. Grave constitutional symptoms, indicating septicopyemia, are present, and the case rapidly tends to a fatal issue. The attacks of colicky pain occur with jaundice, but the latter symptom is less intense than in the catarrhal form. As to hepatic enlargement, the converse is true; this organ takes on progressive enlargement and "may descend as low as the umbilicus, the swelling being uniform and smooth and tender to pressure" (Robson). It should be borne in mind that pain may be absent when the disease is not dependent on gall-stones. Pneumonia and empyema are serious and not uncommon complications. In *ulcerative angiocholitis* severe hemorrhage may occur, resulting either in melena or hematemesis. Mayo Robson reports a case in which hematemesis was the only antemortem symptom and had been the cause of death. The condition being associated with septic infection, it leads to the constitutional disturbances of septicemia or septico-pyemia.

2. **Obstruction of the Cystic Duct.**—This almost invariably causes distention of the gall-bladder (dropsy of the gall-bladder), which may be felt distinctly below the lower edge of the liver as a pyriform, fluctuating tumor. If obstruction of the cystic duct alone occurs, *jaundice* may be entirely absent, the bile in the distended tissues being replaced by a thin, mucoid fluid. This is more apt to exist as the obstruction becomes more chronic. In some instances the distention is so great as to reach below the umbilicus, and the dilated viscus has even been mistaken for an *ovarian tumor*. Osler records a case in which 18 oz. (556.0) of fluid were removed from the gall-bladder. The contents are neutral or alkaline in reaction, albumin being often present in abundance. Catarrhal inflammation of the gall-bladder is often associated, causing *pain* and *sensitiveness* in the region of the organ. The pain may be severe and simulate biliary colic or appendicitis. The examiner can *feel* an elastic, gourd-shaped tumor closely connected with the liver, movable in respiration in the vertical, and also, under the influence of the palpating fingers, in the lateral, direction. I have observed a tongue-like projection of the anterior margin of the right lobe, to which Riedel first called attention. Given a gall-bladder well filled with stones and a relaxed abdominal wall, gall-stone crepitus may be detectable.

The writer has reported some cases giving a more or less characteristic clinical history of cholelithiasis, in which gall-stone crepitus on palpation furnished absolute proof of stones in the gall-bladder. In one case he combined auscultation with palpation and detected a faint grating sound.[1]

If the obstruction persist for a length of time, calcification or atrophy of the bladder are common sequelæ. Complete obliteration of the cavity of the gall-bladder may ensue.

Among rarer sequelæ of chronic obstruction may be mentioned—(*a*) *Empyema of the Gall-bladder.*—When this takes place the organ becomes greatly distended, and has been known to contain as much as a pint of purulent material. The *symptoms* of suppurative cholecystitis simulate and accompany those of purulent cholangitis; they are sometimes preceded by those of catarrh of the gall-bladder and ducts. Perforation may occur, giving rise to circumscribed periportal abscesses or to generalized peritonitis (see also *Acute Infectious Cholecystitis*, p. 858).

More Remote Effects of Gall-stones.—These will be spoken of under three headings:

1. Stricture of the duct, resulting from ulceration and cicatrization produced by the passage of a stone.

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[1] *International Medical Magazine*, Dec., 1899.

2. Intestinal obstruction, due to impaction of gall-stones.
3. Biliary fistulæ resulting from perforations.

1. **Stricture of the Duct.**—Obliteration of the common duct may result from the passage of a gall-stone, giving rise to ulceration and cicatrization, or the stone may become impacted and lead to adhesions and permanent closure of the duct below it (Murchison). When due to ulceration the seat of the stricture is usually low down in the common duct.[1]

Symptoms.—The symptoms are those of chronic obstructive jaundice (Osler). In many cases there will be an antecedent history of the passage of gall-stones. In all cases in which the symptoms of gall-stones are followed by permanent jaundice without pain it may be suspected either that the calculus has become firmly impacted or that it has produced organic stricture or closure of the duct.

2. **Intestinal Obstruction from Impaction of Gall-stones.**—The ileum is commonly the seat of obstruction by gall-stones, that may give rise to intussusception or cause ulceration and gangrene of the bowel with perforation and fatal peritonitis. The latter event, however, occurs more frequently when the biliary concretions are situated in the cecum. Rarely they are found in the appendix, causing, as other foreign bodies, inflammatory changes, followed by ulceration and in many cases by perforation and death. Cases of impaction in the rectum of biliary calculi have been recorded. I have recently seen a case with Dr. R. Bruce Burns.

Symptoms.—If the impaction occurs in the small intestine, the abdomen becomes tympanitic and tender on pressure. The contents of the stomach are first vomited, followed by bile and stercoraceous matter. Obstinate constipation persists, and symptoms of peritonitis develop and continue until either the impaction disappears or death ensues. Ileus, the result of biliary concretions, is common in females of advanced age. The history of previous acute attacks would tend to confirm the diagnosis. The pain is intense. The duration of the last attack is often short.

3. *Perforation* may occur with the establishment of *fistulous communications* between the gall-bladder and stomach, intestinal canal, bladder, vagina, lungs, abdominal parietes, or portal vein. Fistulæ between the gall-bladder and stomach are rare, though cases are recorded by Oppolzer, Frerichs, Cruveilhier, Murchison, and others. Cruveilhier states that vomited gall-stones necessarily reach the stomach through fistulous tracts, as the passage through the pylorus would be impossible.

Fistulæ into the duodenum are of much more common occurrence, ulceration taking place usually in the fundus of the gall-bladder and in the descending or third portion of the duodenum: 39 cases are recorded of fistulous communication with the colon (Osler). I have reported a fortieth case.[2] In 6 of 9 cases reported by Murchison carcinoma of the gall-bladder was present. Fistulæ into the urinary passages may occur, 2 authenticated cases being reported. The distended gall-bladder may come in contact with the urinary viscus, or the stone may perforate into the pelvis of the kidney and pass through the ureter into the bladder.

Fistulous openings through the abdominal parietes are the most common of all fistulæ, the place of exit of the biliary concretions being usually in the region of the gall-bladder or at the umbilicus, to which it may be directed by the suspensory ligament of the liver. As many as 600 stones have been removed from the gall-bladder in this manner. Advanced life and female sex are said to be predisposing causes. Courvoisier's statistics show 184 cases, in 78 of which recovery took place.

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[1] In vol. ix. pp. 22 and 130, *Pathologic Transactions*, two cases are recorded in which the strictures were exactly similar to those of the urethra, one being situated in the hepatic duct of the left lobe and the other in the common duct.

[2] *Clinical Lecture*, *International Clinics*, vol. ii. third series, p. 27.

Fistulæ into the pleura, bronchi, and vagina have been recorded, but are extremely rare. Courvoisier records 24 cases of fistulæ into the lungs, only 7 of which terminated in recovery. Fauconneau, Dufoesne, Frerichs, Bristowe, and Murchison mention cases of fistulæ into the portal vein, with the presence of biliary concretions in the latter.

Diagnosis.—I would strongly urge an exploratory celiotomy as an accurate means of diagnosis in obscure cases.

Treatment of Foregoing Conditions.—The indications for treatment in *cholelithiasis* are (*a*) to remove the cause; (*b*) to relieve the paroxysms of hepatic colic; and (*c*) to adopt palliative or radical measures for the removal of the stones.

Preventive Treatment.—In this, as in the treatment of jaundice, diet and hygiene play an important part. The former should be as simple as possible, consisting largely of skimmed-milk, lean meat, eggs, fruit, and green vegetables. Fatty foods, sugars, starches, and pastries are to be strongly interdicted. All foods should be thoroughly masticated, so as to digest easily, and meals should be taken at regular intervals. Systematic exercise in the open air is of signal value, as it stimulates the flow of bile. Punkhauer strongly recommends horseback-riding, believing this to be efficient in removing obstructions in the common duct.

Among the drugs mostly used in the treatment of this condition I would advise the following: Sodium sulphate, combined with the extract of taraxacum (Harley); ox-gall (Dubney), in 5- to 10-gr. (0.324–0.648) doses, three times daily (to relieve flatulency and stimulate the biliary secretion); sodium salicylate (gr. x to xv—0.648 to 0.972, three times daily); and sodium chlorate (gr. iv to vj—0.259 to 0.388) three times a day (Schiff).

The bowels should be kept freely soluble, constipation being carefully avoided. In my own experience a dram (4.0) of sodium phosphate or of Rochelle salts in concentrated solution in the morning on rising has yielded excellent results. Other laxatives whose use is to be advised and encouraged are cascara sagrada, podophyllin, and rhubarb.

Treatment of the Paroxysm of Biliary Colic.—At the very onset of an attack of hepatic colic the prompt exhibition of morphin or of codein may greatly mitigate an attack. The former may be given hypodermically in $\frac{1}{8}$- to $\frac{1}{4}$-gr. (0.008–0.016) doses every hour until relief follows; the latter is exhibited by the mouth in doses of 1 gr. (0.0648) every hour. Inhalations of chloroform, with morphin hypodermically, the former being continued until the latter has taken effect, may be regarded as the typical treatment during an attack.

Hot baths and hot applications (with counter-irritation) over the liver are valuable aids in the treatment of hepatic colic, being given at a temperature of 98° to 100° F. (36.6° to 37.7° C.), and continued for twenty minutes if endurable, so as to effect relaxation. If cardiac depression results and the pulse becomes weak, the baths should be discontinued.

Hot flaxseed-poultices, cloths wrung out of hot water, hot hop-bags, or turpentine stupes may be applied over the hepatic region until the attack subsides. Ice-poultices have been advised by Buchetan.

If shock or syncope should develop, the body-temperature must be maintained by hot bottles or bricks placed in contact with the surface of the body, together with strychnin (gr. $\frac{1}{30}$—0.0021), atropin (gr. $\frac{1}{150}$—0.00042), and brandy (1 dram—4.0) hypodermically.

Nausea and vomiting may be reduced by 15-drop doses of spirits of chloroform every half hour; also by brandy and soda-water or champagne.

In mild cases sodium salicylate (gr. viij–xv—0.518–0.972 in twenty-four hours), recommended by Prevost and Binet, or codein (gr. j), with phenacetin (gr. x), every few hours gives relief. The free use of olive oil or glycerin in hepatic colic has been followed by a beneficial effect (Rosenberg, Goodhart). The former is given in quantities of 4 to 6 oz. (128.0–192.0) by the mouth every three or four hours, nausea being prevented by concealing the taste with lemon-juice; the latter, recommended by Ferrand, is given in doses ranging from 1 to 2 tablespoonfuls, repeated in the same length of time. Both remedies are supposed to do good by increasing the flow of bile, thus forcing the stone outward toward the bowel.

Treatment for Removal of Gall-stones.—The palliative treatment consists in the administration of agents that tend to increase the flow of bile. The free use of pure water by the mouth, together with copious rectal injections daily of cold water, has been found effective. It may be rendered alkaline by sodium bicarbonate or borate in a 3 per cent. solution.

A course of alkaline treatment at some of the more noted mineral springs (Bedford, Vichy, Carlsbad) is often attended with good results. Perhaps the three best cholagogues that may be mentioned are sodium phosphate, sodium cholate, and ox-gall. Olive oil and glycerin also increase the secretion of bile.

Willoughby reports a case in which prompt recovery ensued from the use of toluylenediamine after three years of unsuccessful treatment; he began with 1 grain daily, and increased to 2 grains.

Agents to dissolve the stone have been tried at various times, among them being *Durande's method* (turpentine and ether), but, so far, all such methods of treatment have been unsuccessful.

Of the various surgical measures for the removal of gall-stones the following are the chief: (*a*) Removal of the stone from the common duct (choledochotomy); (*b*) Removal of the stone from the cystic duct (cholecystotomy); (*c*) Establishing a fistulous opening between the gall-bladder and the bowel (cholecystenterostomy); (*d*) Extirpation of the gall-bladder (cholecystectomy), the latter operation giving a mortality of 17 per cent., according to Murphy's statistics. And operative procedure is indicated in infectious (suppurative) cholecystitis as well as in infectious (suppurative) cholangitis.

The treatment of suppurative cholangitis has for its principal object evacuation and drainage of the purulent contents of the biliary passages. To this end cholecystotomy should be advised as a rule.

CARCINOMA OF THE BILE-DUCTS.

THE biliary passages may be the seat of carcinoma, which may occur primarily and exist over a long period of time without being recognized.

Pathology.—The gall-bladder, as the result of obstruction of the duct, is often greatly distended, measuring as much as 7 inches (17.7 cm.) in length (in a case reported by Harley) from the entrance of the duct to the fundus, and being filled with a cloudy liquid, somewhat resembling barley-water, that contains flakes of epithelium, granular matter, and particles of inspissated bile. If the growth be near the duodenal orifice, the common and cystic ducts are often greatly distended, and the dilatation may extend into the hepatic ducts and their branches. The liver may be enlarged, and in some instances presents the secondary nodules that are characteristic of the disease. Microscopically, carcinoma of the gall-bladder exhibits marked variations in different cases; "it may be either columnar or spheroidal-celled" (Rolleston).

Etiology.—The causes of carcinoma of the bile-ducts are the same here as elsewhere, and among these the mechanical or inflammatory theory of Virchow must be accepted. Tight-lacing and mechanical irritation by gall-stones are followed in many instances by cancerous degeneration; Osler states that " biliary calculi are present in at least seven-eighths of all cases." Among other factors, heredity and age (after forty) play an important part. Although carcinoma of the *liver* undoubtedly occurs more frequently in males, Musser found that out of 100 cases of carcinoma of the *ducts*, 75 were female; and Ames found the ratio to be 4 to 1 in favor of females.

Symptoms.—The signs and symptoms, according to Harley, present nothing characteristic to distinguish them from other causes of obstruction in the ducts. On *palpation* in the early stages the gall-bladder is found moderately enlarged, but later it rapidly undergoes diminution in size. *Jaundice* becomes very intense, and remains permanent. Throughout the course of the disease all the symptoms referable to chronic obstruction of the duct by gall-stones (paroxysmal pain, gastric disturbance, rise of temperature, Charcot's fever) may develop.

Examination of the urine and feces reveals the presence of *bile-pigment* in the former and its absence in the latter. The urine often shows the presence of bile-stained casts (*vide* Fig. 61).

Ascites not rarely occurs during the later stages, with the involvement of surrounding organs by contiguity, as well as with the appearance of secondary nodules in the liver and the development of cachexia.

Diagnosis.—Carcinoma of the biliary ducts cannot always be detected by physical examination. Distinct evidence of chronic obstruction of the duct, as persistent and intense jaundice (which occurs in three-fourths of the cases), the development of cachexia and the absence of cancerous involvement of other organs, however, will tend to characterize it. Often a hard tumor-mass is present in the region of the gall-bladder, projecting in the direction of the umbilicus. It should be recollected that the bile-ducts are oftener the seat of the primary affection than the liver. An assured diagnosis, however, is often impossible.

Prognosis.—The prognosis of carcinoma of the bile-ducts is, like that of other organs, absolutely fatal, though the course of the disease is not so rapid as that of carcinoma elsewhere until secondary involvement of the liver occurs.

Treatment.—The treatment is merely palliative. Operative measures are rarely justifiable, since the disease is rarely recognized before the liver becomes involved. As seven-eighths of the cases follow obstruction of the duct by gall-stones, the preventive treatment of the latter should be carefully observed whenever symptoms of disordered liver-function manifest themselves.

The treatment of the pain, anemia, and emaciation will be described in the discussion of Carcinoma of the Liver (*vide* p. 903).

STENOSIS OF THE BILE-DUCTS.

Stenosis may result from any of the following causes: (*a*) *Round-worms* in the duct (rarely); (*b*) Foreign bodies, as seeds; (*c*) Ulceration and cicatrization following the passage of gall-stones (most commonly); (*d*) Pressure from without, as from tumors (carcinoma chiefly) of the head of the pancreas and pylorus (rare); (*e*) Abdominal tumors; (*f*) Aneurysm of the abdominal aorta or of the celiac axis (rare); (*g*) Secondary enlargement of the lymphatics of the liver (common); (*h*) More rarely in man than in the lower animals distoma hepaticum of liver-flukes and echinococci; (*i*) Adhesions due to chronic peritonitis.

Pathology.—If the stenosis is of recent origin, the liver is enlarged and shows more or less congestion, with some increase of the connective-tissue elements. The substance is firmer than normal, the color varying from an olive-green to a deep bronze. If, however, the obstruction be of long standing, the presence of the dilated intra-hepatic ducts and the increase of connective tissue cause secondary atrophy of the hepatic cells, with a diminution in the size of the organ.

Symptoms.—The symptoms vary greatly according to the cause of the stenosis, but in the main they are those of chronic obstruction of the duct—viz. paroxysmal pain in the region of the liver, referred to the right shoulder; jaundice of varying intensity, but gradually deepening after each attack; and gastric disturbance, with ague-like paroxysms (fever and sweating), the latter being most frequently met with in occlusion from gall-stones.

Diagnosis.—The pathognomonic symptoms determining the nature of the stenosis are very often wanting, and the diagnosis is rendered correspondingly difficult. On the other hand, stenosis or complete occlusion of the bile-passages calls for diagnosis principally on account of the special cause or causes of the given case.

When the condition is due to *lumbricoid* worms reflex symptoms usually appear, as pruritus of the nose and anus, grinding of the teeth during sleep, and convulsions.

In *carcinoma of the head of the pancreas or the pylorus* pressing on the ducts the growth may be detected by palpation, together with a recognition of other more or less characteristic features (*vide infra*, p. 915). and the rapid course of the disease.

Abdominal aneurysm may give rise to obstruction of the duct without being evidenced by physical signs. Usually, however, when the sacculation presses against the bile-duct, the throbbing in the epigastrium, the tumor (which can often be grasped), and the expansile pulsation on palpation will tend to establish the cause of the obstruction.

When due to *cancerous nodules in the liver* there is usually a history of primary carcinoma of the stomach, mammary gland, rectum, or of one of the pelvic viscera. Osler records a case in which jaundice (thought to have been catarrhal in origin) developed seven weeks previously. On careful examination "a small nodule was detected at the umbilicus, which on removal proved to be scirrhus."

When the stenosis is due to *ulceration* following the passage of gall-stones, the history of biliary colic and of the presence of calculi in the dejecta, and the paroxysmal pain with jaundice and intermittent fever, will serve to establish the cause.

If the fever be of the continued type and the liver uniformly enlarged, with the development of jaundice, the case is probably one of *hypertrophic cirrhosis;* whereas if the enlargement be progressive and nodules can be detected on palpation in addition to the appearance of cachexia and jaundice, *carcinoma* is undoubtedly present.

Physical signs aid but little in the diagnosis, as obstruction of the common duct is usually unattended by any great enlargement of the gall-bladder.

In many cases only by remembering the various causes and eliminating them carefully, one by one, can the diagnosis be positively made.

Prognosis.—It may be said of the prognosis, as of the symptoms, that both vary according to the cause of the stenosis. Generally speaking, the outlook is rather grave, since many of the causative conditions are fatal. If the obstruction is due to cicatricial contraction, the prognosis is guardedly favorable as to life, but hopeless as to recovery. If the obstruction is permanent, the case ends fatally.

Treatment.—The treatment of occlusion of the bile-ducts varies according as it is due to cicatricial contraction following ulceration or to foreign bodies (seeds or lumbricoid worms), or to gall-stones or tumors pressing upon or involving the ducts or adjacent organs (pancreas, pylorus). If the stenosis follows ulceration in the duct, and is sufficient to cause almost complete occlusion with biliary retention, the operation of cholecystenterostomy may become necessary in order to prevent dilatation of the gall-bladder with resorption of bile.

Foreign bodies in the duct may be removed by free purging, aided by the liberal use of alkaline mineral waters. In critical cases the operation of cholecystotomy has been practised.

Gall-stones form the most frequent cause of stenosis, and the treatment, both for the prevention and removal of calculi, has already been described in the discussion of Biliary Calculi (*vide* p. 863).

ICTERUS NEONATORUM.

Definition.—Jaundice occurring in the new-born. This may be either *pathologic* or *physiologic*—a slight tinting of the skin occurring quite commonly in the new-born.

Pathology.—The morbid anatomy of the *pathologic* form varies with the cause of the jaundice. The secretion of bile, like the secretion of urine, begins long before birth, and Zweifel has found bile-pigment and bile-acids in the contents of the intestines of a three-months' fetus. Hence children may be born laboring under an attack of well-marked jaundice.

In well-marked cases of pathologic jaundice the skin presents a deep greenish-yellow hue. The internal tissues are also stained. Knopfermacher has studied the condition of the blood, and found that the red cells presented no signs of destruction, but rather of active new-formation.

Etiology.—Of the *physiologic forms*, the following are the main causes: 1. The *ductus venosus* may remain patulous, allowing some of the portal blood, containing bile, to flow into the systemic circulation (Quincke). 2. Diminished pressure in the portal vessels from ligation of the umbilical vein causes increased tension in the hepatic capillaries and absorption of bile.

Pathologic Icterus.—The causes are the following: (*a*) Congenital stricture or absence of the duct; (*b*) Syphilitic disease of the liver; (*c*) Septic processes set up by infection through the umbilical vein.

Symptoms.—In *physiologic* jaundice the skin is tinted greenish-yellow, resembling somewhat that of chlorosis. The mucous membranes are pale and the conjunctivæ pearly-white. The pulse is feeble and sometimes rapid. Auscultation over the base of the heart often reveals a soft systolic murmur transmitted to the vessels of the neck and associated with a venous hum. According to Murchison, false or physiologic jaundice differs from the true or pathologic form in that—1. The conjunctivæ are of a natural color; 2. The urine is free from bile-pigment; 3. The yellow color gradually fades from the skin after a few days; 4. The child is quite well and the bowels are acting properly.

In *pathologic* jaundice the skin and conjunctivæ are more or less intensely icteroid, the urine is loaded with bile-pigment, while the feces are of the pipe-clay variety. Hemorrhage from the cord may occur and destruction of life may be rapidly accomplished, or the condition may last for some weeks without serious impairment of the general health, with final recovery.

Treatment.—In the milder cases calomel in minute doses, combined with lactopeptin and sodium bicarbonate, can be recommended. In malignant cases treatment is of no avail.

VASCULAR (CIRCULATORY) AFFECTIONS OF THE LIVER.

ANEMIA.

THE physical symptoms of this condition are absolutely *nil*, and its existence only discoverable *postmortem*. Its most common causes are those of general anemia, fatty and amyloid degeneration.

HYPEREMIA.

Definition.—An excess of blood in the liver. This may be of two varieties: (*a*) *active* and (*b*) *passive*, the latter being the more common.

ACUTE HYPEREMIA.

(*Active Congestion.*)

Definition.—An excess of arterial blood in the liver.

Etiology.—Among the common causes are rich living, sedentary habits, alcoholism, traumatism, acute infectious diseases (typhus, typhoid), and pernicious malaria. The condition may also be vicarious, due to a sudden cessation of menstruation or of hemorrhage in other parts of the body. A *physiologic* condition is the temporary hyperemia that occurs during the ingestion of a full meal.

Symptoms.—There are no symptoms characteristic of this condition; those present in the different cases are varied and referable to disturbances of other viscera, as in coexisting cardiac hypertrophy or gastrointestinal catarrh. Often, however, there is a sense of *fulness* and *distress* in the right hypochondrium after eating, with *tenderness* on palpation over the lower margin of the organ.

Prognosis and Course.—It is impossible to make any definite statement as to the course and prognosis of active hyperemia, these depending wholly upon the cause of the affection. When due to errors of diet and hygiene the condition is easily remedied; the prognosis of hyperemia accompanying hepatic cirrhosis, however, is decidedly grave.

PASSIVE HYPEREMIA.

(*Passive Congestion.*)

Definition.—An increase of venous blood in the liver.

Pathology.—The organ is enlarged in size and changed to a deep-red color, its substance being firmer than normal. The center of the lobule (the area of the hepatic vein) becomes deeply pigmented, the periphery (occupied by the portal vein) being lighter in color, sometimes owing to fatty infiltration. Because of its mottled appearance this has received the name of the "nutmeg liver."

In long-standing passive congestion there is an increase of connective tissue, due to a proliferation of round-cells, causing atrophy of the parenchyma. The blood in the central capillaries becomes altered, the capillaries themselves are distended, and brown pigment is deposited about the center of the lobules. The organ becomes very much darker in color, and to this condition the name "cyanotic induration" or "cardiac liver" has been given. Later, contraction of the connective tissue occurs, causing a diminution in the size of the organ, and forming the so-called "atrophic nutmeg liver."

Etiology.—The causes that lead to passive hyperemia are both *local* and *general*. Among *local* causes may be mentioned the following:

1. Pressure over the portal area from without, as from a tumor or cyst.
2. Disease of the walls of the veins, as in syphilitic phlebitis.
3. Coagulation of the blood in the veins (thrombosis).

Among the *general* causes are—

1. Chronic valvular disease affecting the right side. Passive hyperemia is also common in mitral disease.
2. Pulmonary emphysema and cirrhosis of the lung.
3. Intrathoracic tumors, which by their mechanical action cause an increased pressure in the efferent branches of the hepatic veins.

Symptoms.—Often the patient experiences a sensation of *fulness* and *weight* in the region of the liver that amounts in some instances to actual *pain*. *Jaundice* is usually present, but varies in intensity, and is due to obstruction of the smaller ducts by the distention of the hepatic venules. *Hematemesis* is not rare, and symptoms of gastro-intestinal disturbance are usually present. In marked cases the *stools* are *clay-colored*, showing the absence of bile; the *urine* is loaded with bile-pigment; and jaundice deepens with the development of *ascites* or *anasarca* from portal obstruction. On *palpation* the organ is tender and increased in size, extending in some instances fully a hand's breadth below the costal margin. In marked cases the whole organ pulsates, owing to the regurgitation of blood into the hepatic veins (see also p. 623).

Diagnosis.—The diagnosis of passive congestion, *per se*, is often very difficult, but when secondary to heart- and lung-diseases it is rendered more plain.

The **prognosis** and **treatment** depend wholly upon the causal factors.

DISEASES OF THE PORTAL VEIN.

THROMBOSIS AND EMBOLISM.

Pathology.—In the early stages the clot presents a grayish-red or yellowish appearance, and on loosening it is found to adhere more or less closely to the inner coat of the vein. Later it becomes a mass of small white fibrin tightly adherent to the sides of the blood-vessel, which itself undergoes fibroid change, giving rise to the so-called adhesive pylephlebitis. Organized thrombi are rarely found, except in the smaller branches of the portal area. If the thrombus obstruct the vessel, collateral circulation may be established for years, as in a case recorded by Osler. Septic softening, however, is a very common result, and most frequent of all is pylephlebitis. If a parietal or channelled thrombus be formed, partial or complete circulation may be re-established and recovery take place. Hemorrhagic infarction may take place, but is very rare.

Etiology.—*Thrombi* are rare occurrences in the portal vein. Among the *causes* that lead to their occurrence, however, may be mentioned—(*a*) Traumatism; (*b*) cirrhosis; (*c*) carcinoma of the liver, involving the portal area; (*d*) pressure from without, as in proliferative peritonitis involving the gastro-hepatic omentum, abscesses, enlarged glands, or impacted calculi pressing on the veins; (*e*) it may be occasioned by ulcerative affections of the bowels and appendicitis, and pylephlebitis may precede its occurrence; (*f*) slowing of the circulation due to splenic diseases, such as marasmus.

Symptoms.—Symptoms may be almost lacking in portal obstruction, or the condition may simulate cirrhosis of the liver. In ordinary cases the symptoms are very slight, the hepatic circulation, as shown by Cohnheim and Litton, being "sufficient for the nourishment of the liver and secretion of the bile" (Henry).

If the occlusion be *complete*, *edema* followed by the rapid development of *ascites* may occur. In such cases loss of strength is persistent and progressive, and death may result from exhaustion. *Hemorrhages* due to venous stasis may occur from the nose, stomach, and intestines. *Jaundice* and *diarrhea* occur frequently, the former being the result of obstruction to the biliary passages from the same causes that produce the thrombosis or the diminished pressure in the portal area. On *palpation* the liver is found slightly enlarged and tender on pressure, and projecting below the lower margin of the ribs; the *spleen* is also enlarged. *Percussion* also reveals enlargement over the splenic area. If ascites is present, percussion will reveal dulness in the flanks, changing with the position of the patient; and on gently tapping one side of the belly-wall, with the hand on the opposite side, a wave of fluctuation will be felt.

Diagnosis.—The diagnosis of portal thrombosis is often extremely difficult. "A suggestive symptom, however, is sudden onset of the most intense engorgement of the branches of the portal system" (Osler).

Sequelæ.—If the emboli are septic in origin, an abscess, with all its accompanying symptoms, will be the result. Hemorrhagic infarction may occur, but is very rare, since a free anastomosis exists between the lobular plexuses and the hepatic artery.

"Pylethrombosis may be regarded as probable if no other possible cause of the portal obstruction seems likely, and if we are able to discover a cause for thrombosis, like a former attack of circumscribed peritonitis" (Strümpell).

The **prognosis** is always unfavorable, although certain cases have been demonstrated by autopsy to have improved temporarily.

Course and Duration.—Nothing definite can be stated in regard to the course and duration of this affection, since these depend entirely upon the cause.

Treatment.—The symptoms resulting from portal congestion, due to thrombi in the portal vein, are those described under Cirrhosis of the Liver, and the treatment is identical with that of interstitial hepatitis. In rare instances septic emboli give rise to abscesses that are usually multiple; when these occur the treatment is purely symptomatic.

SUPPURATIVE PYLEPHLEBITIS.

Definition.—A purulent inflammation of the portal vein or its branches.

Pathology.—If noted in the early stages, the coats of the portal vein are distended and thickened, and the connective tissue surrounding the portal area is infiltrated and the seat of minute ecchymoses. The inflammation usually originates in the smaller veins of the portal system or in the hepatic branches of the vein itself; the main trunk is attacked least often. Numerous thrombi are found obstructing the vein and its branches, which finally undergo suppuration. From these, emboli enter the circulation and are carried to all parts of the liver, forming metastatic abscesses: In advanced cases the whole organ (especially the peripheral parts) becomes infiltrated with pockets of pus, that communicate with the portal vein or its branches, and extend in some instances into the mesenteric or gastric veins. A single large abscess may be present, but multiple abscesses are the rule. The contents may be very fetid and bile-stained, or, as in many instances, they may be composed of thick, creamy laudable pus. From this focus of suppuration embolic abscesses may extend to the lungs, brain, kidneys, and joints.

The macroscopic appearance, with the organ *in situ*, is sometimes practically normal. The liver may present a uniform enlargement, the surface being of normal color and the capsule non-adherent. More commonly, however, the cortex presents a mottled appearance, and numerous yellowish-white spots are seen beneath the capsule.

Etiology.—The most frequent source of purulent pylephlebitis is appendicitis with abscess. Rarely the disease arises idiopathically.

Among other causes are the following: (*a*) A secondary (becoming a general) pyemia. (*b*) Ulceration of the intestines, occurring in dysentery and, more rarely, in typhoid fever. (*c*) Gastric ulcer. (*d*) Pelvic abscess; abscess of the spleen. (*e*) Specific infection through the umbilicus, occurring in the new-born.

Symptoms.—The symptoms vary according as to whether the case remains one of suppurative pylephlebitis or terminates in hepatic abscess. If the condition is part of a general *pyemia*, the symptoms referable to the liver may be almost negative. The *liver* is usually enlarged, and tender on pressure, the enlargement being most marked when an hepatic abscess exists. Though *pain* is present, in many cases it is not a marked feature; it is frequently referred to the epigastrium, and may radiate laterally or downward. *Percussion* in the left axillary line shows splenic enlargement, and the organ can in some instances be felt below the costal margin, constituting the "*acute splenic tumor*" of septicopyemia.

The *fever* is of an irregular septic type, the elevation in temperature is accompanied by rigors or chills and followed by profuse sweating. *Jaundice* of varying intensity is present, although usually it is not pronounced, the complexion being merely doughy or muddy. *Diarrhea* is not an infrequent symptom of this condition. Nausea and vomiting are often marked. As the case advances the pulse becomes rapid and small, and delirium develops, followed by stupor, coma, and death.

Duration and Prognosis.—The duration of suppurative pylephlebitis is usually from one to three or four weeks or longer. The prognosis is absolutely fatal.

Diagnosis.—The diagnosis of suppurative pylephlebitis is sometimes extremely difficult, unless the case is complicated by hepatic abscess, as enlargement of the liver is not constant in the former condition. The etiology, septic temperature, enlargement of the spleen, jaundice, and pain in the region of the liver would all, however, point to this affection.

The *differential diagnosis* of hepatic abscess will be spoken of later.

Treatment.—Unfortunately, the treatment of suppurative pylephlebitis can only be palliative. Surgical measures are rarely curative, unless the abscess is single and localized and shows signs of pointing. The circulation is to be supported by free stimulation. The leading symptoms should be met as they arise.

STENOSIS.

Obstruction of the portal vein may be due, as before mentioned, to (*a*) thrombosis; (*b*) cicatricial contraction from cirrhosis or syphilis of the liver; and (*c*) tumors pressing on the portal area. The first cause is the more common, chiefly because mechanical obstruction, by causing a stasis of the blood-current, induces the formation of a thrombus.

The **symptoms** of portal stenosis may be *nil;* if the stenosis occurs slowly, the hepatic artery furnishes sufficient blood to carry on the functions of the liver, the compensatory circulation being established by means of the systemic vessels. If due to thrombosis, the symptoms of portal engorgement appear suddenly with the development of edema and ascites. The liver is rarely enlarged in this condition.

Prognosis.—This depends wholly upon the cause of the affection. Thrombi in the portal vein often give rise to a suppurative pylephlebitis, terminating in hepatic abscess; tumors are rarely accessible; whereas fibroid conditions of the liver causing cicatricial contraction are incurable. As a rule, the prognosis may be said to be guardedly unfavorable.

AFFECTIONS OF THE HEPATIC BLOOD-VESSELS.

OSLER records a case of *stenosis of the hepatic veins* that was associated with fibroid obliteration of the inferior vena cava, with a greatly enlarged and cirrhotic liver.

Among other affections of the hepatic veins are (*a*) Emboli, originating from a thrombus in the right auricle, and (*b*) Dilatation, from stasis of the blood-current flowing to the right heart, due to enlargement of the latter.

Affections of the *hepatic arteries* are exceedingly rare, but may occur in one of the following forms: (*a*) *Aneurysm.*—Only 10 or 12 cases of aneurysm have been reported. (*b*) *Hypertrophy and Dilatation.*—These may occur in connection with general hepatic cirrhosis, the cicatricial bands obstructing the lumen of the artery, and causing thickening in some places, and ampullæ, or sac-like dilatations, in others. (*c*) *Sclerosis.*—This may form a part of a general arterio-sclerosis, though it occurs oftener in connection with cirrhosis or syphilitic hepatitis.

ATROPHY AND HYPERTROPHY OF THE LIVER.

(*a*) *Atrophy.*—Simple atrophy of the liver may result from pressure (corset-liver), syphilis, advanced cirrhosis, senility, and from the toxic action of phosphorus, arsenic, or chloroform—all factors that induce rapid fatty degeneration with cell-destruction.

(*b*) *Hypertrophy* is of two kinds—(1) *true* and (2) *false*. (1) *True hypertrophy* may be subdivided into *simple* and *numerical* (hyperplasia), the latter referring to an increase in the number of the parenchymatous cells, and not, necessarily, implying an increase in the size of the organ.

The two causes of simple hypertrophy are active and passive congestion. Among the causes of numerical hypertrophy may be mentioned the following: Leukemia, hypertrophic cirrhosis, atrophic cirrhosis (hyperplasia), syphilis, diabetes, and malaria.

(2) *Pseudo-* or *false hypertrophy* occurs in amyloid and fatty infiltration, carcinoma, and abscess, and consists in an increase in the tissues least concerned in the function of the organ.

HEPATIC INFILTRATIONS AND DEGENERATIONS.

AMYLOID INFILTRATION.

(*Waxy, Lardaceous, Bacony, or Albuminoid Infiltration; Amyloid Disease.*)

Definition.—A deposit in the hepatic connective tissues of a peculiar substance having some of the reactions of, and resembling, starch. A physiologic example of amyloid infiltration may be found in the *corpora amylaceæ* of the prostate gland, in which there is a concentric arrangement somewhat resembling a starch-granule.

Pathology.—The organ is larger than normally and of firmer consistence. The edges are rounded and not well defined, and the surface is of a light color, presenting in some instances a mottled appearance. On section the surface presents a grayish-brown, glistening appearance, which when scraped fails to exude oil-droplets, as in the fatty liver.

On microscopic examination the connective-tissue trabeculæ and the intima and media of the capillary walls (the starting-points) are chiefly affected, the lumen of the latter being lessened; this decreases the blood-supply to the liver, and often directly induces fatty degeneration. The hepatic cells may be atrophied and show evidences of fatty change. Amyloid material is structureless, and appears in small cloudy masses under the microscope. Chemically, it contains small amounts of potassium and phosphorus and an excess of sodium and chlorin.

Etiology.—Amyloid infiltration may occur primarily in the liver, but it is often a part of a general infiltration, affecting especially the spleen (*sago spleen*) and kidneys. It is also found in some syphilitic scars and in certain tumors and old thrombi.

Dickinson believes that the deposition of amyloid material is due to a decrease in the alkalinity of the fluid of the body, the pus (in cases of long suppuration) having removed a large quantity of the natural potassium salts. In malarial cachexia, however, such losses could not have occurred.

It is a frequent sequel to long-standing and exhausting suppurating and cachectic affections, as necrosis of the bones, hip-joint disease, and pyelitis; "especially is this the case when they occur in an hereditary tuberculous or syphilitic constitution" (Harley). Amyloid disease may also complicate chronic malaria. In children tuberculosis and rachitis not uncommonly contribute to amyloid infiltration.

Tests and Characteristics of Amyloid Material.—Although of animal origin, amyloid matter is closely related to a vegetable albuminoid starch. It is, however, slow to decompose, and is not acted on by weak acids and alkalies, whereas strong alkalies dissolve it. Iodin gives a blue color upon the addition of sulphuric acid. Lugol's solution (the aqueous solution of iodin and potassium iodid) gives a brown tint to amyloid liver-substance and stains ordinary hepatic tissues a yellow color. Gentian-violet gives a reddish or pinkish hue to amyloid substance, while normal tissue is stained blue.

The following is taken from Harley's *Comparative Table of Amyloid Tests:*

	STARCH.	AMYLOID.	CHOLESTERIN.
Water.	Dissolves on boiling.	Dissolves on boiling.	Unchanged.
Ether.	Insoluble.	Insoluble.	Dissolves.
Heat.	Dries up.	Dries up.	Melts.
Sulphuric acid.	Chars.	Swells up, reddish-brown.	Becomes green, blue, etc.
Iodin.	Becomes blue.	Blue color with H_2SO_4, which is destroyed by excess.	Remains unchanged.
Sulphate of indigo.	. . . .	Amyloid tissue soaked in it becomes a brilliant blue, while with ordinary liver-tissues the blue fades to a pale green.	

Symptoms.—When amyloid disease occurs in *children* the subjects are poorly developed and puny, the complexion is, as a rule, muddy or sallow, and the abdomen usually prominent. Occasionally the *skin* is exceedingly transparent. At any age *gastro-intestinal symptoms* occur, prominent among which are marked constipation and a capricious appetite. *Mental phenomena*, as impairment of memory and inability to concentrate, are not unusual in this disease. *Pain* about the hepatic region is a rare symptom. The *spleen* is usually enlarged from coexistent amyloid infiltration. The *urine* often contains albumin (globulin is nearly always present) and waxy tube-casts; it is of high specific gravity, and is usually scanty and dark colored. The *physical signs* show an increase in the area of hepatic dulness; the edges of the organ extend below the costal margin and have a rounded outline. Sometimes, however, the edge, even in a very great enlargement, is sharp and large. Wilks speaks of an amyloid liver weighing 14 lbs.—6.35 kgms. (Osler). In rare instances the liver is reduced in size.

Diagnosis.—The foregoing symptoms and physical signs, in conjunction with an ordinarily clear etiology, are sufficient to establish the diagnosis.

Treatment.—As amyloid disease is almost invariably a secondary condition, the treatment must be directed to the removal of the primary cause, whether syphilis, tuberculosis, or rickets. The *diet* should consist of nitrogenous or animal substances, with a minimum amount of fatty or farinaceous foods. French rolls and bran- or gluten-bread are allowable, together with lean meat and green vegetables. Stimulants are to be strictly avoided. Moderate exercise, with the judicious use of Turkish (hot-air) and Russian (hot-vapor) baths, is also of great value.

Many drugs are mentioned in the treatment of this disease, among the more important being the ammonium salts (the chlorid, gr. v to x—0.324 to 0.648—three or four times a day), and other alkalies, together with tonics and laxatives.

When syphilis has been clearly established as an etiologic factor of the disease, the tincture of iodin in 10- to 15-minim (0.666–0.999) doses, well diluted, has been recommended to be given three or four times daily. Cod-liver oil as a nutritive has been tried with good effect. Of tonics, the dilute mineral acids, given in moderate doses over a long period of time, have probably achieved the best results.

FATTY INFILTRATION.

Definition.—A deposit of fat in the hepatic tissues due to the ingestion of fats and albuminates.

Pathology.—The infiltration occurs often in localized areas, and may be so intense that the organ when cut presents a shiny, oily appearance. The liver is often evenly enlarged, and may weigh twelve to fifteen pounds. The edges are rounded and the substance less firm than normally. Portions of the liver-substance float in water, being of low specific gravity. The color is light-yellow or grayish. On microscopic examination the protoplasm of the cell is seen to be pushed to one side by the fat-droplets, which tend to coalesce. When the fat is removed the cells resume their normal outline and appearance.

Etiology.—(*a*) Fatty infiltration may form part of a general obesity or it may follow excessive over-eating or sedentary habits. (*b*) It often occurs in wasting diseases, as carcinoma, syphilis, chronic malaria, and tuberculosis, and often accompanies fatty degeneration.

Symptoms.—The subjective symptoms of fatty infiltration may be entirely wanting, since the function of the liver is not impaired to any extent. When they are present progressive *anemia* and *debility* are noted, and are accompanied by *nervous irritability* and *insomnia.* In marked cases the cardiac rhythm is disturbed, causing a *feeble* and *irregular impulse.*

The *physical signs* are usually well defined, and the area of hepatic dulness is uniformly increased, extending in some instances as low as the umbilicus. The enlargement, however, is not so great as in amyloid disease.

Differential Diagnosis.—Fatty infiltration of the liver is not apt to be mistaken for any other affection of this organ. The occurrence of general obesity, together with an entire absence of symptoms of obstruction to the portal vessels or bile-ducts or of other evidences of *fatty degeneration* (particularly feeble heart-sounds), will help to distinguish it from this latter condition. The etiologic factors above mentioned will also aid in the differentiation.

Prognosis.—This is decidedly favorable, as the function of the liver in many instances is not impaired in the slightest degree.

Treatment.—As the disease is of gradual development and long duration, a modification of the *diet* constitutes the first essential of the treatment. That prescribed under the Treatment of Amyloid Liver is admirably suited to this affection. Saccharine and farinaceous articles of food (potatoes, oatmeal, and sweetmeats) must be eschewed. Wheat-bread must be partaken of sparingly, and in its place gluten- and bran-bread or crusts of French rolls should be used. Fish, lean meats, fresh vegetables, and fruits are also allowable. Alcoholic beverages must be interdicted.

Graduated daily exercise and Turkish or Russian baths, judiciously used, are important factors in the treatment. Medicinally, the salts of the alkalies are highly recommended: sodium sulphate (in dram—4.0—doses, taken on an empty stomach) and ammonium carbonate (gr. xv to xxx—1.0 to 2.0—in twenty-four hours).

FATTY DEGENERATION OF THE LIVER.

Definition.—A conversion of the albuminates of the cells into fat; it is characterized anatomically by a destruction of the liver-substance, with atrophy of the organ, and clinically by biliary, gastro-intestinal, cardiac, and renal symptoms.

Pathology.—On examining a liver that is the seat of marked fatty degeneration the organ is found smaller than normally, and the substance is light yellow in color, soft, pliable, and easily torn. On section the relation between the interlobular connective tissue and the acini is lost, the latter being replaced by fat-cells and oil-droplets. Scattered areas of pigmentation may be observed throughout the organ.

Microscopically, the cells lose their shape and become globular; the nuclei tend to coalesce, and finally disappear, together with the cell-wall, giving rise to compound globule-cells, which do not tend to coalesce and are stained black by osmic acid. Crystals, granular débris, Lener's spheres, cholesterin, tyrosin, and phosphatic crystals are also found in this form of granular change.

Etiology.—The following are among the recognized causes of the affection: (*a*) The excessive use of beer or alcoholic liquors. (*b*) It may be a sequence of amyloid disease, and hence result from any of the causes of the latter. (*c*) Diminution of the oxygen-supply to the tissues, occurring in phosphorus-, chloroform-, or arsenic-poisoning and in certain wasting diseases (carcinoma, phthisis, and chronic dysentery). (*d*) It may occur as a complication in the grave anemias, especially pernicious anemia, and in acute infectious diseases and the intoxications; also as a part of the pathology of acute yellow atrophy of the liver.

Symptoms.—I feel convinced that partial or *mild cases* of fatty degeneration of the liver present no morbid symptoms of diagnostic import. Pain, jaundice, and ascites may occur separately or conjointly, but form the exception rather than the rule. The *severe forms* are characterized ndition, the treatment must be directed to the removal of the primary ause, whether syphilis, tuberculosis, or rickets. The *diet* should consist f nitrogenous or animal substances, with a minimum amount of fatty or arinaceous foods. French rolls and bran- or gluten-bread are allowable, ogether with lean meat and green vegetables. Stimulants are to be strictly avoided. Moderate exercise, with the judicious use of Turkish (hot-air) and Russian (hot-vapor) baths, is also of great value.

Many drugs are mentioned in the treatment of this disease, among the more important being the ammonium salts (the chlorid, gr. v to x—0.324 to 0.648—three or four times a day), and other alkalies, together with tonics and laxatives.

When syphilis has been clearly established as an etiologic factor of the disease, the tincture of iodin in 10- to 15-minim (0.666–0.999) doses, well diluted, has been recommended to be given three or four times daily. Cod-liver oil as a nutritive has been tried with good effect. Of tonics, the dilute mineral acids, given in moderate doses over a long period of time, have probably achieved the best results.

FATTY INFILTRATION.

Definition.—A deposit of fat in the hepatic tissues due to the ingestion of fats and albuminates.

Pathology.—The infiltration occurs often in localized areas, and may be so intense that the organ when cut presents a shiny, oily appearance. The liver is often evenly enlarged, and may weigh twelve to fifteen pounds. The edges are rounded and the substance less firm than normally. Portions of the liver-substance float in water, being of low specific gravity. The color is light-yellow or grayish. On microscopic examination the protoplasm of the cell is seen to be pushed to one side by the fat-droplets, which tend to coalesce. When the fat is removed the cells resume their normal outline and appearance.

Etiology.—(*a*) Fatty infiltration may form part of a general obesity or it may follow excessive over-eating or sedentary habits. (*b*) It often occurs in wasting diseases, as carcinoma, syphilis, chronic malaria, and tuberculosis, and often accompanies fatty degeneration.

Symptoms.—The subjective symptoms of fatty infiltration may be entirely wanting, since the function of the liver is not impaired to any extent. When they are present progressive *anemia* and *debility* are noted, and are accompanied by *nervous irritability* and *insomnia*. In marked cases the cardiac rhythm is disturbed, causing a *feeble* and *irregular impulse*.

The *physical signs* are usually well defined, and the area of hepatic dulness is uniformly increased, extending in some instances as low as the umbilicus. The enlargement, however, is not so great as in amyloid disease.

Differential Diagnosis.—Fatty infiltration of the liver is not apt to be mistaken for any other affection of this organ. The occurrence of general obesity, together with an entire absence of symptoms of obstruction to the portal vessels or bile-ducts or of other evidences of *fatty degeneration* (particularly feeble heart-sounds), will help to distinguish it from this latter condition. The etiologic factors above mentioned will also aid in the differentiation.

Prognosis.—This is decidedly favorable, as the function of the liver in many instances is not impaired in the slightest degree.

Treatment.—As the disease is of gradual development and long duration, a modification of the *diet* constitutes the first essential of the treatment. That prescribed under the Treatment of Amyloid Liver is admirably suited to this affection. Saccharine and farinaceous articles of food (potatoes, oatmeal, and sweetmeats) must be eschewed. Wheat-bread must be partaken of sparingly, and in its place gluten- and bran-bread or crusts of French rolls should be used. Fish, lean meats, fresh vegetables, and fruits are also allowable. Alcoholic beverages must be interdicted.

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Symptoms.—I feel convinced that partial or *mild cases* of fatty degeneration of the liver present no morbid symptoms of diagnostic import. Pain, jaundice, and ascites may occur separately or conjointly, but form the exception rather than the rule. The *severe forms* are characterized by the symptoms seen in phosphorus-poisoning and acute yellow atrophy, to the discussion of which the reader is referred.

Complications.—The disease may be complicated with fatty change in the *kidneys*. Under these circumstances the *urine* is diminished in amount, of low specific gravity, and contains an abundance of albumin, fatty or oily casts, and crystals of cholesterin, leucin, and tyrosin. In marked cases there is a very *feeble* and *irregular cardiac impulse*, accompanied by attacks of *vertigo* and *syncope*, the latter symptoms indicating beginning degeneration of the cardiac muscle. *Edema* of the lower extremities and *anasarca* may occur as complications of this condition.

The **physical signs** elicited by *palpation* and *percussion* show increasing diminution in the size of the liver as the disease advances.

Diagnosis.—The chief diagnostic points of fatty degeneration may be summated thus: (*a*) A history of alcoholism, of poisoning by drugs (arsenic, phosphorus, or chloroform), or of an acute infectious disease (acute yellow atrophy); (*b*) Grave general symptoms, as albuminuria, edema, ascites, cardiac failure, terminating often in acholia or cholemia; (*c*) Progressive diminution in the size of the organ. When these occur conjointly the diagnosis is established beyond a doubt.

Prognosis.—The prognosis is entirely dependent upon the cause. If due to an excessive use of stimulants, the process, if recognized early, may be arrested; if associated with acute yellow atrophy or other infectious disease, the outlook is unpromising.

Treatment.—The indications for treatment may be divided into the *dietetic*, *hygienic*, and *medicinal*. The same precautions regarding diet should be observed as in fatty infiltration. An open-air existence, short of injurious exposure, aided by hot salt-water, Turkish, or Russian baths, under restriction, is sure to improve the general condition of the patient.

The medicinal treatment varies according to the cause of the disease. If due to grave anemia, iron (tinct. ferri chlorid. or syrup. ferri. iodid.) may be given in ascending doses. Poisoning by drugs that produce fatty degeneration of the liver is to be combated by their respective antidotes. Gastro-intestinal disturbances, if coexistent, demand appropriate treatment. For the latter Frerichs recommends highly the salts of the alkalies (sodium sulphate in dram—4.0—doses taken on an empty stomach and ammonium carbonate). Ascites and cardiac asthenia, when occurring as complications, must be met by suitable measures.

PERIHEPATITIS.

ACUTE PERIHEPATITIS.

(*Pyo-pneumothorax Subphrenicus.*)

Definition.—An inflammation, either suppurative or fibrinous, of the peritoneal covering of the liver and the corresponding portion of the diaphragm.

Pathology.—The morbid changes may consist in a purely plastic inflammation, the serous layers being thickened, opaque, and covered with a fibrinous exudate leading to adhesion. In the majority of cases, however, the inflammatory product is chiefly purulent, and is ribboned by fibrous bands so as to form circumscribed areas, filled with pus, lying between the liver and the diaphragm; this constitutes the *subphrenic abscess*. The latter is found more commonly to the right than to the left of the suspensory ligament. It may contain much pus (1 quart—1 liter—or even more), which in most cases is mixed with air or gas derived from the gastro-intestinal canal. Rarely, bilirubin-crystals are found, betraying the presence of bile. If the latter be present in large amount, the pus assumes an ocher-yellow hue.

Etiology.—The *fibrinous variety* may result from the direct extension of one or other of the acute forms of inflammation of the liver (abscess, hydatid cyst), from a pleurisy spreading along the lymphatics in the diaphragm, or from traumatism—particularly a blow. The *suppurative form* (*pyo-pneumothorax subphrenicus*, Leyden) may be caused in the same manner as the former, but far oftener—in more than one-half of the instances—it follows perforation of a gastric ulcer, and far less commonly perforation of a duodenal or colonic ulcer. Appendicitis and penetrating wounds are not infrequent causes. Perihepatitis is a grave complicating event in carcinoma (of the stomach, esophagus, and intestines), in lobar pneumonia, and purulent pleuritis.

Symptoms.—Those of the *acute fibrinous variety* are either altogether missing or too vague to admit of correct interpretation. The coappearance, however, of severe *pain*, increased on deep breathing, and *tenderness* over a circumscribed area either in the right hypochondrium or the epigastrium, after the action of some known cause or the occurrence of one of the causative affections, is strongly suggestive of this form of the complaint. A *friction-sound* may at times be heard below the seventh rib in the mammillary and the ninth rib in the axillary line, or over the epigastrium, as in two cases in my practice. It is of short duration, and is limited usually to the end of inspiration. It must be recollected that plastic pleurisy may be an associated condition.

In *suppurative perihepatitis* the symptoms are sometimes screened by those characterizing the special causative complaint; but in my experience, in cases due to perforation—the most common cause—the *onset* is rapid and severe, and is marked by *acute pain* referred to a circumscribed spot in the hepatic region, great *tenderness*, rapid, embarrassed, and *painful respiration* (owing to implication of the diaphragm), by *vomiting* (often bilious, though at times hemorrhagic) or *nausea*, and by faint *jaundice* in some cases. Shortly the *general features* of circumscribed peritoneal abscess also appear—rigors, irregular fever, sweats, and progressive prostration and emaciation.

Physical Signs.—*Inspection* discloses bulging of the right hypochondrium and often of the epigastrium. The same regions are immobile, but this is best appreciated by *palpation*. The anterior edge of the liver is felt even as low as the umbilical level. *Percussion* reveals a variable increase of hepatic dulness upward, sometimes touching the fourth rib. The upper level of the fluid is movable on changing the position of the patient, and this is particularly striking if air or gas is contained in the abscess; the presence of the latter also causes a zone of tympanitic resonance above the dull area, while overlying the latter there is the semi-tympanitic area of the retracted lung. *Auscultation* reveals an absence of breath-sounds and of the vocal resonance over the dull and tympanitic areas, while the respiratory sounds over the displaced lung are broncho-vesicular.

Differential Diagnosis.—Acute perihepatitis often remains unrecognized during life. It may be confounded with *empyema* of the right side, but the two conditions have different modes of development. Perihepatitis is preceded and accompanied by abdominal symptoms; empyema manifests thoracic symptoms—*e. g.* cough and pleuritic pain. At a later stage the exaggerated respiratory murmur above the dull area, the slighter cardiac displacement toward the left, and the greater hepatic displacement downward in suppurative perihepatitis aid in the differentiation. The introduction of the trocar in the seventh or eighth intercostal space in the mid-axillary line may also be helpful, especially if the exudate be found to contain bile-pigment. *Pfuhl's sign*—the more ready escape of the fluid during inspiration on aspiration of abscesses below the diaphragm—may not be without value. The points narrated above may likewise serve to separate pyo-pneumothorax from suppurative perihepatitis (see also Pneumothorax, p. 572).

Course and Prognosis.—In the milder or fibrinous variety the outlook is favorable and the course is brief. On the other hand, the suppurative type due to perforation, if not early brought under proper surgical treatment, often terminates unfavorably by gradual asthenia. Rarely the pus is resorbed, or it may find an outlet through the lungs, abdominal walls, or other avenue, followed by slow recovery.

The **treatment** is the same as for localized peritonitis. The first evidence of the presence of pus is the signal for appropriate surgical interference—evacuation and drainage.

CHRONIC PERIHEPATITIS.

(*Zuckergussleber.*)

This affection is a chronic inflammation of the perihepatic fibrous membrane, which becomes opaque and thickened. Contraction of this capsule ensues, with compression of the liver and atrophy to even one-half the size of the normal organ (as in a case reported by Rumpf[1]), and partial or total occlusion of the vessel and bile-ducts. Perhaps these changes are most marked in cases that follow acute suppurative perihepatitis. Genuine instances show no hyperplasia of the interstitial connective tissue; hence the condition is closely related pathologically to "Glissonian cirrhosis" (*vide* p. 890).

The main **causes** of chronic perihepatitis are great and protracted local pressure, as from a corset, and certain occupations. It may represent a portion of a more general chronic inflammation of the serosæ. Finally, I am of the belief that syphilis is the leading single cause, and could discover no other factor present in two cases that yielded to antisyphilitic treatment.

The **diagnosis** is generally problematic. Of especial clinical worth are the etiology, pain in the right hypochondriac region—particularly in cases due to syphilis—absence of the signs of stasis of the gastro-intestinal tract, and the very protracted course.

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[1] *Deutsch. Arch. f. klin. Med.*, March 13, 1895.

The **treatment** is purely palliative, apart from the effort to remove the special cause, whether this be syphilis, occupation, or other influential factor.

ABSCESS OF THE LIVER.

(*Hepatic Abscess; Suppurative Hepatitis.*)

Definition.—A circumscribed collection of pus in the hepatic parenchyma.

Pathology.—If examined *in situ*, a liver that is the seat of abscess-formation is usually found to be symmetrically enlarged, and on careful palpation one or more areas of fluctuation (either deep or superficial, according to the location of the abscess) may be detected. If single, its position is usually in the right lobe near the convexity of the organ (70 per cent. of cases). The tissue surrounding the abscess-wall is usually deeply injected, the wall itself in acute cases being poorly defined, but grayish in color, irregular and shreddy, and composed of necrotic liver-cells, pus-corpuscles, and often amebæ. In chronic cases it becomes greatly thickened and often cartilaginous in appearance.

Microscopically, the hepatic cells are altered in shape and devoid of nuclei; they undergo rapid degeneration. A round-celled infiltration occurs about the blood-vessels, their walls being filled with small emboli containing innumerable staphylococci and streptococci. As the suppurative process continues liquefaction-necrosis occurs, resulting in complete destruction of the hepatic parenchyma.

The amount of fluid contained in a liver-abscess may exceed 2 or 3 quarts (2–3 liters), and its color varies from grayish-white to a creamy, reddish-brown. The collection in some instances resembles healthy pus. I have spoken of the methods of infection and of some of the different varieties of hepatic abscess in the discussion of Dysentery (see p. 98).

Various odors are described, depending largely on the extent of bacterial invasion and the degree of necrosis. In this connection it may be said that in amebic dysentery, with abscess of the liver as a complication, the abscess is often single (involving more often the right lobe), whereas other forms due to septic infection give rise to multiple abscesses.

In the latter instances the surface of the organ presents many small yellow areas beneath the capsule, varying from 5 to 15 mm. ($\frac{1}{5}$–$\frac{3}{5}$ inches) in diameter. Usually, in such cases infection has taken place through the portal circulation, and on section the appearances of a suppurative pylephlebitis present themselves. If thrombi have formed in the portal tributaries, localized necrotic areas are the result, but more often the invasion affects the whole portal system, the liver being riddled with abscesses. If the abscess is secondary to obstruction by gall-stones or inspissated bile, the ducts are greatly distended and the gall-bladder is filled with pus mixed with bile.

Etiology.—Idiopathic abscess of the liver is rare even in tropical climates. The affection, even when excited by mechanical causes, as traumatism or obstruction by gall-stones, is invariably septic in character, and the infecting material reaches the interior through the hepatic vessels or the biliary passages.

Septic emboli enter the liver by means of the vascular system through the hepatic artery or portal vein, the latter being the more common channel of transmission. Gastric ulcers, or typhoid fever, typhilitis (?), or appendicitis, may be followed by a purulent portal pylephlebitis, resulting in abscess-formation. On analyzing 500 cases of suppurative hepatitis Kelsch found that in 85 cases in 100 the disease was associated with dysentery! In general pyemic processes or in bone-suppurations of long standing infection occurs through the blood (hepatic artery). Suppurating wounds of the head are not uncommonly followed by abscess of the liver. Because of obstruction of the common duct by gallstones and resulting pressure-necrosis pathogenic organisms may enter the liver and cause abscess-formation through the medium of the bile-ducts. The most common method of infection, however, is *through the portal vein*. Among other causes may be mentioned *foreign bodies* travelling up the ducts, as parasites, round-worms, liver-flukes; also, more rarely, suppuro-perforation by mechanical irritants (needles, pins, fish-bones, and the like), and suppuration occurring in the course of an hydatid cyst. Leick has tabulated 19 cases of hepatic abscess caused by the ascaris lumbricoides.

Symptoms.—In a typical case of hepatic abscess the most prominent symptoms are—*hectic temperature*, *pain*, *tenderness*, and *enlargement of the organ*, and often *slight jaundice*, although it must not be forgotten that any or all of these may be absent during the development of an abscess. The multiple abscesses occurring in pyemic conditions, which are frequently diagnosed when in view upon the postmortem table, form an instance of this.

To facilitate the subject I shall consider the more important symptoms *seriatim: Pain* is circumscribed to the hepatic region, and radiates to the right shoulder in conjunction with the other symptoms and physical signs; it is very characteristic, although not pathognomonic of hepatic abscess. In the earlier stages this symptom is not pronounced unless the abscess or abscesses lie superficially. It is usually of a dull, boring character, differing in severity with the patient's position; it is usually aggravated by pressure over the costal margin and by lying on the left side, this tending to drag the liver by its own weight from its normal position. Luschka explains the radiation of pain to the right shoulder by stating that filaments of the phrenic nerves that distribute themselves in the suspensory ligament and Glisson's capsule are irritated. The phrenic arises from the third, fourth, and fifth cervical nerves, and, as the fourth supplies sensation to the right shoulder, the impression is thus transmitted through the central nervous system.

In acute cases accompanied by rapid destruction of the hepatic tissues the *temperature* usually rises rapidly, reaching 103° or 104° F. (39.4°–40° C.) in the course of from twenty-four to thirty-six hours. Its course, however, is irregular and intermittent, and it may be hectic in character; just as often it resembles a tertian or quartan intermittent or a remittent temperature. *Rigors* or *decided chills* frequently accompany the rise of temperature, and during the decline profuse sweatings may take place, thus simulating to a certain extent the symptoms of malarial fever. In chronic abscess of the liver pyrexia may be entirely absent. Less commonly the temperature may remain continuously high, with slight morning and evening exacerbations and remissions. The *pulse* is usually rapid in proportion to the temperature.

The **physical signs** in a case of hepatic abscess are always present to a greater or less degree, and are often pathognomonic.

Inspection may reveal nothing during the entire course of the disease, although in cases accompanied by intense congestion in which the abscess involves the anterior surface of the right lobe, bulging of the ribs on that side will occur, with a marked prominence in the hypochondriac region extending three or more finger-breadths below the costal margin.

Palpation confirms inspection and reveals tenderness on pressure below the costal margin in the mammary line. The liver, if projecting below the edge of the ribs, is usually enlarged uniformly, unless the abscess involves the surface of the margin. As the upper right lobe is more often involved, especially in a large single abscess, the increase in size is in an upward direction, thus rendering palpation negative. In rare instances the abscess gives rise to fluctuation on palpation, and if the peritoneum be inflamed a friction fremitus may be detected.

Percussion.—The area of hepatic dulness may be increased uniformly, but it is usually most marked upward and to the right, in some instances reaching at the anterior axillary line to the fifth rib, and posteriorly to the level of the angle of the scapula. This high position of the upper boundary of dulness which starts about the nipple-line serves to differentiate abscess from other affections of the liver, in which the enlargement extends in a downward direction.

Other Symptoms.—The *skin* is pale and shows slight icterus, the *conjunctivæ* being often bile-stained; intense *jaundice*, however, is rare. Progressive loss of flesh and strength, with *gastro-intestinal disturbance* (fulness in the epigastrium, flatulence, water-brash, nausea, and occasional vomiting), are common symptoms at the onset. The *bowels* are variable, and constipation usually alternates with diarrhea, the stools in some cases containing the ameba coli. *Ascites* may develop from pressure on the inferior vena cavæ, but these cases are rare. The *spleen* may undergo active hyperplasia in acute abscess-formation. *Pulmonary symptoms* (severe cough, characteristic reddish-brown sputum, resembling anchovy sauce, broncho-vesicular breathing, râles) are commonly present; they are due to compression of the base of the lung by the abscess pressing upon the diaphragm. In fatal cases certain *nervous symptoms* (muttering delirium, cephalalgia, subsultus tendinum, stupor, coma) make their appearance.

Complications and Sequelæ.—The abscess may perforate into the pleural cavity (pyothorax), bronchi, lungs, intestinal tract, stomach, pericardium, peritoneal cavity, or externally through the abdominal wall, giving rise to various symptoms. If rupture occurs into the intestinal tract, sudden diarrhea, with the discharge of large quantities of pus, takes place; there is then an amelioration of the pain, fever, and other symptoms. If the rupture is into the lung, the physical signs will reveal the sudden development of weak, tubular breathing over the base, with increased tactile fremitus and percussion-dulness, together with the occurrence of profuse and typical expectoration. Reese and Lafleur found the ameba coli in the bronchial discharge. S. Flexner has reported two cases of amebic abscess of the liver in which perforation into the vena cava occurred. Rupture into the abdominal cavity gives rise to the rapid development of a purulent peritonitis that is often fatal. Rarely, the abscess is emptied into the pericardium, giving rise to fatal acute pericarditis. Septic emboli have been known to lodge in the circle of Willis, producing fatal brain-abscess.

Diagnosis.—The clinical symptoms of hepatic abscess are of diagnostic importance only when taken in the aggregate, since the pain, fever, enlargement, and even hectic symptoms occur singly in other conditions unaccompanied by suppuration. The principal points in the establishment of the diagnosis of the affection may be summed up as follows: Residence in tropical countries, the previous existence of typhoid or dysenteric ulceration (or other gastro-intestinal inflammation), the characteristic expectoration, enlargement of the liver, with pain and tenderness on pressure, and in some instances fluctuation on palpation. Lastly, aspiration may reveal pus-corpuscles, hepatic cells, staphylococci and streptococci, the ameba, and bile-pigment, which when found are pathognomonic; if the abscess be secondary to an echinococcus cyst, the presence of hooklets will be detected.

Differential Diagnosis.—Hepatic abscess may be misdiagnosed for *empyema*, *malarial fever*, and *hepatic calculi*.

Empyema.—In empyema there may be the history of a perforating wound of the chest, the rupture of a bronchiectatic or tuberculous cavity, or the pre-existence of a sero-fibrinous pleurisy; whereas hepatic abscess may be preceded by an attack of amebic dysentery or intestinal ulceration, or it may follow the impaction of biliary calculi. In both there may be the occurrence of a hectic temperature, with chills and sweating; but in empyema cough and dyspnea are prominent, and, if the pleural cavity communicates with a bronchus, profuse muco-purulent expectoration containing pus-cells, staphylococci, streptococci, and in many cases elastic tissue and tubercle bacilli. Rarely, an abscess of the liver penetrates the diaphragm, and, entering the bronchi, is expectorated. The recognition of hepatic abscess under these circumstances is to be based mainly upon clear evidence of the affection prior to the occurrence of perforation, and copious, blood-tinted, purulent expectoration. The detection of the ameba coli in the sputum alone would set the diagnosis at rest. The contents of hepatic abscess obtained by aspiration consist of the micro-organisms of suppuration, and in addition broken-down liver-cells, bile-pigment, and in some cases the ameba coli. Inspection in empyema reveals bulging of the intercostal spaces on that side, while percussion gives absolute flatness over the base of the chest, rising posteriorly and changing with the change from a dorsal to a sitting position. In abscess of the liver the lung is slightly displaced upward, being often bound to the diaphragm by adhesions; and the upper boundary of dulness is lower, particularly in front, and is not changed with the decubitus of the patient.

HEPATIC ABSCESS.	MALARIA.
History of traumatism, dysentery, intestinal ulceration, or residence in tropical countries.	History of previous attacks. Residence in warm, damp climates among the lowlands.
Hectic character of the temperature—high every evening and low every morning; irregular chills, followed by fevers and sweatings.	Regularly recurrent rise of the temperature (intermittent or remittent, quotidian, tertian, quartan, or septinarian), and the rise occurring during the chill, followed by profuse sweating; chills more often in morning.
An irregular, fluctuating tumor or multiple nodules in the liver; no splenic enlargement; rapid emaciation, with or without jaundice, but no cachexia.	The spleen is enlarged; also there is a yellow-brown coloration of the skin, more or less marked; and, in long-standing cases, the occurrence of *cachexia*.
Blood shows simple anemia and leukocytosis, and in marked cases disintegration of red blood-cells.	The presence of the hematozoa of Laveran and free pigment in the blood.
Abscess-contents show the staphylococci, streptococci, amebæ, or bacillus coli communis, and pus.	Absent.

Impacted Calculi.—In this condition attacks of *hepatic colic* are often first noticed, followed by jaundice, and, if impaction be not absolute, by the occurrence of stones in the feces. In abscess the pain is not paroxysmal, but dull and boring in character, increasing in severity as the disease progresses. In *chronic impaction* jaundice, dull pain over the hepatic area, distention of the gall-bladder (which in some instances may be palpated), and clay-colored feces, constitute the principal symptoms. There occurs also an intermittent fever as in hepatic abscess, but it is occasional—*i. e.* the febrile paroxysms recur at longer intervals. Again, the course of intermittent hepatic fever associated with biliary calculi is much more chronic than the fever-stage of suppurative hepatitis. On the other hand, in abscess of the liver jaundice is comparatively rare, and, unless the abscess rupture into the gastro-intestinal tract, the stools show nothing abnormal. In some instances biliary abscesses may follow impacted calculi, and it is always a secondary affection.

Among other liver-conditions that are liable to be mistaken for hepatic abscess may be mentioned *carcinoma*, *hypertrophic cirrhosis*, and *hydatid cyst*, the differential diagnosis of which will be spoken of under these diseases.

Prognosis.—The prognosis of hepatic abscess is unfavorable, the disease generally progressing to a rapidly fatal termination. Prompt evacuation of the abscess when its location can be detected, however, may be successfully performed. The mortality ranges from 50 to 60 per cent. In rare cases the walls of the abscess become calcified and the disease remains latent. The single large abscess that most often follows dysentery offers the best opportunity for surgical measures.

Treatment.—Barring operation, the treatment of abscess of the liver is purely symptomatic, being in many instances identical with that of septico-pyemia. The temperature often responds to repeated spongings with cool water (65° F—18.3° C.). For the pain mustard-poultices, the turpentine stupe, or hot fomentations over the hepatic area, in conjunction with full internal doses of opium, prove beneficial. Full and free stimulation and the free exhibition of quinin as soon as the condition is detected both support the system and control, in a measure, the pyemic process. If the abscess be single and localized, prompt evacuation should be resorted to. Patients who have been thus cured should not return to a climate in which tropical dysentery occurs frequently, since as in a case reported by Marshall another attack of dysentery accompanied by abscess may ensue.

ACUTE YELLOW ATROPHY.

(Malignant Jaundice; Icterus Gravis.)

Definition.—An acute and probably infectious disease, characterized by a rapid destruction of the parenchyma of the liver and by a diminution in the size of the organ; also by jaundice, hemorrhage, and grave cerebral phenomena.

Pathology.—Macroscopically, in a case of acute yellow atrophy the liver is seen to be much reduced in size, weighing but 15 or 20 ounces (480.0–640.0), instead of its normal weight (50 oz.—1.6 kgms.). The capsule is shrivelled and the organ is of a pulpy consistence, and changed in appearance from a mahogany-brown to a grayish-yellow hue. Sometimes the liver is primarily enlarged. The cut section often presents areas of red and yellow discoloration, the so-called "red atrophy" and "yellow atrophy," the former being a later stage of the latter. The red appearance is due to an excess of blood in the capillaries, with free pigment that has been liberated by destruction of the red blood-cells. *Microscopic examination* reveals destruction or necrosis of the hepatic cells. The nuclei have disappeared, and the cell-wall contains a number of fat-globules of various sizes containing free pigment. In advanced cases, accompanied by total disintegration of the cells, fat-droplets, granular débris, cholesterin-plates, leucin-spheres, tyrosin-needles (first discovered by Frerichs, both in the cells and in the blood-vessels), and crystals of bilirubin may be found. Findlay[1] found the fibrous tissue to be increased and in the periphery of the lobules attempts at regeneration (proliferation of the hepatic cells). The common duct is patulous.

In well-marked cases both the *heart* and *kidneys* show evidences of fatty degeneration. The *spleen* is greatly enlarged from active congestion, giving rise to the so-called "acute splenic tumor." The splenic substance is soft and easily torn. The *skin* and *mucous membranes* may be the seat of numerous ecchymoses, and dropsy of the serous cavities is frequently noted. The *blood* is dark and fluid (disintegrated). Microscopically, it is seen to contain crystals of leucin and tyrosin.

Etiology.—The causes of acute yellow atrophy are both primary and secondary. *Primary* or idiopathic acute yellow atrophy is rare and its course as yet unsettled. Among the *secondary* predisposing causes may be mentioned age (fifteen to thirty-five years), female sex, parturition, syphilis, and certain acute fevers (puerperal fever, typhoid, septicemia, malaria). Acute phosphorus-poisoning sometimes presents changes resembling those of acute yellow atrophy. The disease rarely accompanies cirrhosis of the liver, and may follow a debauch. Rarely, an endemic form is assumed, but the exciting cause is thus far unknown. The disease is probably microörganismal or toxic in nature, and although various germs have been discovered, their claim to specificity has not been established.

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[1] *British Medical Journal,* June 2, 1900.

Symptoms.—The clinical history of acute yellow atrophy varies considerably in the early stages of the disease, the graver symptoms of the later stage alone being pathognomonic. The attack is usually ushered in by *headache*, *malaise*, *anorexia*, *nausea*, and *vomiting*, moderate *fever*, and after a few days *jaundice* appears. *Physical examination* at this time shows the area of hepatic dulness to be normal or only slightly increased. After a period varying from a few days to two or three weeks (during which the typical features of catarrhal jaundice have been present), grave *nervous* and *cerebral* symptoms present themselves, as restlessness and violent headache, followed by delirium, which often becomes maniacal. *Convulsions* then appear, and are succeeded by stupor and coma, the latter occurring usually within forty-eight hours from the onset of the period of cerebral excitement. Often *coarse tremors* are noticed in the voluntary muscles, and with the onset of the second stage the jaundice usually deepens.

The *temperature* often remains normal until just before death, when it may rise one or two degrees. The *pulse* is much diminished both in volume and tension, and is rapid in proportion to the temperature. The *tongue* at the onset is covered with a light coating, most marked on the dorsum and tip. Later, it changes to a thick yellow color and becomes dry and fissured, with the development of a typhoid state. Vomiting appears usually during the premonitory stage and often becomes intense; the *vomit* consists at first simply of the gastric contents, which later in the disease becomes mixed with blood (hematemesis). *Hemorrhages* also frequently occur into the skin (ecchymoses) and from the mucous membranes, giving rise to epistaxis, hematuria, melena, hemoptysis, and menorrhagia. *Constipation* with clay-colored stools is common.

The *urine* in acute yellow atrophy is often scanty in amount, high colored, and shows an increase in specific gravity (1028–1032). The urea is greatly diminished, but bile-pigments and albumin, tube-casts, *leucin* and *tyrosin* are found both on chemical and microscopic examination. The latter can be easily demonstrated by allowing a drop of the urine to evaporate on a cover-glass and examining under the microscope. Tyrosin-crystals are deposited in the form of sheaves and rosettes, leucin as globular masses. These bodies are not constantly present. Thus, out of 34 cases collected by Thierfelder, in which the urine was examined in this relation, "in 7 the result was negative; in 17 both were found; in 3 tyrosin only; in 7 leucin only." Among other products found in the urine worthy of mention are creatinin, lactic and sarcolactic acids, and other bodies belonging to the fatty acid series.

Acute yellow atrophy of the liver is a striking example of acid-intoxication due to rapid and widespread destruction of proteids as the source of the fatty acids—sarcolactic, lactic, diacetic, and β-oxybutyric. It is probable that the rare nervous phenomena of the disease are, in part at least, due to the diminished alkalinity of the blood arising from the abnormal metabolism.

The **physical signs** reveal tenderness over the hepatic region, often amounting to actual pain. During the second stage, in extreme cases, the edges of the organ cannot be palpated under the costal margin. Percussion, moreover, shows a great diminution in the size of the liver, the area of dulness in a case recorded by Harley extending over but 1 inch (2.5 cm.) in the mammary line and $1\frac{1}{4}$ inches (3.1 cm.), measured perpendicularly, in the mid-axillary line.

The left lobe is often the first to show physical signs of atrophy, percussion giving tympany instead of flatness in the upper epigastric region. As the atrophy continues the tympany exends below the seventh rib from above and advances upward from the costal margin, leaving but a small circumscribed area of hepatic dulness. The atrophy is usually progressive until death occurs, although favorable cases have been recorded in which the liver increased in size perceptibly during recovery (Harley, p. 260).

Diagnosis.—The symptoms occurring during the second stage of the disease are usually so characteristic as to leave little doubt concerning the diagnosis. The occurrence of gradually increasing jaundice with vomiting, grave delirium, hemorrhages, the presence of an immense amount of bile, with leucin and tyrosin, in the urine, and greatly diminished size of the liver, all combine to form a typical clinical picture. Unfortunately, leucin and tyrosin are also found in the urine in acute phosphorus-poisoning and rarely in severe acute infective diseases.

Differential Diagnosis.—In *hypertrophic cirrhosis* the onset is more gradual. There is generally a negative previous history; and examination of the urine fails to reveal leucin and tyrosin; fever is rarely present in cirrhosis, and the physical signs often show a considerable increase in the area of hepatic dulness.

The differential diagnosis between this disease and phosphorus-poisoning is given under the latter condition (*vide infra*).

The **prognosis** is almost invariably fatal, since every case of true yellow atrophy is associated with a destruction of liver-cells that is accompanied by acute toxemia.

Treatment.—As yet no specific treatment has been discovered, all remedies used being directed to the relief of symptomatic indications. The gastro-intestinal system should be relieved at the onset by divided doses of calomel. For the vomiting cracked ice, with 1-minim (0.066) doses of the wine of ipecac repeated every half hour or divided doses of opium, may be given. Marked nervous phenomena with delirium I have seen controlled by cool baths and the ice-cap, together with camphor, chloral, or other antispasmodics used internally. Free stimulation should be begun early and persisted in throughout the course of the disease.

THE LIVER IN PHOSPHORUS-POISONING.

Following the ingestion of a dose of phosphorus varying from gr. $\frac{1}{8}$ to gr. 1 (0.008–0.0648) symptoms of poisoning manifest themselves (Taylor, Wormley) as follows:

After a period of time varying from three to twelve hours a sense of wretchedness, nausea, abdominal pain (not intense), and often vomiting, occur. The *vomitus* consists of the gastric contents, with bile, and during the first few hours it may contain phosphorus, which gives it a luminous appearance in the dark.

After the second or third day the vomiting usually ceases with the appearance of *jaundice*, which may become intense as the process continues. Later in the course of the case emesis recommences, the vomita consisting of altered blood, giving rise to the so-called "black vomit." At this stage *nervous symptoms* usually manifest themselves (headache, insomnia, vertigo, and delirium, with convulsions and coma in fatal cases), death closing the scene usually in from thirty-six to forty-eight hours.

The *bowels* are constipated, although attacks of diarrhea may supervene, the evacuations being in some instances phosphorescent.

Fever is irregular and usually is not marked, the temperature swinging from 99° to 101° F. (37.2°–38.3° C.). In fatal cases the temperature may become subnormal just before death.

The *urine* is scanty, of high specific gravity, and contains bile, bile-acids, albumin, sarcolactic acid, and in rare cases leucin and tyrosin (Wood). Renal epithelium and free fat-globules have also been found. When occurring in pregnant women, abortion or miscarriage invariably follows.

Physical examination reveals a liver uniformly enlarged and tender on pressure. In protracted cases atrophy of the organ may rarely occur.

Etiology.—The most common causes are—(*a*) Occupation, workers in match-factories being the most frequent sufferers; (*b*) The accidental swallowing of phosphorus (*e. g.* rat-poison, friction-match heads).

Pathology.—On opening the abdominal cavity in a case of phosphorus-poisoning the liver is seen to extend below the costal margin, its surface being lighter in color than normal and mottled in appearance, and its substance softer in consistence and friable.

The cut section presents marked evidences of fatty degeneration, the acini being lighter in color than the interlobular tissue. Portions of the hepatic parenchyma are deeply bile-stained, and on scraping the cut surface bile- and fat-globules will be found on the edge of the knife. The gall-bladder may be either full or empty. Microscopically, disintegrated liver-cells, fat-globules, granular débris, biliary coloring-matter, leucin-spheres, cholesterin-plates, and tyrosin-needles are noted.

The *gastric mucosa* is found thickened, opaque, and yellow-white in appearance, due, as pointed out by Virchow, to a universal gastro-adenitis, and not to the local action of the poison. Ulcerative or erosive gastritis is very rare in phosphorus-poisoning.

The *kidneys* may show beginning atrophy, the epithelium in the cortices undergoing granular and fatty degeneration, with final destruction of the cells.

The *blood* is dark, fluid, and not easily coagulable. Concato found that during life the white corpuscles are increased in number, and that the red are changed in shape and smaller than normal (Wood). Petechiæ and ecchymoses frequently appear in all parts of the body.

Diagnosis.—The diagnosis of acute phosphorus-poisoning is always extremely difficult and often impossible. The disease with which it is most apt to become confounded is *acute yellow atrophy of the liver.* The differential points may be summated as follows:

ACUTE PHOSPHORUS-POISONING.	ACUTE YELLOW ATROPHY.
There is a history of accidental ingestion of poison (friction-match heads, rat-poison) or occupation.	There may be an endemic history.
The onset is sudden; violent nausea, vomiting, and pain over the region of the liver. Jaundice appears on the second or third day.	A slow onset—malaise, slight fever, with nausea and vomiting; jaundice is a beginning symptom.
Nervous symptoms appear late in the disease—always preceded by jaundice.	Nervous symptoms may appear early, even before the occurrence of jaundice.
The vomit and stools are phosphorescent. Black vomit precedes death.	Black vomit occurs early and persists throughout.
Temporary arrest of symptoms between the occurrence of jaundice and black vomit.	Progressive march of symptoms with no remission.
Sarcolactic acid is present in the urine, and rarely leucin and tyrosin.	Leucin and tyrosin are common in the urine.

Prognosis and Duration.—The prognosis in phosphorus-poisoning is bad, as small a dose as gr. $\frac{1}{8}$ (0.008) of white phosphorus having caused death (Wormley). The duration is usually from one to six days, although the symptoms have been known to persist for twelve days before death. In violent cases the end may come within twenty-four hours.

Treatment.—The initial plan of treatment is by causing emesis to free the system of the poison that still remains undigested. For this purpose copper sulphate (gr. x—0.648) in divided doses (gr. ij or iij—0.129 or 0.194—every five minutes) should be given until free vomiting occurs. As copper sulphate is a chemical antidote, forming with phosphorus black copper phosphid, it should be continued in less frequently repeated doses (gr. ij—0.129—every half hour) and guarded by morphin to prevent vomiting. If emetics by the mouth fail to afford relief, apomorphin muriate (gr. $\frac{1}{5}$—0.0129), hypodermically, may be resorted to. The free evacuation of the stomach should be followed by the administration of the French oil of turpentine. Wood recommends that 1 part be given to every 100th part of the poison ingested. Ordinary turpentine is useless, but combined with mucilage of acacia 2 fluidrams (8.0) of French oil of turpentine may be given every fifteen minutes until 1 ounce (32.0) has been taken.

Alkalies (magnesia) have been given, but are practically valueless. Free purgation should be effected if possible by Rochelle salts or magnesium citrate. Demulcent oils are never allowable, as they dissolve the phosphorus and hold it in solution. After absorption of the poison and degeneration of the tissues have taken place all known remedies are futile.

CIRRHOSIS OF THE LIVER.

(*Sclerosis of the Liver; Nutmeg Liver; Gin-drinker's Liver; Interstitial Hepatitis.*)

Definition.—A chronic disease of the liver, characterized, pathologically, by an excess of connective tissue. It presents various biliary, gastro-intestinal, circulatory, and cerebral symptoms.

Pathology.—There are three pathological varieties: (*a*) atrophic cirrhosis, or "gin-drinker's liver"; (*b*) hypertrophic cirrhosis; and (*c*) biliary cirrhosis.

(*a*) **Atrophic Cirrhosis** (*Laennec's*, or *alcoholic cirrhosis*) is the most common form, at least in the earliest stages, as Foxwell's studies teach; the alcoholic (indurative) liver is more commonly enlarged than decreased in size. Morse[1] examined the records of 37 cases of cirrhosis, and found that among these there were 13 instances of enlarged liver, 11 of normal size, and 12 smaller than normal. In typical examples the capsule is thickened, the organ greatly reduced in size, hard, granular, and much altered in shape. On section (which resists the cutting-knife) the surface presents grayish-white bands of connective tissue surrounding yellowish areas (acini) that project above the surface from compression (hob-nails); hence the term "hob-nailed liver."

Microscopically, the process is seen to commence as an increase in the connective-tissue element surrounding the terminal branches of the portal vein. Compression of the liver-cells and of the portal veins, with consequent obstruction of the circulation, constantly increases with the progress of the proliferation of the connective tissue and its secondary contraction. Atrophic changes in the hepatic cells, however, are often comparatively slight. The biliary canaliculi may be increased in number. Weigert and his disciples contend that atrophy or degeneration of the acini is often the primary change, and the connective-tissue production the secondary—filling the gap, so to speak.

In alcoholic cirrhosis the liver is sometimes large, smooth, or slightly granular, soft rather than hard, as ordinarily the case, and presents a light yellow color (*fatty cirrhosis*). Histologically, this is a form of true cirrhosis, as shown by the presence of an increase in the connective tissue, with which, however, fatty infiltration of the acini is associated.

(*b*) **Hypertrophic Cirrhosis** (Hanot).—On examining the liver *in situ* during hypertrophic cirrhosis the organ is found enlarged (sometimes enormously), the lower border projecting several fingers' breadths below the ribs. The margin of the organ is well defined, the substance firmer than normal, and it cuts with difficulty. The organ is lighter in color than in health, and presents a yellow or mottled-green appearance. On treating a section with compound iodin solution (Lugol's) the color changes to that of a deep mahogany-red. The acini are darker in hue than the interstitial tissue.

Microscopically, the peripheral zones of the acini are first seen to be the seat of a round-cell infiltration, with the formation of embryonal tissue; later, the interlobular connective tissue undergoes hyperplasia, causing obstruction of the biliary ducts with retention of bile, and subsequent atrophy of the liver-cells. New-formed bile-ducts are proliferated.

(*c*) **Biliary Cirrhosis.**—French writers have described "biliary cirrhosis" as opposed to a "portal cirrhosis" or atrophic. It results from obstruction of the bile-ducts; this causes retention of bile with swelling of the organ as a consequence. The irritant substances that are the result of stagnation of the bile start a cirrhotic process around the small bile-ducts (reactive inflammation). The *microscopic appearances* of the organ simulate those of hypertrophic cirrhosis; but the hepatic cells are more deeply bile-stained. *Microscopically*, the first discoverable changes are spots of insular necrosis in the peripheral zones of the acini (Stengel). These are shortly replaced by proliferation of the interlobular connective tissues. The formation of new-ducts and liver-cells is common.

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[1] *Boston Med. and Surg. Journ.*, March 10, 1898.

There is also a so-called Glissonian cirrhosis (perihepatitis) in which the capsule of the organ is surrounded by a dense white fibrinous membrane, which contracts, reducing the size of the liver and altering its shape. This I have described elsewhere (*vide* Chronic Hepatitis, p. 881). Syphilitic cirrhosis of the organ receives special consideration in the section devoted to Syphilis (*vide* p. 337).

Etiology.—(*a*) **Atrophic Cirrhosis.**—1. *Alcoholism.*—Freyhan, Osler, and I myself have found this causal factor operative in nearly all cases. Clinical history tends to prove that the stronger the alcoholic beverage (*e. g.* raw spirits) and the larger the amount consumed the sooner cirrhosis develops, although the quantity necessary to produce the disease varies greatly in different individuals. Doubtless by the side of alcoholism all other causes combined are comparatively insignificant.

2. *Spicy foods* are, according to some, classed as predisposing agents. Tiraboschi records a case that had long been induced by the use of spicy foods and by over-eating. In many cases ptomains, the products of malassimilation through faulty digestion, are supposed to be the exciting cause.

3. *Male Sex and Middle Life.*—The cases produced by alcohol occur chiefly in males. According to my experience, females who misuse potable alcohols, particularly the more concentrated liquors, are less susceptible to the poison than males. Two-thirds of the fatal cases occur between the ages of 35 and 50 (Hawkins), although cases have been known to occur at both extremes of life. In children the disease is mostly caused by inherited syphilis, in which the organ is the seat of a general fibroid process. It may follow the acute infectious diseases, notably scarlet fever, in the young.

4. Certain *chronic diseases* (syphilis, rickets, diabetes, gout, malaria, carcinoma, tuberculosis) that favor the formation of connective tissue are apt to be complicated by cirrhosis, usually partial.

5. *Passive congestion*, secondary to chronic cardiac lesions or to obstructive lung-disease, not infrequently gives rise to hepatic cirrhosis.

6. Fatty cirrhosis results from the abuse of malt liquors in some cases, and is often associated with more or less obesity.

(*b*) **Hypertrophic Cirrhosis** (Hanot).—In most cases there is an absence of recognizable causes. Sex is a strongly predisposing cause, males being the most frequent victims, in the proportion of 6 to 1. It is not uncommon in young adults. In catarrhal jaundice the morbid processes may rarely extend to the liver and there persist, giving rise to hypertrophic cirrhosis. Cases are met with in children, in whom it may follow the acute infectious diseases. Alcohol plays an unimportant rôle in the causation of Hanot's cirrhosis. The disease is most common among the inhabitants of warm climates.

(*c*) **Biliary Cirrhosis.**—This form is produced by chronic obstruction of the bile-ducts (see also Obstruction of the Common Duct, p. 860).

Symptoms.—**Atrophic Cirrhosis.**—The symptoms of this variety of cirrhosis may present nothing characteristic as long as the sclerotic process does not interfere with the portal circulation. In some cases the collateral (compensatory) circulation is maintained throughout the long course and symptoms fail to arise. Among the *prodromal symptoms*, a gradual loss of flesh, anorexia, constipation, a coated tongue, slight jaundice, dyspepsia, and occasionally hematemesis are to be mentioned.

As the obstruction of the portal circulation becomes more marked the mucosa of the gastro-intestinal tract becomes more and more swollen and congested, and gives rise to augmenting *nausea* and *vomiting* (most marked in the morning), and *hemorrhages* from the stomach (hematemesis) and intestines (melena), which may be frequent and profuse, but are rarely fatal. Severe hemorrhages may also occur from enlarged, varicose esophageal veins. The *tongue* is coated.

Owing to the establishment of a compensatory circulation the superficial epigastric and internal mammary veins enlarge, forming about the umbilicus the so-called "caput Medusa."

Hemorrhoids also are not uncommon, and are due to passive congestion of the inferior hemorrhoidal veins. As the disease progresses the *general emaciation* becomes more marked. The face assumes a pinched expression, the tip of the nose has a purple tinge from distended veins; the eyes are sunken, the cheeks hollow, and the skin presents a sallow tint (*hepatic facies*). The failure of the compensatory circulation gives rise to *ascites*, and the latter causes in many instances hydroperitoneum, leading to enormous distention of the abdomen. The *spleen* becomes enlarged. At any stage, although generally in advanced cases, *toxemic symptoms* may develop, due to some poisonous product in the blood, the exact nature of which is unknown: these are violent headache, followed by wild, noisy delirium, convulsions, stupor, and coma. They not uncommonly occur without jaundice, and have been mistaken for uremia.

Fever may be absent throughout the course of the disease, but is often present, and may reach 100°–102° F. (37.7°–38.8° C.).

Examination of the *urine* shows it to be of increased specific gravity, loaded with urates, and containing bile. In a small proportion of cases it is slightly albuminous, and contains casts, though out of 28 urinalyses in cases of cirrhosis Henry discovered the presence of albumin in but one. The amount of urea is constantly diminished, owing to the disturbance of the urea-forming function of the liver. An excess of indoxyl sulphate in the urine is a frequent occurrence.

The *physical examination* in a typical case of atrophic cirrhosis reveals a distention of the abdomen; there may be also an extreme enlargement of the superficial veins over the surface of the body. An icteroid tint of the skin is present in about 25 per cent. of the cases.

Palpation of the liver and spleen may be greatly interfered with by the large amount of peritoneal fluid present. On withdrawal of the latter, however, the spleen is found greatly enlarged.

The liver may show slight enlargement in the beginning of the disease; but it soon atrophies, and in emaciated subjects with lax abdominal walls its finely granular or nodular edge can be *felt* above the margin of the ribs. *Percussion* shows its vertical diameter, which normally extends from the sixth interspace to the costal margin, and averages about 4 inches (10 cm.), diminished, especially toward the median line. Posterior dulness begins lower than normally. It must be recollected that the liver is often enlarged in otherwise typical cases.

Fatty cirrhosis, in which the organ is sometimes enlarged, may be latent and remain unrecognized or be discovered on the post-mortem table. In five of the six cases that have fallen under my observation, however, the symptoms closely resembled those of the ordinary form of atrophic cirrhosis, apart from the alteration in the size of the liver.

(*b*) **Hypertrophic Cirrhosis.**—In this variety of the disease there is usually an absence of any alcoholic history, and it is apt to be met in young adults and even children (*vide* Etiology). *Moderate enlargement* of the liver may be present before any *subjective symptoms* are observed. The latter may be absent, except the presence of slight jaundice and an occasional disturbance of digestion, until late in the course of the disease. Intense jaundice, fever, and hepatic enlargement may then appear, with the rapid development of a grave general condition. The *urine* contains bile-pigment, but the stools are not typical (pale drab or slate colored). Paroxysms of *pain* resembling hepatic colic, though less severe, may occur at irregular intervals. *Hemorrhages* into the skin from the mucous surfaces (due to passive congestion) are also common. In long-standing cases albumin and tube-casts may be present in the urine. Leucin and tyrosin have also been found, but are not constant. These symptoms are probably due to recent inflammatory infiltration arising in the course of an old cirrhosis. Splenic enlargement occurs, but ascites is exceedingly rare. The cases run an extremely chronic course, and in an instance under my care in a lad of 14 years, the grave symptoms mentioned above suddenly developed and carried off the patient after four years of slight, though decisive jaundice, and moderate hepatic enlargement. The stools were dark and bilious looking, and hemorrhages from the mucous surfaces frequently occurred; petechiæ, with urticaria and lichen, marked the skin, while pruritus was exceedingly distressing. There was a leucocytosis.

Physical examination shows a decided, and, in some cases, a uniform enlargement of the organ; the lower border is felt distinctly outlined below the costal margin, its edges being rounded and at times granular.

Percussion shows an increased area of hepatic dulness.

Late in the disease, in addition to the grave symptoms described above—icterus gravis, high fever, hemorrhages, and the like—serious nervous symptoms, as delirium, convulsions, stupor, and coma, may supervene. The temperature now usually ranges from 102° to 104° F. —38.8°–40° C. (*febrile jaundice*)—although fever may sometimes be absent throughout the course of the disease. Death results either from an intercurrent disease or progressive asthenia.

Hemochromatosis (Opie).—Recklinghausen first called attention to hemochromatosis in connection with cirrhosis. Its association with diabetes mellitus and bronzing of the skin I have previously referred to (p. 380). There are cases, an illustration of which was reported by Opie, in which bronzing of the skin, cirrhosis of the liver, and chronic interstitial pancreatitis occur without diabetes. Opie's conclusions may be cited: (1) "There exists a distinct morbid entity, hemochromatosis, characterized by the widespread deposition of an iron-containing pigment in certain cells and an associated formation of iron-free pigments in a variety of localities in which pigment is found in moderate amount under physiologic conditions. (2) With the pigment accumulation there are degeneration and death of the containing cells, and consequent interstitial inflammation, notably of the liver and pancreas, which become the seat of inflammatory changes accompanied by hypertrophy of the organ. (3) When chronic interstitial pancreatitis has reached a certain grade of intensity, diabetes ensues, and is the terminal event in the disease."

(*c*) **Biliary Cirrhosis.**—*Symptoms and Diagnosis.*—The clinical interest of this form centers principally around the symptoms of the causative condition—chronic obstruction of the bile-ducts—which have been given in detail elsewhere (*vide* p. 860). With the latter may be associated the features, both local and general, of either catarrhal or suppurative cholangitis. *Jaundice* is usually more intense than in the hypertrophic form, particularly during the earlier stages. *Intermittent hepatic fever* is commonly observed. The *physical signs* are similar to those of Hanot's cirrhosis.

The *diagnosis* of biliary cirrhosis rests on the presence of the characteristic features of prolonged obstruction of the bile-ducts, from impaction by gall-stones, a tumor or stricture of the duct, and the like, with slow and gradual, smooth, or slightly granular, hepatic enlargement. It is to be recollected that when obstruction of the gall-ducts becomes complete, or "acute fermentative changes" are set up in the retained bile, the cases may terminate acutely (*e. g.*, in acute atrophy).

General Diagnosis.—(*a*) **Of Atrophic Cirrhosis.**—An assured diagnosis may be based on the following points: 1. A clear history of the most common causes (inebriety, male sex and middle life, rickets, diabetes, gout, malaria). 2. The combined presence of ascites, with Hippocratic facies, and diminution in the size of the liver, as shown by the physical signs. 3. Absence of the characteristic features of acute disease, and the negative character of results from an examination of the heart, lungs, and kidneys. It is to be recollected that the volume of the liver is not invariably decreased, and even may be increased.

With the atrophic form of cirrhosis, *chronic peritonitis with effusion* is most liable to be confounded. In the latter disease there are characteristic abdominal tenderness, fever, and usually associated tuberculous lesions of other organs (lungs, kidneys, intestines); but the hepatic facies and clearly indicative history of atrophic cirrhosis are absent. A large peritoneal effusion is in favor of cirrhosis

(*b*) **Of Hypertrophic Cirrhosis.**—The principal diagnostic points are an absence of the usual alcoholic history, slight icterus, extending over a variable and oftentimes long period, paroxysms of pain, mucous and cutaneous hemorrhages, with moderate enlargement of the liver, and the rapid development of grave symptoms at any stage, as intense jaundice, fever, and sometimes marked nervous phenomena.

Differential Diagnosis of Hypertrophic Cirrhosis.—This may be confounded with *carcinoma of the liver*, *hydatid cyst*, *hepatic abscess*, *and the biliary form of cirrhosis.*

HYPERTROPHIC CIRRHOSIS.	CARCINOMA OF THE LIVER.
Absence of recognizable causes.	Hereditary history.
Occurs in young adults and in childhood.	Usually occurs after forty years of age.
Usually a primary affection.	Often occurs as a secondary growth.
Jaundice is slight unless grave symptoms develop; there is no cachexia.	Anemia is present, and also the development of a typical cachexia.
Paroxysms of pain. The case runs a slow course, usually lasting many years.	Pain more constant with rapid emaciation. The case terminates usually within one year.
Enlargement is uniform.	The liver is irregularly enlarged, and contains umbilicated nodules.

(See Fig. 58.)

HYPERTROPHIC CIRRHOSIS.	MULTILOCULAR HYDATID CYST.
History negative as to alcohol. More common in warm climates.	History of ingestion of the embryo of tænia echinococcus with improper food.
Occurs idiopathically.	Simultaneous occurrence in colonies or in others in the vicinity.
Fever, jaundice, and ascites may be present singly or together.	No fever, pain, jaundice, or ascites.
Anemia and emaciation slowly progressive. There is a leucocytosis.	Emaciation not marked; no leukocytosis.
Regular enlargement of the liver. No fluctuation nor thrill.	On palpation an irregular, fluctuating tumor is felt over the hepatic area, giving an "hydatid thrill."
Aspiration is negative.	Aspiration gives a clear, serous fluid, rich in chlorids, and containing hooklets.

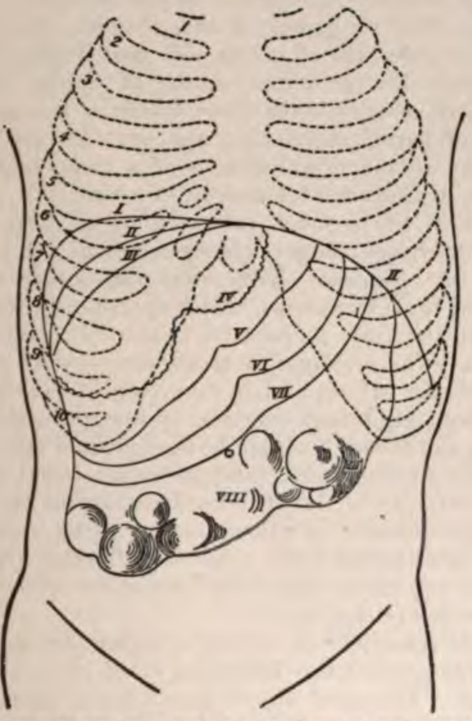

FIG. 58.—Showing approximate enlargement of the liver corresponding to the different diseases described in the text (after Rindfleisch): *I*, position of the diaphragm to the maximum enlargement (carcinoma); *II. II*, normal situation of the diaphragm; *II*, *III*, relative dulness; *IV*, border of the liver in cirrhosis; *V*, border in health; *VI*, lower border of the fatty liver; *VII*, of the amyloid liver; *VIII*, of carcinoma, leukemia, and adenoma.

HYPERTROPHIC CIRRHOSIS.	ABSCESS OF THE LIVER.
Etiology usually negative. May rarely follow acute infectious diseases.	History of dysentery, traumatism, or pyemia.
There are tenderness on deep pressure and paroxysmal pain.	Severe and constant pain; marked tenderness.
Hectic symptoms absent although a continued fever may develop usually late.	Hectic symptoms appear early (fever, chills, and sweating).
Slow course, lasting months or years,	Acute course, lasting a few weeks.

HYPERTROPHIC CIRRHOSIS (*continued*).	ABSCESS OF THE LIVER (*continued*).
Slow enlargement, regular, or slightly nodulated. No fluctuation.	Rapid development of a fluctuating tumor in the hepatic area.
Aspiration gives negative results.	The aspirating-needle reveals pus.

So-called *fatty cirrhosis* may be distinguished from abscess, hydatid cyst, and carcinoma of the liver in a manner similar to hypertrophic cirrhosis, if one makes due allowance for its etiology, alcoholism.

(*c*) *Biliary cirrhosis* causes enlargement of the liver, but to a much more moderate extent than hypertrophic cirrhosis. In the former the symptoms of chronic obstruction of the bile-ducts are in evidence, so that jaundice is usually marked. The duration of biliary cirrhosis is, on the whole, shorter than that of hypertrophic, and the organ is more likely to undergo terminal diminution in size (atrophy).

Prognosis.—The prognosis of the atrophic form of cirrhosis is decidedly unfavorable, the function of the liver-cells having been impaired, although the principal source of danger is probably the ascites; and death usually takes place within a few months or a year after symptoms of portal obstruction appear. In rare cases the symptoms abate, owing to the establishment of a compensatory circulation, and may remain in abeyance for months or years.

The prospect of life is much enhanced by an early recognition and removal of the overshadowing cause—alcoholism. I have seen a few cures made in this manner. Even after the occurrence of jaundice, hematemesis, and toxic symptoms, under appropriate treatment patients have been known to enjoy comparative health for years.

Treatment.—The *prophylactic* treatment consists in improving the general health of the patient and in removing, if possible, the cause of the affection. Rest, graduated exercise, systematic bathing, and regular hours for eating and sleeping should be inaugurated and strictly adhered to. Alcohol, strong coffee, spices, and gastro-intestinal irritants of every nature must be interdicted. H. C. Wood states that tavern-keepers and bartenders who are unable or will not cease using alcohol may greatly prolong life by substituting hard cider for all other drinks. The *diet* should be simple and easily digestible. An exclusive milk-diet has been highly recommended (Semmola).

The *medicinal* treatment is largely symptomatic, no remedy having been discovered to prevent the formation of, or remove, the new-formed connective tissue. The chief object is to deplete the portal system and prevent, if possible, the occurrence of ascites. The bowels should be kept freely open by the use of saline purgatives (concentrated solution of Epsom salts), elaterium, or compound jalap powder. The skin is to be kept active by means of Turkish or Russian baths (under supervision), and in extreme cases by the steam bath or hot pack, employed just short of the point of exhaustion. The kidneys should also be kept active by the hydragogue diuretics, as potassium acetate, squills, calomel, digitalis in the form of the infusion, or Niemeyer's pill. Klemperer and others have also recently recommended urea as an efficient diuretic, and from 20 to 30 grains (1.29–1.94) may be given in solution.

If the case be syphilitic in origin, potassium iodid should be exhibited.

Ascites, when it appears, calls for free and thorough diuresis, diaphoresis, and catharsis; and if not relieved in the course of a few days, tapping should be resorted to.

The operation of *paracentesis abdominis*, if performed under strict antiseptic precautions, is free from danger. The bladder having been emptied, a spot over the linea alba about 3 inches (7.5 cm.) above the symphysis pubis is anesthetized (preferably with a compress of cracked ice and salt), and a trocar is quickly thrust through the abdominal wall for a distance of about 1 inch (2.5 cm.). The distance is determined by the fore-finger, which is placed at the desired distance from the point of the cannula before its insertion. The patient must be in a sitting or semi-reclining position, so as to allow the ascitic fluid to collect by gravity in the lower part of the abdominal cavity. A tube having been attached to the cannula to convey the liquid to a receptacle, the trocar is withdrawn, the fluid allowed to run out, the cannula removed, and the wound closed by antiseptic gauze or a pledget of cotton. Turlington's balsam is then smeared over the site of puncture, and the abdominal binder, which has been previously applied, is tightened.

Morison, Frazier, and others have reported cases of *epiplopexy* (suturing the great omentum to the anterior abdominal wall for the purpose of establishing a collateral venous circulation), for the relief of the ascites in cirrhosis. The operation is founded upon physiologic premises, hence is promising.

Complications, as cardiac hypertrophy, tuberculous peritonitis, or chronic meningitis, demand appropriate treatment.

CARCINOMA OF THE LIVER.

Definition.—A malignant growth of the liver, occurring usually after the age of forty, and characterized by pain, progressive emaciation, cachexia, and the appearance of a nodular mass in the hepatic parenchyma. It may be primary or secondary, though the former is rare.

Pathology.—Histologically, the cells are not distinctive, being identical with those of carcinoma elsewhere; they are epithelial in character, having a small vesicular nucleus and much protoplasm. They are altered greatly by pressure, and vary in shape, being hexagonal, polyhedral, or amorphous. Large giant-cells and spots of pigment known as "brownish granules" are not uncommonly found in the cancerous mass. The so-called colloid cancers are nearly always mucoid, and the cells have undergone a mucoid change; the stroma of connective tissue surrounding the cancer-nests in some instances undergoes hyaline or myxomatous degeneration. In other instances the interstitial trabeculæ completely surround the epithelial nests, which are separated by a basement-membrane; this variety is termed *adeno-carcinoma*.

When examined microscopically, *medullary cancer*, either in a large mass (primary) or in secondary nodules scattered throughout the organ, is the most common variety found in the liver. On examining a liver that is the seat of carcinoma, one of two conditions usually presents itself: *First*, the organ may be apparently normal with the exception of one lobe (usually the right), which contains a dense whitish growth of firm consistence, being distinct and sharply defined from the surrounding liver-tissue. On section the tumor is often of uniform density, bluish-white in appearance, and exudes a milk-white fluid known as "cancer-juice," which, when examined microscopically, is found to contain large, nucleated, and irregularly-shaped cells containing free granular matter. The center of the tumor may have undergone liquefaction-necrosis, with the formation of a cyst, or it may be the seat of an abscess. Various smaller nodules may be scattered throughout the organ by metastasis from the primary growth. The *second* and most common condition is secondary carcinoma of the liver, the primary lesion being situated in the mammary glands, pylorus, or the cervix uteri. The organ is greatly enlarged, as a rule. Numerous nodules are scattered throughout, and can usually be seen projecting beneath the capsule, those superficially situated having received the name of "Farre's tubercles." In the center of these nodules characteristic pits or umbilications are often present, caused sometimes by contraction of the interstitial trabeculæ and sometimes by a central softening. On section they are usually grayish-white in color and of firm consistence, although cysts, hemorrhages, pus-cavities, or areas of hyaline and fatty degeneration are often found. The cells are identical with those of the primary growth, and are composed for the most part of cylindric epithelium.

In rare instances carcinoma occurs simultaneously with *cirrhosis* in the same liver, the organ presenting an uneven, nodular appearance, and being slightly increased in size and of firmer consistence than normal. When examined *in situ* the external appearance does not differ materially from that of cirrhotic liver, but on section the whole organ is found to be infiltrated with various-sized cancer-nodules surrounded by bands of cicatricial tissue. In some cases the excess of connective tissue and the amount of contraction are extreme, and the size and weight are reduced below the normal.

Etiology.—Among the more important predisposing factors may be mentioned—

(*a*) *Age.*—The disease seldom occurs before thirty-five or forty years of age, although cases have been known to occur in children. Descroizilles reports the case of a child eleven years old who died with a tumor in the right hypochondriac and iliac region, the autopsy revealing a liver studded with cancerous nodules, the nature of which was demonstrated microscopically.

(*b*) *Sex.*—Men are more often the victims of carcinoma of the liver than women. When occurring in the latter it is often secondary to carcinoma of the uterus or mammary gland.

(*c*) *Heredity* is said to be the cause of hepatic carcinoma in at least 20 per cent. of all cases, and is one of the strongest arguments in support of the diathetic theory of the disease.

(*d*) *Traumatism* may contribute.

(*e*) *Mechanical Obstruction.*—Primary carcinoma of the gall-bladder and bile-ducts not infrequently follows chronic obstruction by gall-stones.

Symptoms.—There may either be almost no symptoms of carcinoma involving the liver, or its manifestations may be intense and varied according to the extent and location of the growth or growths. Associated gastric symptoms, which increase as the disease advances, usually attend. A more or less marked cachexia may be the first noticeable feature. The chief symptoms may be considered in detail, as follows:

(*a*) *Jaundice.*—Discoloration of the skin and tissues is often by no means intense, and may be entirely absent. Harley states that true icterus was present in only 6 out of 100 cases seen by him, though few observers agree with him in his extreme view as to the rarity of this symptom. The reason given for its lack of intensity is that in the great majority of cases the growth is situated in the right lobe of the liver, and neither compresses the bile-ducts nor destroys the secretory cells of the liver.

(*b*) *Pain* is usually present to a marked degree, though it also may be entirely wanting. It is dull and boring in character, and localized generally in the right hypochondriac region. In some instances (as in the case of impacted biliary calculi) it may radiate to the right shoulder and the scapular region. It usually appears as the hepatic enlargement progresses, although cases of enormous-sized cancerous tumors of the liver have been known to occur without pain. The character and location of the pain are of diagnostic importance, and will be spoken of under the differential diagnosis.

(*c*) *Ascites.*—When the cancerous growth compresses the portal vessels, and also in cases of cirrhosis with carcinoma, obstruction to the portal circulation occurs, and results in the development of ascites. This may cause distention of the abdominal cavity to such an extent as to occlude the physical signs of hepatic enlargement. The cancerous growth may invade the peritoneum and cause an effusion. This symptom, however, is not frequent, at least two-thirds of all cases terminating without the appearance of ascites.

(*d*) *Fever* is usually absent until the later stages of the disease. It may then appear and rise to hyperpyrexia (105° F.—40.5° C.), but it is usually moderate in degree, irregular, and intermittent in type.

(*e*) *Cachexia.*—In every case of carcinoma, at some stage of the disease, cachexia develops; when pronounced, it is almost pathognomonic.

(*f*) *Cerebral Symptoms.*—These may be absent throughout. In the advanced stages, however, the deleterious products in the blood, due to the perverted functions of the liver and the toxemic condition of the patient, often produce such striking symptoms as violent headache, mental hebetube, or delirium (less frequently) which may be maniacal in character. These symptoms resemble those of cholemia (*vide* Hepatic Cirrhosis, p. 891). The patient may die in sudden coma.

Physical Signs.—*Inspection* often reveals enlargement of the superficial veins over the abdomen, and a prominence in the upper epigastric and hepatic regions, varying with the degree of enlargement, may also be seen. In the nodular form and late in the disease, when emaciation has become extreme, elevations that are movable with respiration can be noticed beneath the skin.

On *palpation* the organ can be distinctly felt projecting below the costal margin and extending in some instances to a point below the level of the umbilicus. During deep inspiration the liver can be felt to move downward, and during expiration upward, the organ being under the influence of the diaphragmatic excursions. In emaciated subjects the cancer-nodules are readily appreciable, and in some instances the central pits or depressions are palpable, forming a pathognomonic sign. Cancerous infiltration of the anterior margin is most easily felt, and in any enormous enlargement of the organ I have frequently detected them on the posterior surface as well. Rarely the liver is found to be uniformly large. Palpation may also show splenic enlargement, due to passive congestion.

Percussion shows flatness, extending in many cases in both an upward and a downward direction. In primary carcinoma (usually found in the right lobe) the area of hepatic dulness is increased irregularly downward and generally to the right. On the other hand, in secondary growths (usually massive) the nodules are oftener distributed equally throughout the liver. In such cases the area of dulness may extend across the epigastrium to the left hypochondriac region, the heart and other viscera being now displaced. Posteriorly, dulness may extend upward on a level with the fourth rib, and anteriorly downward to the iliac fossa. The organ may now weigh from 15 to 20 lbs. (6.5–9 kgms.), while the weight of cancerous livers in ordinary cases varies between 3 and 6 lbs. (1.3–2.6 kgms.).

Diagnosis.—In forming a positive diagnosis the family tendency, the history of primary carcinoma elsewhere in the body, the age of the patient, the localization of the pain in the right hypochondrium, the cachexia, and the progressive enlargement of the liver, with the characteristic umbilicated nodules, are the most reliable points. The appearance of jaundice or ascites, or both, is confirmatory.

Differential Diagnosis.—Among affections of other organs that are likely to be mistaken for carcinoma of the liver may be mentioned—(1) carcinoma of the *pylorus;* and (2) carcinoma of the *colon and omentum.* The chief diseases of the liver itself apt to be diagnosed as carcinoma are—(*a*) *abscess,* (*b*) *syphilis,* (*c*) *benign growths* (*adenomata, angiomata*), (*d*) *hydatid cysts,* and (*e*) *hypertrophic cirrhosis.*

(1) *Carcinoma of the Pylorus.*—In carcinoma of the pylorus the physical examination frequently shows a hard nodular tumor that is most plainly outlined in the epigastric region. In a typical case, on deep inspiration, the tumor is pressed downward by the liver, but is not pulled upward by forced expiration, as in hepatic carcinoma. In many instances, however, adhesions bind the stomach firmly to the under surface of the liver, which may be the seat of secondary involvement. The absence of early nausea and vomiting and the presence of jaundice, as well as the negative results from an examination of the gastric contents, would tend to eliminate pyloric carcinoma.

(2) *Carcinoma of the Colon and Omentum.*—Secondary carcinoma of the intestine affects most frequently the sigmoid flexure. The symptoms of intestinal obstruction arise, constipation being followed by attacks of serous diarrhea due to irritation, and later by the presence of blood in the stools. In carcinoma of the liver, on the other hand, the bile-ducts may be obstructed, causing clay-colored stools, but otherwise the dejecta are normal; the seat of the nodular enlargement and pain is located in the right hypochondrium. Jaundice and ascites are absent in carcinoma of the colon. The tumor, if palpable, in the latter condition is more movable and is less under the influence of the diaphragm. It does not

give an absolutely flat percussion-note, as does hepatic carcinoma. Carcinoma of the omentum is usually secondary. The absence of small movable tumors in the umbilical, lumbar, or hypogastric regions, ranging in size from that of a pea to a walnut, aids in the elimination of carcinoma of the omentum. As the latter affection advances the abdomen becomes distended and painful to the touch, the bowels are obstinately constipated, and the physical signs reveal the presence of an effusion which, when aspirated, is generally serous, but sometimes bloody. Microscopic examination may possibly reveal the presence of cancer-cells, though their recognition is difficult. The liver, unless primarily involved, is not enlarged, and cachexia does not usually appear until late in the course of the disease.

From *hepatic abscess* the points of differentiation are—

CARCINOMA OF THE LIVER.	HEPATIC ABSCESS.
Is often hereditary. There is a history of a primary growth or chronic irritation.	There is a history of traumatism or of intestinal ulceration, as in dysentery.
Occurs usually after the age of forty.	Occurs at any age.
Jaundice is rare.	Jaundice is sometimes present.
Fever is absent or slight.	Hectic temperature, chills, and sweating.
Cachexia is present and almost pathognomonic.	Anemia may be present, but *never cachexia.*
Pain is dull and boring in character, and more constant.	Pain is sharp, lancinating, and paroxysmal.
A nodular, umbilicated tumor or tumors may be detected.	A fluctuating tumor may sometimes be detected below the costal margin.
The enlargement is downward.	The enlargement usually upward.
The duration is a few months to one year.	The duration is usually a few weeks.
Microscopic examination reveals disintegrated liver-cells, cancer-nests, and in some cases the micro-organisms of suppuration.	The microscope reveals pus, liver-cells, staphylococci and streptococci, the bacillus coli communis or the amœba coli.

Benign Growths (Adenomata, Angiomata).—Occasionally growths are detected in the liver, and may occur at any age; when these are present at or about the age of forty, they may be mistaken for carcinoma. The absence, however, of a primary growth in some one of the other viscera, together with the duration of the growth and the absence of cancerous cachexia, would tend to differentiate them from cancerous involvement. An examination of the blood may be of service, leukocytes being more common in carcinoma.

The **prognosis** is invariably fatal, the disease terminating rapidly in from a few months to a year. The most rapid course is run by secondary carcinoma of the organ.

Treatment.—The treatment is purely symptomatic. An easily digested, nutritious diet should be given, together with active stimulation to support the system. The pain may be relieved by the free use of morphin, given by the mouth, rectum, or hypodermically. For the nausea and vomiting that are apt to supervene the carbonated waters, cracked ice with champagne, or repeated doses of creasote (beechwood), dilute hydrocyanic acid, or wine of ipecac (2 minims—0.133—every hour until relieved) may be given. If violent delirium should occur during the later stages of the disease, cold compresses to the forehead or vertex, and bromids and chloral hydrate given in rectal enemata, may prove efficient.

OTHER NEW GROWTHS IN THE LIVER.

(a) Angioma, Adenoma, and Cyst.

Occasionally, benign growths occur in the liver, and often with an absence of symptoms unless their increase in size gives rise to mechanical obstruction. One of the most common of these is **angioma**, which is often found in the livers of old people. Angiomata consist of tortuous and dilated capillaries in the hepatic connective tissue; they rarely attain to a size larger than a crab-apple, and usually cause no symptoms. Although most common in adults, they have been known to occur in children.

Adenomata and cystomata may also occur in the liver. They are both benign growths. The former is of the tubular variety, consisting of connective-tissue nests lined with cylindric epithelial cells. Von Bergman removed a portion of a tuberous adenoma of the liver with perfect recovery and non-recurrence of the growth. Cysts are quite rare. Lippmann,[1] who searched the literature, found reported 3 retention cysts, 9 cystic adenomas, 1 lymph cyst, and 3 cases that could not be classified.

(b) Sarcoma.

Of the many varieties of sarcomata, those occurring most commonly in the liver are the small and large round-celled and the melanotic variety, the latter often being secondary to sarcoma of the choroid coat of the eye. These grow rapidly, causing a widespread destruction of the liver-structure, with a change in the size and shape of the organ that is often demonstrable by palpation. E. R. Axtell reports a case in which at the *postmortem* the upper two-thirds of the liver revealed an entire absence of hepatic structure, and consisted of three tumor-masses which, on microscopic examination, were found to be small round-celled sarcomata. On section the tumor is seen to be of firmer consistence than the surrounding liver-tissue, and presents a dark, grayish-white, striated appearance. If the growth be of the pigmented variety, patches of a deep black or of different shades of pigment may be scattered throughout the mass. Metastasis is rapid and widespread (lungs, kidneys, heart, skin), as is shown by the fact that other organs are invariably found involved at the time of the growth and development of the sarcoma in the liver.

The *symptoms* are those of mechanical obstruction, and consist of gastro-intestinal disturbances due to passive congestion, edema, and ascites. Anemia and emaciation may become marked late in the disease, but cachexia does not develop. The passage of an intensely dark-colored urine (melanuria) has been noted in some cases. Secondary nodules may appear on the skin-surface.

The *diagnosis* can often be made from the primary growth (melanosarcoma of the choroid or sarcomata of the lymphatic glands) and from the rapid development of the tumor. From *carcinoma* of the liver melanosarcoma may be distinguished by the presence of ocular symptoms, particularly blindness of one eye, by the rapid widespread metastasis, the melanuria, perhaps, and by the absence of a true cancerous cachexia.

The *prognosis* is absolutely fatal, and the *treatment* merely palliative.

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[1] *Deutsche Zeitschrift für Chirurgie*, February, 1900.

X. DISEASES OF THE SPLEEN.

DISEASES of the spleen are mostly secondary to other diseases, the consideration of which embraces an appropriate description of the associated splenic disorders. The intimate relation between the spleen and blood accounts for the frequency with which this organ is involved in many of the blood-diseases.

DISLOCATION OF THE SPLEEN.

(*Floating Spleen.*)

Etiology.—This may be either congenital or due to the increased weight of an enlarged spleen, to tight-lacing, to relaxation of the ligaments, or to traumatism, and is met in splanchnoptosis. Carcinomatous enlargement of the left lobe of the liver caused it in a case I saw recently.

Symptoms.—The symptoms are vague, and are the result of pressure. By *physical examination* we discover with the touch the spleen as a mobile tumor pendant from the left hypochondrium; the tumor is superficial, blunt-edged, and notched on its anterior border, and may be replaced by the hand nearly in its normal position. On percussion over the splenic area the normal dulness is found to be absent.

Diagnosis.—It is important to distinguish between *floating spleen* and simple enlargement; also between the former and *movable kidney*.

The **prognosis** is guarded as to cure, though favorable as to life. Twisting of the pedicle of a floating spleen has been followed by strangulation and consequent necrosis.

The **treatment** must be mechanically supportive, consisting of pads and bands. Splenectomy has given excellent results.

SPLENIC HYPEREMIA.

Acute or **active hyperemia** may be found as the result of the acute infectious diseases, giving rise to the *acute splenic tumor*, or as the result of amenorrhea, or of injuries and inflammation (*circumscribed hyperemia*). The organ is uniformly enlarged (except in the last-named cases), and is darker in color and softer in consistence; the capsule also is tense. This condition merges insensibly into *acute splenitis*.

Chronic or **passive hyperemia** is due to some mechanical obstruction of the portal circulation caused by tumors, cardiac, hepatic, and pulmonary disease, and pylephlebitis. The spleen is enlarged, firm, dark-red in color, and the capsule is somewhat thickened.

The *symptoms* are vague, and may consist of simply a sense of weight, fulness, and pressure, and some tenderness in the left hypochondrium. In cases of extravasation of blood and rupture of the spleen the symptoms of intestinal perforation, hemorrhage, and collapse may supervene.

On *physical examination* the edge of the spleen may be palpated below the margin of the ribs. The percussion-dulness is increased in area, especially downward and forward, and may encroach upon the slightly-curved umbilico-axillary "resonant line."

The detection of acute or chronic splenic hyperemia (enlargement) is often of invaluable aid in the diagnosis of the causative disease.

The *prognosis* and *treatment* are embraced in those of the disease causing the congestion.

SPLENITIS.

Definition.—This term comprises acute and chronic (hypertrophic) proliferative splenitis and suppurative inflammation.

Pathology.—Next to the kidneys, the spleen is the favorite seat of metastatic inflammation and *embolic infarction.* Splenitis, due to a benign embolus originating in the left side of the heart or from the aorta above the splenic arteries, is usually circumscribed to a zone of sero-hemorrhagic infiltration about the resultant infarct. The latter is hemorrhagic at first, and later becomes particolored or *mixed*, and is of a yellow color, owing to partial fatty degeneration; still later it may become whitish and remain as a wedge-shaped (the base being peripheral), cheesy (necrotic softening), or even calcareous mass or as a fibrous cicatrix. Infection of the infarcts by pus-micrococci leads to the development of *small abscesses*, and the trabeculæ surrounding the latter may give way until several abscesses or one large pus-sac may be formed.

Perisplenitis generally follows, and sometimes with adhesions attached to adjacent hollow organs, as the stomach and colon, through which the perforating abscess may discharge its purulent contents. An unfortunate termination is the bursting of the abscess into the peritoneal cavity; a more fortunate ending results in an external opening. In acute splenic tumor there is an active congestion, with round-cell infiltration and some proliferation of the splenic cells. The spleen is moderately enlarged, dark, soft, pulpy, and friable.

In cases of intense vascular engorgement, as in the acute splenic tumor of severe typhoid fever, intermittent fever, and epilepsy (during the paroxysm), *hemorrhagic extravasation* may occur, and there may finally be even a rupture of the capsule and a passage of the blood into the peritoneal cavity. In chronic splenic tumor there is a persistent hyperplasia of the splenic cells, and frequently also of the trabecular cells, minus the acute engorgement. *Cirrhosis of the spleen* (chronic interstitial splenitis) differs characteristically from that of other organs (as the liver and kidneys) in that there is *enlargement instead of contraction.* Added to the increase in the size of the spleen, there are in both forms of chronic splenitis thickening of the capsule, patches often of old perisplenitis, and a slaty color of the tissues, with more or less pigmentation.

Etiology.—The disease probably never starts primarily in the spleen itself. *Acute proliferative* or *hyperplastic splenitis* (*acute splenic tumor*) is seen as the result of the acute infectious diseases (typhoid, typhus, relapsing, malarial fevers.) *Chronic proliferative splenitis* (*chronic splenic tumor*) is due to chronic malarial infection or repeated acute attacks, to splenic anemia, chronic passive congestion of the spleen, and leukocythemia. The leukemic spleen represents a somewhat different form of chronic proliferative splenitis from the ordinary forms. *Acute suppurative splenitis* (*abscess*), either diffuse or circumscribed, is usually secondary to infectious (pyogenic) emboli, as in ulcerative endocarditis and pyemia. Again, as the result of simple valvulitis of aortic thrombosis, *embolic infarction* of the spleen may be found, which may soften and break down in abscess-formation from subsequent infection. Abscess of the spleen may also follow traumatism, perforation of a gastric ulcer, and the extension of adjacent inflammation.

Symptoms.—These are indefinite or absent in most cases. Usually there is no pain or tenderness unless perisplenitis exists. Considerable enlargement of the spleen may be attended with a *sense of weight, tension*, or *distress* in the left hypochondrium, and perhaps by slight *dyspnea.* Any *suppurative fever* present will most probably be disassociated from the idea of abscess of the spleen, provided the local signs of pus be absent. *Sudden pain* appearing in the gastric region, followed by the *vomiting* of *pus* and *blood*, in the course of an infectious disease, with *splenic enlargement*, may be due to the rupture of an abscess of the spleen. *Ascites* may also be present.

The **physical examination** may reveal some bulging on inspection, and a fluctuating tumor may be palpated. The enlargement may be sufficient to enable the examiner to feel the notch in the spleen, and also the anterior and lower borders, reaching even to the umbilicus and to a level with the pelvic brim. The percussion-dulness is correspondingly increased.

Diagnosis.—This may be made from a consideration of the physical signs in conjunction with a study of the primary disease. In cases in which pus is suspected an exploratory puncture may clear the diagnosis. The splenic inflammation is rather an aid to diagnosis than a condition essentially needful of recognition in itself, by reason of its almost invariably secondary nature.

Differential Diagnosis.—Acute suppurative splenitis might be mistaken for *gastric* or *pancreatic disease;* but the previous history in the former, as contrasted with that of the latter affection, conjoined with the local symptoms that are more or less characteristic of the organ involved, will generally furnish an accurate means of differentiation.

The huge enlargements of chronic splenitis may be confounded with *hepatic, renal, omental,* or *ovarian growths.* Here a careful, discriminating observation of the constitutional state and of the physical signs is requisite for a diagnosis; even then it is often puzzling and difficult to attain. Care must be taken that splenic enlargement be not assumed when a *large pleural effusion* on the left side is causing the depressed lower border of the organ to be felt. Finally, *fecal accumulation in the splenic flexure* of the colon may be mistaken for moderate enlargement of the spleen. The former gives an irregular, doughy tumor, tympanites, vomiting, and a history of constipation alternating sometimes with diarrhea; there is no increase in the splenic area of dulness.

Prognosis.—This will depend upon the primary systemic condition in most cases. Abscess of the spleen is always a very grave complication, the main danger consisting of rupture and fatal peritonitis. Even in acute splenic tumor of a violent type there may be a hemorrhagic extravasation so severe as to burst the capsule. Chronic splenitides are not in themselves grave disorders.

Treatment.—This is to be directed mainly at the causative condition. Quinin and arsenic are often useful in the malarial form, and the chalybeates, iodids, and ergot have been recommended for the various chronic splenic enlargements. Abscess must be treated by splenotomy and drainage. Splenectomy may be useful in certain cases of simple hypertrophy, but records show only about 20 per cent. of recoveries from the operation. The state of the patient must be well considered. Splenectomy is probably never justifiable in leukemic enlargement.

AMYLOID DEGENERATION OF THE SPLEEN.

(*Sago Spleen.*)

THIS occurs as a part of the cachectic condition attending amyloid or waxy degeneration of other organs (liver and kidneys). The condition develops in the course of cases of prolonged and wasting discharges (phthisis, empyema, suppurative ostitis, syphilis, chronic peritonitis, chronic entero-colitis). The spleen is, as a rule, greatly enlarged, putty-like, and rotund. The capsule is tense and glistening. There are two forms of waxy degeneration—namely, the so-called "*sago*" *spleen* and the *diffuse waxy* or *lardaceous spleen*. In the former the Malpighian bodies are chiefly affected and appear on section like sago-granules; in the latter the whole splenic pulp, and even the trabeculæ, are more or less degenerated, and on section the spleen appears pale, smooth, and homogeneous (boiled-ham appearance). This may be but a late stage of the "sago" spleen.

The *symptoms* are those of general cachexia, and the *diagnosis* rests upon the detection of an enlargement of the organ associated with evidences of amyloid disease in other organs.

The *prognosis* is unfavorable, and the *treatment* does not differ from that indicated for the underlying and causative disease.

MORBID GROWTHS OF THE SPLEEN.

THE principal new-growths are the granulomata, as tubercles and syphilitic gummata; also secondary carcinoma, melanotic sarcoma, and hydatid and other cysts. Lymphadenoma (*e. g.* in leukemia) may be included among tumors of the spleen (Stengel).

These affections of the spleen are all of rare occurrence, and are not readily, if at all, discoverable during life. They are of no clinical or therapeutic interest apart from the general or primary disease. It may be stated that *carcinoma* of the spleen is always secondary; it may be diagnosticated by a physical examination, showing the organ to be enlarged, with the unmistakable signs of the primary carcinoma, as of the stomach. Secondary sarcoma is more common, and is recognized by an irregular enlargement and the presence of a primary tumor.

Syphilitic gummata of the spleen are often associated with amyloid degeneration and enlargement.

RUPTURE OF THE SPLEEN.

THIS may occur as the result of an intense hyperemic engorgement, both in splenitis from the rupture of an abscess and from traumatism. In the acute splenic tumor of typhoid fever, in malaria, and during an epileptic paroxysm, rupture of the capsule has been known to occur on account of the extravasation of blood. The *symptoms* are usually mistaken for those of intestinal perforation with internal hemorrhage. The *treatment* is palliative.

XI. DISEASES OF THE PANCREAS.

ACUTE PANCREATITIS.

INVESTIGATIONS of late years have rendered it probable that this disease is not so rare an occurrence as was formerly presumed, when it was not so readily recognized, owing partly to insufficient clinical and pathologic data, and partly to an indifference as to its existence.

HEMORRHAGIC PANCREATITIS.

Pathology.—The pancreas is enlarged, usually firm, and somewhat chocolate-colored. Irregular areas show the circumscribed as well as the diffused form of hemorrhagic infiltration of the interstitial fat-tissue, with thrombosis of the pancreatic veins in some cases (Day). There is also an infiltration with round-cells of the interlobular tissues. Some cases are examples of degeneration (non-inflammatory). The adjacent tissues may also be found to be hemorrhagic, as the mesentery, mesocolon, omentum, and perinephric tissues. The gastro-intestinal mucosa may be hyperemic, ecchymotic, or in a slightly catarrhal state. Evidences of a localized peritonitis (*peripancreatitis*) are not frequent, though they should be looked for.

Disseminated fat-necrosis is quite commonly associated with hemorrhagic pancreatitis. Small areas of a peculiar (tallow-like) substance, ranging from the size of a miliary tubercle to that of a pea or even larger, are found scattered in the fatty interlobular pancreatic tissue in the omentum, mesentery, and sometimes in the abdominal fat. H. U. Williams and Kätz and Winkler, from experimental researches, conclude that some substance in the pancreas, probably the fat-splitting ferment, is capable of causing changes similar to fat-necrosis. Flexner's experiments render it probable that the escape of pancreatic secretions into the peri- and parapancreatic tissues is the origin of the fat-necrosis.

Mention should be made here of the fact that as the result of the infectious fevers we find the pancreas showing diffuse, parenchymatous, and granular degenerative changes. Chiari has also recently pointed out the fact that *postmortem* digestion is very frequent in the pancreas.

Etiology.—Most of the cases reported have occurred in men, and in persons past fifty years of age. An especial *vredisposition* to the disease seems to be the result of cases of severe and obstinate dyspepsia (gastro-duodenal), alcoholism, glycosuria, gall-stones (Fitz), and traumatism. Hemorrhage into the pancreas may lead to subsequent pancreatic inflammation. It is seen occasionally *postmortem* in cases of acute tuberculosis, of the specific fevers, and of septico-pyemia. The *direct cause* is probably an infection through the ducts of the gland. Flexner[1] injected acids, alkalies, and bacterial cultures into the duct of Wirsung and the interstitial tissue, and produced hemorrhagic, suppurative, and necrotizing pancreatitis, often with fat-necrosis.

Symptoms.—The *onset* is sudden and violent. It is characterized by *excruciating, deep-seated pain*, usually in the epigastrium or between the xiphoid and umbilicus. There are also *nausea* and *severe retching* and *vomiting, constipation*, and speedy *collapse*, ending fatally within a few days (second to the fourth—Fitz). The vomitus may consist at last of slimy mucus or dark blood. *Fever* is generally slight at first, though it may touch 103° or 104° F. later. *Dyspnea* and a rapid, feeble *pulse*, with jactitation and *marked anxiousness* or an *afebrile delirium*, may perhaps be present. In some cases there may be *diarrhea*, with thin and watery stools containing free fat. Instances may be repeated in which, owing to the coincident presence of gall-stones, there may be *jaundice* and *colicky pains* over the right hypochondrium. The jaundice, however, may sometimes be due to a considerable swelling of the head of the pancreas, which presses upon the common bile-duct. *Tympanites* occurs in a majority of the cases. *Hiccough* and *albuminuria* have also been noted. The pain and collapse may be due either to a circumscribed peritonitis or to pressure upon the solar plexus.

Diagnosis.—This is at all times difficult, since many or all of the symptoms enumerated may be present in other affections. A careful inquiry into the previous history is important. The sudden development of an intense, deep-seated pain in the epigastrium, followed by vomiting, collapse, abdominal distention, with circumscribed resistance in the epigastrium, and the presence of constipation and slight fever, should point strongly to hemorrhagic pancreatitis. The detection of free fat in the dejections, and the discovery of scattered points of tenderness, when they occur, are also of corroborative significance.

Differential Diagnosis.—The temperature is apt to be higher and the pain and tenderness less localized and more constant in *peritonitis*. Fecal vomiting would indicate *obstruction of the bowel*. Here also we may determine the patency of the bowel by injection or inflation. Intestinal obstruction is of comparatively rare occurrence in the epigastrium, where the pain and distention of acute pancreatitis are localized; there are likely to be present more marked and general tympany, including the flanks, and a circumscribed distention of the intestinal coils.

In perforating gastric or duodenal ulcer there is a history of pain after eating, hemorrhages from the digestive tract, and of anemia or chlorosis occurring more commonly in the young female.

Corrosive poisons may be excluded by the history of the case and by an examination of the mouth and vomitus. *Hepatic colic* must be excluded; the pain is intermittent, and referred more to the right side than in pancreatitis. There is in pancreatitis also an early collapse.

Acute gastro-duodenitis is characterized by fever, by a history of injudicious eating, followed by mild inflammatory symptoms within a few hours, and by an absence of the sudden prostration and collapse so common to hemorrhagic inflammation of the pancreas.

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[1] "Experimental Pancreatitis," Festschrift in honor of Wm. Henry Welch.

Prognosis.—Acute hemorrhagic pancreatitis in most cases ends in death. It is but fair to state, however, that in view of the ease with which the disease may be overlooked it is quite possible that certain cases of a less severe type may often recover; in these the recovery has been said to follow an entirely different affection. Osler reports a case diagnosticated as one of intestinal obstruction in which abdominal section was performed and recovery followed. Thayer and Körte have also reported cases of cure in which a celiotomy decided the diagnosis.

Treatment.—This must needs be merely palliative and symptomatic. The treatment as for shock by the use of external heat and of warm saline solutions (by injection), hypodermics of morphin, atropin, strychnin, and of diffusible stimulants may probably be of some avail.

SUPPURATIVE PANCREATITIS.

Pathology.—The suppuration may be diffuse, with numerous small abscesses; more commonly a single abscess may exist in the head or body of the pancreas, which may be considerably enlarged and the glandular structure extensively destroyed. The abscess may communicate with peripancreatic areas of suppuration, or it may evacuate either into various organs (stomach, duodenum, peritoneal cavity) or externally. Pylephlebitis and hepatic abscess or pyemia may follow. A disseminated fat-necrosis is not found so frequently as in hemorrhagic pancreatitis.

Etiology.—Most of the cases collected have occurred in adult males *prior* to fifty years of age. Intemperance, debauchery, gluttony, trauma, and dietetic errors are among the *predisposing* causes. Infection takes place through the ducts, or from extension of neighboring septic foci.

Symptoms.—These may be *acute*, *subacute*, or *chronic*. Acute cases occur less frequently than the latter, there being a marked tendency of the disease to chronicity. **Acute suppurative pancreatitis** usually begins *suddenly*, with *severe epigastric pain*, *vomiting*, *hiccough*, *chills*, and an *irregular pyemic temperature*, *progressive tympanites* (at times limited to the left half of the abdomen), and perhaps acute splenic enlargement. *Constipation* may be followed later by *diarrhea* (sometimes fatty), and slight jaundice or glycosuria may appear. *Prostration* is generally great, and death may set in within one week from the onset.

Not seldom, however, the *course* is prolonged to three or four weeks, the symptoms persisting with progressive emaciation and final exhaustion. *Rupture* of the circumscribed peritoneal abscess, evidenced by copious dejections in which the sloughing pancreas has been found, and rapid diminution in the size of the abdomen, may take place.

Again, the onset may be less severe, and yet the case progresses steadily downward with little pain, slight suppurative fever, anorexia, anemia, and gradually increasing debility, lasting for months or even a year, and ending in anasarca and death. A pancreatic swelling is rarely *palpable*.

Diagnosis.—A limitation of the pain and tympany to the epigastrium, irregular fever, and the constitutional indications of suppuration are probably all that can be relied upon in arriving at a diagnosis. In fact, the diagnosis is hardly made *antemortem*.

For the differentiation from *circumscribed* peritonitis, *perforative gastric ulcer*, and *acute obstruction of the bowel*, *vide Hemorrhagic Pancreatitis.*

The **prognosis** is fatal and the **treatment** surgical.

GANGRENOUS PANCREATITIS.

Pathology.—The pancreas may be found in various stages of necrosis, depending upon the duration of the disease. It may be a dark-brown, flabby, soft, friable, shreddy, and putrid mass, with areas of hemorrhagic infiltration and yellow softening, and surrounded by a dirty-greenish, thin, purulent, and ichorous fluid. In cases lasting for from three to seven weeks the gland may be found completely sequestrated, lying in the omental cavity as a small, thin, brownish-black, shreddy, and foul-smelling detritus, soaked in a dark-colored, ichorous, and purulent fluid. The peri- and para-pancreatic tissues are usually involved with acute peritonitis. Splenic thrombo-phlebitis is commonly associated, and, as in the hemorrhagic, so in the gangrenous pancreatitis, disseminated fat-necrosis is frequently seen. The sloughed pancreas may be discharged into the intestine.

Etiology.—Males and females seem to be equally liable, and persons past thirty years of age are most commonly affected. Hemorrhagic pancreatitis is the most frequent antecedent of the gangrenous form. The disease may result also from perforative ulceration of the gastro-intestinal or biliary tract, or from the extension of a catarrhal inflammation of those tracts into the pancreatic duct (Fitz). Traumatism is a cause, though rare.

Symptoms.—These are essentially the same as those of hemorrhagic pancreatitis. The *course* may last longer, however, so that death may not occur until the second or fourth week, preceded by symptoms of collapse. The necrotized pancreas may be discharged per rectum, followed in some cases by recovery.

CHRONIC PANCREATITIS.

Pathology.—The pancreas is indurated from an increased development of interstitial fibrous tissue. The secreting glandular substance may be nearly obliterated, or at least considerably changed, and, owing to occluding pressure upon the duct of Wirsung, small pancreatic cysts may be formed. Interstitial hemorrhages and peripancreatic adhesions may be present. In *chronic suppurative pancreatitis* there may either be several small circumscribed abscesses or one large pyogenic cyst.

Etiology.—Chronic pancreatitis may be due to several attacks of the acute disease. Chronic inflammation of the pancreatic duct—often secondary to gastro-duodenal catarrh—is the most frequent cause. Persistent inflammations of contiguous structures and frequent irritation from biliary calculi may excite it. Chronic alcoholism and syphilis probably lead to this disease. The condition may be limited to a part of the organ.

Symptoms and Diagnosis.—The symptoms are hardly indicative of the disease. For a long time the symptoms of *chronic gastric catarrh*, frequently attended by *diarrhea*, may compose the clinical picture. Later there may be paroxysms of *deep epigastric pain*, and slight *fever*, with *great anxiety* and *faintness*, occurring at irregular intervals. Some *ascites* and occasional *jaundice*, due to pressure, may be observed. The detection of *free fat* in the dejections (without jaundice), and the occurrence of *glycosuria* and *lipuria*, would be of distinct diagnostic value. The presence of glycosuria in this variety of pancreatitis probably indicates an extreme degree of destruction of this gland (Fitz). A *cachectic*, emaciated appearance may be associated. *Circumscribed resistance* on palpation in the pancreatic area has been noted. Evidences of hepatic cirrhosis or of chronic renal and arterial disease may be present.

The **prognosis** is grave. It is to be recollected, however, that the greater portion of the gland may become functionless, as the result of progressive fibrous change, without much impairment of the general health.

Treatment.—The major treatment is *dietetic*. Fats and starches, since they demand the pancreatic ferment for their conversion, are to be interdicted, or, if permitted, are to be, so far as may be, artificially digested by the administration of tablets of pancreatin and soda (gr. v–x—0.324–0.648) fifteen or twenty minutes after meals. Malt diastase, combined with alkalies, should also be tried. Becher has found that carbonated waters stimulate pancreatic secretion in dogs, and hence their use may be advised. Minced pancreas promotes the digestion of fat (Abelmann).

PANCREATIC HEMORRHAGE.

(*Pancreatic Apoplexy.*)

It is only in recent years that this fatal affection has been clearly isolated and defined, and mainly through the observations of Zenker, Draper, and especially Fitz.

Pathology.—The pancreas may or may not be enlarged; it may also be soft and friable. The hemorrhage is apt to occur into circumscribed areas of the gland, particularly its head, the interstitial and subperitoneal tissues both usually being the seat of hemorrhagic infiltration of a dark-purple color. Extensive hemorrhage may be found in the omentum, transverse mesocolon, in the retroperitoneal fat-tissue, and surrounding the kidney even. Hemorrhages into the adjacent mucous surfaces have been detected in some cases. Secondary reactive inflammations and necrosis are commonly noted.

Etiology.—*Slight hemorrhages* into the pancreas may be found that are secondary to excessive chronic passive congestion or to hemophilic or purpuric cases, and they may be met with in acute infective diseases. These have, however, no clinical import. The precise cause or causes of *marked hemorrhage* into the pancreas are not known. Most cases have occurred in males (in 25 of 34 instances collected by the writer), and in adult or advanced middle life (the age in 13 of 30 cases having been over forty-five years). In the majority of cases the previous health was apparently good. Traumatism may be a direct cause. Again, some local vascular weakness or lesion (*e. g.* necrosis), superinduced by alcoholic habits or a rich diet in an atheromatous person; or some corrosive action of the pancreatic secretion may operate as causes. There was a history of chronic alcoholism in 12 of 18 cases (66.6 per cent.) in which reference to this habit was made. Severe *symptomatic* pancreatic hemorrhage is dependent on a variety of primary affections of the gland —*e. g.* acute pancreatitis and carcinoma.[1]

Symptoms.—The patient may have been in apparently robust health when the attack comes on with *sudden* and *startling* gravity. The most prominent early symptom is *intense pain* located in the epigastric region or in the lower chest, together with a sense of *constriction*. *Nausea* and *vomiting* may be associated, and the latter is usually obstinate and gives only temporary relief. *Tympanites* may also occur. There are early and constant *general evidences* of *internal bleeding*—an anxious countenance, restlessness, depression, yawning, pallor, cold sweat, a lowered surface-temperature, and a small, rapid, and weak pulse. *Prostration* and *syncope* follow, and death ends the case in from half an hour to twenty-four hours. Death is caused by reflex paralysis of the heart, due either to some coincident vascular affection, or to pressure, perhaps upon the solar plexus and semilunar ganglion (Zenker).

My table of cases includes 24, exclusive of 16 reported by Fitz, in which the condition led to speedy death from shock or possibly from compression of the solar plexus. Owing to its apparently "idiopathic" character in many cases, its suddon development, and quick destruction of life, pancreatic hemorrhage assumes intense medico-legal interest and importance. A small percentage of cases reach death more gradually.

Diagnosis.—Given the sudden-developed signs of a concealed internal hemorrhage, with pain referred distinctly to the epigastrium, and vomiting and rapid collapse, a *probable* diagnosis may be made.

Treatment.—This consists in relieving the pain by opiates and in overcoming the collapsed condition by free stimulation.

CARCINOMA OF THE PANCREAS.

Pathology.—Primary carcinoma is the more frequent variety. It is of the scirrhous form in most cases, and usually involves the head of the gland, which may attain to the size of a child's head. Not rarely the adjacent organs are found affected, either by direct or metastatic extension of the disease, or by the pressure of the growth; the liver, peritoneum, stomach, portal vessels, bile-ducts, and aorta may thus be involved. The pancreatic duct may be occluded, so as to form retentoin-cysts. In 1000 autopsies at the Johns Hopkins Hospital there were 5 cases of secondary carcinoma of the pancreas. Simple extension of carcinoma of the stomach or of the duodenum may involve the pancreas.

Etiology.—*Men* past forty years of age are most liable to carcinoma of the pancreas, though it has been met with even in the *new-born*. Mirallié has collected 113 cases of primary carcinoma of this viscus (Fitz).

Symptoms.—These are scarcely ever sufficient to indicate the disease with any certainty. There are usually a *stubborn dyspepsia*, a *progressive loss of flesh* and *strength*, *anemia*, and a *dull*, or sometimes *neuralgic*, *epigastric pain*. *Nocturnal paroxysms* of pain are common, and are often accompanied by signs of *collapse*. In some cases *vomiting* and

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[1] "Pancreatic Hemorrhage," *Journal American Medical Association*, Dec. 2, 1899, by the writer.

diarrhea are present. The *stools* may be light in color and greasy, and may contain blood. There may also be found an abundance of *undigested muscular fibers* in the stools in the absence of diarrhea; this is an incontestible proof of faulty pancreatic digestion. Steatorrhœa is not commonly present. Among the *pressure-effects* due to carcinoma of the head of the pancreas there may be *jaundice* (due to pressure upon the common duct), which persists and "is associated with an enlargement of the liver and gall-bladder." *Ascites* may appear from pressure on the portal vein. Chylous ascites, from pressure upon the thoracic duct, has been observed in 2 cases. The inferior vena cava may be compressed, causing *dropsy* of the lower half of the body; also the duodenum, followed by *gastrectasis* or by signs of *intestinal obstruction.* Carcinoma of the tail of the pancreas may be a cause of *hydronephrosis* of the left kidney, from pressure upon the ureter (Fitz). *Marasmus* and the *cachexia* grow from bad to worse, and emaciation may become so extreme as to permit of a satisfactory *palpation* of the *tumor*, which occupies a position near the median line above the umbilicus. Very often, however, the growth is too deep-seated to be felt, being palpable in about one-third of the cases only. *Glycosuria* may be associated.

Diagnosis.—Carcinoma of the pancreas is probably present in a given case in which there are rapid and progressive emaciation, deep-seated epigastric pain, muscular fibers in the stools without diarrhea, late persistent jaundice, and enlargement of the gall-bladder, and the detection of a deeply-situated, fixed, and firm tumor in the region of the gland. The quantity of indican in the urine is diminished.

Aortic abdominal aneurysm may be mistaken for carcinoma of the pancreas because of the transmission of the aortic pulsation to the tumor. But in aneurysm the impulse is expansile instead of to and fro, and the contact is neither so sharp nor so sudden; moreover, the cancerous cachexia is absent in aneurysm, and the history of the case may be clearly indicative.

It is sometimes difficult to differentiate a malignant tumor of the pancreas from *carcinoma of the pylorus*, of the *stomach*, or of the *transverse colon* or *omentum;* the following points will help in the differentiation of the former two:

CARCINOMA OF THE PANCREAS.	CARCINOMA OF THE PYLORUS.
The tumor is deep-seated and fixed; later it becomes slightly movable. It is not associated with gastric dilatation.	The tumor is more freely movable, and is usually associated with dilatation of the stomach.
Symptoms of chronic dyspepsia manifest themselves.	There are more marked gastric symptoms.
The vomitus is bilious; rarely contains blood; often is that of gastrectasis.	There is "coffee-ground" vomitus; it is seldom bilious.
HCl is present, while there is an absence of lactic acid.	HCl is absent from the gastric contents; lactic acid is present.
The stools contain undigested muscle-fibers. There is an absence of pancreatic secretions. The urine may contain sugar.	Usually the bowels are constipated, with occasional diarrhea. The stools are black after a hemorrhage. The urine does not contain sugar.
There is usually jaundice; sometimes ascites is present.	Usually there is no jaundice or ascites.
Inflation of the stomach shows the absence of a pyloric growth.	Inflation shows the presence of a pyloric tumor.
The course is more acute. Death may occur within a few weeks or months.	The course is more chronic, and secondary growths appear in the liver.

Neoplastic growths of the transverse colon are also more often superficial, and are movable and definable with the palpating fingers. There are symptoms of intestinal obstruction here, and inflation of the colon will show the relation of the tumor to the gut. In carcinoma of the colon the urine generally contains an increased amount of indican.

A discussion of the **prognosis** and **treatment** of carcinoma of the pancreas is obviously unnecessary.

Other Tumors of the Pancreas.—Exceptionally, sarcoma, adenoma, and lymphoma occur. Sarcoma is very rarely primary. Secondary nodules are somewhat more frequently observed. According to Körte, of 10 cases of tumor of the pancreas operated upon of late years, 6 recovered.

PANCREATIC CYST.

Pathology.—Pancreatic cysts may be single or multiple, and large or small. When large they develop chiefly to the left of the median line. Single cysts may grow to an enormous size, containing as much as several gallons of fluid. The contents may at first consist simply of retained pancreatic juice, and usually the liquid is dark gray or dark brown, alkaline, and hemorrhagic or albuminous. A hematoma may be converted into a serous cyst. The specific gravity is from 1010 to 1024. Atrophy of the pancreas may ensue. Examined *microscopically*, the contents reveal leukocytes, red blood-corpuscles, oil-drops, fatty degeneration of the epithelium, and crystals of fatty acids and cholesterin.

Etiology.—Cysts of the pancreas may be due to occlusion of the pancreatic duct or its branches by compression from within or without the gland. They may also be due to tumors, to impaction of biliary or pancreatic calculi, to cirrhosis or angular displacements of the gland, or to the obstructive swelling from extension of catarrh of the bowel (Krecke). Of 121 cases collected by Körte, 33 were traced to traumatism. Lloyd suggests that the cysts that follow local injury are in reality instances of encysted peritonitis involving the lesser omentum or that portion covering the pancreas (*pseudocysts*). Cysts of the pancreas usually occur in adults—in 66 of 116 cases in the third and fourth decades of life (Körte). Railton, however, met a case at six months of age.

Symptoms.—*Pain* may be absent, or it may occur as colicky paroxysms, referred either to the epigastrium, the left hypochondrium, or even the left shoulder. *Jaundice* and *ascites* are present in large tumors. *Vomiting*, *constipation*, or *fatty diarrhea* (rarely), with undigested muscular fibers in the dejecta, or clay-colored, pasty, and offensive stools, may be present. *Albumin* and *sugar* may be found in the urine. *Emaciation* is not infrequent. *Intestinal hemorrhage* may occur and recur. A late and constant symptom is a *feeling of pressure* in the epigastrium. Rarely there is increased salivary secretion (pancreatic salivation). Occasionally all subjective symptoms are absent, and these cysts may temporarily disappear.

On physical examination a smooth, elastic, lobulated *tumor* is discovered in the region of the pancreas if the growth is moderate in size.

Sometimes a very large cyst develops in a remarkably short space of time—*i. e.* in a few weeks. When very large in size fluctuation is easily elicited. It may be slightly movable in the grasp and during inspiration. It usually presents between the stomach and transverse colon an area of dulness, and unless the tumor be of large size it is surrounded by tympanitic resonance of deeper timber above than below. Auscultation may reveal a murmur caused by compression of the aorta. When the cyst attains enormous dimensions the usual *mechanical pressure-effects* are produced. Körte points out that cysts without any inflammatory or traumatic etiology may exist for many years or even decades.

Diagnosis.—The diagnosis rests on the typical physical signs—the discovery on palpation of a smooth, elastic, lobulated, or rounded tumor that is slightly movable, and on percussion of a dull area that is not continuous above with the spleen- and liver-dulness. Resort has been had to filling the stomach with air and the colon with water (after purging), and thus proving by *palpation* the deep-seated situation (behind the stomach and omentum) of the tumor. If pancreatic fluid be obtained from the supposed cysts, it will digest albumins and emulsify fats. This test is not wholly reliable, however. A pancreatic cyst may be mistaken for an *ovarian cyst*, for *renal tumors* (*cysts*), *dropsy of the gall-bladder*, and *retroperitoneal sarcoma* (*Lobstein's cancer*). The differentiation must be made by a careful study of all the points in the case.

The **prognosis** is good under proper treatment—incision and drainage. Of 31 reported cases thus treated, only 2 proved fatal.

PANCREATIC CALCULI.

Pathology.—These are grayish-white, rounded concretions, consisting principally of calcium carbonate. The calculi may be as fine as dust or as large as an almond. Among their pathologic effects are fistulous communications with the colon, peritoneal cavity, and stomach; also cystic dilatations of the duct and abscess-formation. Atrophy of the organ and carcinoma due to irritation of the stones may be associated.

Etiology.—Pancreatic calculi presuppose a catarrhal condition of the pancreatic duct, with retention of secretion, anomalies of the pancreatic secretion, or the presence of cysts or some other form of obstruction of the pancreatic duct. The condition is comparatively rare.

The **symptoms** are developed when, during the passage of the stones along the duct to the duodenum, the latter excite inflammation. In consequence, paroxysms of *pain* occur (*pancreatic colic*) that are usually attributed to gall-stones, and we are often unable to differentiate the two conditions. The *radiation* of pain along the lower left costal border to the back rather than to the right side, and possibly the *detection of free fat in the stools* or *glycosuria*, may aid markedly in the diagnosis.

The finding of characteristic *calculi* in the stools is entirely confirmatory. Minnich has reported a case in which the calculi were found in the stools following an attack of colic.

The **prognosis** is mainly dependent upon the associated lesions and upon certain sequelæ—pancreatic cysts and chronic pancreatitis.

The indications for **treatment** do not differ materially from those of hepatic colic. Surgical intervention should be considered.

XII. DISEASES OF THE PERITONEUM.

ACUTE PERITONITIS.

Definition.—An acute inflammation of the peritoneum. The condition may be primary or secondary. Clinically, two varieties—general and circumscribed—are recognized, while, pathologically, the disease is classified according to the nature of the exudate.

Anatomic and Physiologic Peculiarities.—The surface area of the peritoneum is quite extensive, being almost equal to that of the skin. Fluids of all sorts are rapidly absorbed by the peritoneum, and thus, if they be poisonous, constitutional infection is speedily propagated.

Pathology—Upon opening the abdomen in **acute generalized peritonitis** vascular injection both of the serous covering of the intestine and of the parietal layer is observed. Even in the most recent cases the coils of intestine may be feebly glued together by lymph, while in those of longer duration the adhesions are quite firm. As in the analogous inflammation of the pleuræ or pericardium, we distinguish the following forms pathologically: (*a*) A *plastic* or *fibrinous*, in which there may be also a small amount of serum present. (*b*) *Sero-fibrinous* (inflammatory ascites), chiefly characterized by considerable sero-fibrinous fluid; additionally, the coagulated fibrin forms a covering for the parietal and visceral layers of the peritoneum. (*c*) *Purulent* (most frequent). The amount of inflammatory exudate varies greatly, and is frequently enormous, exceeding 30 liters (quarts). Putrefactive decomposition of the pus may occur, especially in cases due to gangrene of the gut or to puerperal peritonitis (violent forms), giving rise to a thin fluid that is grayish-green in color, is sometimes distinctly sanious, and ill-smelling. Offensive gases are present with relative frequency. These may come from the intestinal canal, following the track of perforations; or they may be due to decomposition of the purulent exudate. (*d*) *Hemorrhagic*. This form is common in cases that are of a cancerous or tuberculous nature, and in subjects whose vitality has been lowered by various other affections. It may also be of traumatic origin.

Changes in the Intestines.—The effect of acute peritonitis is to thicken the coats by inflammatory edema; soon the musculature is paralyzed. An associated catarrh of the mucosa of the intestine is sometimes observed.

The different pathologic varieties above described may be limited to definite portions of the peritoneal sac, when they are termed "encapsulated" or **localized acute peritonitis** (*vide supra*). In localized purulent peritonitis further extension of the process is arrested by the rapid formation of circumscribed adhesions due to the exudation of lymph; there are also undoubted instances of circumscribed, aplastic peritoneal abscesses. The milder forms of limited plastic and sero-fibrinous peritonitis pursue a slower course than the purulent variety, and commonly lead to the development of firm adhesions (*adhesive peritonitis*). Since the histologic changes in acute peritonitis do not differ from those observed in other inflammations of serous membranes, the reader is referred to the section on Pleurisy (p. 545) for their consideration.

Etiology.—The irritants causing acute peritonitis may be—(*a*) Or-

ganized inflammatory agents (*bacteriologic irritants*). These may be *specific* or *non-specific*. Among the non-specific agents are the pyogenic bacteria. Grawitz has shown that the latter can only cause peritonitis under certain conditions: they excite the disease when injected into the peritoneal cavity or when poured out from the diseased or injured membrane more rapidly than the peritoneal tissue can dispose of them; also when the epithelial layer has from any cause been removed. Absorption may be interfered with, while the pyogenic micrococci continue to enter from the bowel or other viscera in great numbers. Unfortunately, the clinical practitioner often meets with cases of peritonitis in which these pyogenic organisms are the only positive agents. These essential conditions obtain when the membrane is wounded by the perforation of gastric and intestinal ulcers, and also in perforation of the gall-bladder, in rupture of the liver, kidneys, and spleen, when the latter are the seat of abscesses, and, with uncommon frequency, in appendicitides, in purulent inflammation of the ovaries and of the Fallopian tubes. "There are instances in which peritonitis has followed rupture of an apparently normal Graafian follicle" (Osler). These perforative forms of peritonitis are at the same time the most serious and the most important. "Death may result from the injection into the peritoneal sac of putrid liquid if the dose be large enough; but it is practically the same whether the fluid is injected into the blood-stream at once or allowed to find its way into the peritoneal cavity, and the result follows nearly as quickly in the one case as in the other" (Moullin). The rapid absorption of liquid substances gives full opportunity for the phagocytic action of the white blood-corpuscles.

Among specific organic irritants the *tubercle bacillus* deserves especial mention, though, as before intimated, a discussion of its characteristics is not in place here. The *streptococcus pyogenes* is probably responsible for the most violent forms of peritonitis (*e. g.* those occurring in puerperal sepsis and post-operative varieties). The *staphylococcus pyogenes aureus* (or *albus*) has also been found in such instances.

The *bacterium coli commune* (always present in the intestinal tract) is frequently the leading factor in peritonitis of intestinal origin, and usually in association. The streptococcus is often present also in these cases. In 12 cases of primary peritonitis, 11 were instances of mono-infection; and in operations upon the peritoneum (not involving the intestine), 25 of 33 cases were mono-infections, the staphylococcus aureus being present alone in 12 and the streptococcus in 5 (Flexner). Occasionally other organisms, as the *pneumococcus*, the *bacillus of Friedländer*, or the *bacillus pyocyaneus*, *typhosus*, and *proteus*, the *gonococcus*, the *aërogenes capsulatus*, and the *anthrax bacillus*, have been found.

Multi-infection is quite common. Some contend that all forms of peritonitis are due to bacteria or their toxins.

(*b*) **Chemical Irritants.**—These are rather numerous and varied, though all produce their effects in one of two ways. First, the irritant acts upon the membrane, exciting an exudation of lymph. Here constitutional intoxication is secondary. Secondly, the chemical irritant may be quickly absorbed, and produce systemic intoxication immediately (rare).

(*c*) **Mechanical irritants**, as, for example, a hernia, which may produce a localized peritonitis.

(*d*) Peritonitis may be due to a direct extension of infective processes from the intestinal tract or other adjacent organs (*secondary peritonitis*). The bacteria often penetrate the intestinal wall and gain the peritoneum by way of the lymph-channels. The disease is often secondary to pleurisy, the irritants passing through the diaphragm along the course of the lymphatics. Peritonitis may be secondary to chronic Bright's disease, gout, and arterio-sclerosis; in such cases the special irritants probably reach the membrane through the general circulation. In most cases this variety is *protective* in character and results in local adhesions.

(*e*) The disease is very rarely *primary* (*idiopathic*). It has been attributed to exposure to cold or wet (rheumatic peritonitis). These so-called idiopathic cases are probably instances of cryptogenetic infection.

Clinical History.—The symptoms are both of a *local* and a *general* nature. In sthenic cases of perforative peritonitis they occur simultaneously with great severity and suddenness. On the other hand, in asthenic cases, such as occur frequently in those already afflicted with some serious disease that is apt to result in perforation (for example, typhoid fever), both the local and constitutional symptoms are more or less overshadowed by the disturbances due to the primary affection. Again, circumscribed abscesses of the peritoneum often lead to diffuse suppurative peritonitis, and the change may take place so insidiously as to defy detection. These anomalies from the typical onset and course of the disease are by no means exceptional, and should ever be distinctly borne in mind by the physician.

Local Symptoms.—Among these, *pain* is the chief. At the commencement its seat of greatest intensity corresponds, in most instances, with the seat of origin. Hence the character of the causal disease is often betrayed by the location of the chief pain. For instance, if this appears in the region of the stomach and is referred to the back or shoulders, we would think of gastric ulcer; if in the ileo-cecal region, of appendicular disease; and so on. It follows that quite commonly the severest pain is in the lower half of the abdomen. It is almost constant, increases in severity, and finally becomes general and excruciating; it is also much increased by deep respirations, by pressure, and by bodily movements. It remits, but does not intermit, though it may be slight in asthenic cases. Here the patient is excessively weak, while his sensibilities are greatly blunted. Gastro-intestinal symptoms are prominent, more particularly *vomiting*, which occurs early and is apt to recur with comparative frequency. It may follow the taking of food, though, in my own experience, it has more frequently taken place spontaneously; the *vomitus* then consists of a watery liquid greenish in color and contains mucus. In rare instances the matter vomited is a dark-brown liquid. Vomiting may sometimes be absent, however, owing to the presence of marked *asthenia* or *coma*. *Eructations* are common, and *constipation* is usually present and may become exceedingly obstinate. On the other hand, there may either be *diarrhea* throughout the disease, or this symptom may precede the constipation. Constipation is due chiefly to paralysis of the musculature of the intestine. It is to be ascribed to an increased peristalsis due to intestinal catarrh. The *apex* of the heart is elevated; the *tongue* at first is furred and moist, and later it is dry, brown, and often fissured.

Constitutional Symptoms.—At the onset the patient in *sthenic* cases is seized with a *rigor* that may be repeated. The *shock* sustained by the nervous system in acute peritonitis is most intense; the *temperature* rises immediately, though it does not, as a rule, attain to a high level, and it frequently presents a curve more or less characteristic of suppuration. The rectal temperature is often relatively high; the respirations are shallow and much accelerated, ranging from thirty to forty per minute. We have, as factors to account for this increased frequency, (*a*) a crowding upward of the diaphragm, (*b*) the greatly enfeebled heart, and (*c*) the pain occasioned by throwing the diaphragm into action. The *heart* early becomes excessively weak, and, as would be expected, the *pulse* is rapid, small, and soft. The pulse toward the close becomes exceedingly frequent (130 to 150 beats per minute) and is almost imperceptible; during the early stages the pulse ranges from 100 to 130. Other evidences of more or less marked *circulatory collapse* soon manifest themselves. The patient wears an anxious facial expression, the eyes are sunken, the features pinched and cool, the lips cyanotic, and the extremities are likewise cold and somewhat livid. The patient invariably assumes the *supine position*, with the lower extremities drawn up, so as to lessen the tension of the abdominal muscles, and thus to secure the greatest possible comfort. The *urine* is scanty and high-colored, and contains indican. There may be a retention of urine; though oftener, perhaps, micturition is more frequent than in health. Marked *nervous symptoms* do not appear; indeed, the mind usually remains quite clear to the close. Moderate delirium, however, which sometimes gives way to mild stupor, is met with occasionally. In connection with these facts it should be pointed out that in the *asthenic form* of acute peritonitis the constitutional features differ from those above described. The *temperature* is usually subnormal (except in the rectum), the pulse is exceedingly feeble and running, and the signs of collapse are well marked from the onset.

Physical Signs.—*Inspection* reveals the gradually increasing abdominal distention, that frequently becomes excessive if the intestinal walls are more or less completely paralyzed. Often the amount of effusion soon becomes large, when the abdomen appears widened. The degree of distention bears a definite relation to the severity of the inflammatory process, and is in inverse ratio to the development of the abdominal muscles. Thus, when the latter are poorly developed or greatly relaxed the expansion is enormous. On the other hand, when they are strong the muscles are apt to be quite tense, permitting of a relatively slight enlargement; the abdomen may even show a small concavity, in which case the walls are of a board-like hardness. The cardiac apex-beat is displaced upward and outward, occupying the fourth interspace.

Palpation elicits extreme tenderness, more particularly in the vicinity of the umbilicus. Rigidity of the abdominal wall is the most important symptom in perforative peritonitis (J. C. Wilson). In not a few instances of acute peritonitis have I been able to detect a distinct friction-rub. *Percussion* gives at first an exaggerated tympanitic note. There is often an absence of liver-dulness in the mammary line, and rarely also it is absent in the mid-axillary line. In pneumo-peritoneum, resulting from perforation of the gut or stomach, we often meet with an absence of liver-dulness, especially when a large purulent effusion coexists. Again, a great diminution in, or even the total effacement of, the dull area may be caused by coils of intestine forcing their way up between the anterior surface of the organ and the inner surface of the abdominal wall. When air is present within the abdominal cavity and the patient lies upon his right side, splenic dulness disappears from displacement by the air. The lower level of cardiac dulness is as high as the fifth rib.

By means of *percussion*, sooner or later, fluid effusions are usually detectable in sthenic cases. On the other hand, there may be in markedly asthenic cases an amount of liquid exudation present that is often too small to admit of detection. When the effusion is considerable in quantity, there is dulness on percussion over the most dependent parts; when tympanitic distention is excessive, however, even a copious effusion may be so effectually hidden as to elude discovery in this way. I have elsewhere reported one such instance.[1] When the decubitus can be altered, the line of dulness will be found to be movable, but the degree of mobility varies exceedingly, depending upon the extent of the peritoneal adhesions present. The effused material is partly contained in pouches, giving rise to areas of circumscribed dulness.

Course and Prognosis.—Asthenic forms of diffused peritonitis are perhaps invariably fatal. Though the local symptoms and signs are not marked, the characteristic evidences of collapse or of general septicemia appear and grow in intensity to the end. The *duration* in sthenic cases rarely exceeds one or two days; in asthenic cases it is longer, lasting from four or five to six or eight days. Death sometimes occurs quite suddenly, owing to cardiac exhaustion or primary shock. The clinical peculiarities and the course of an individual case are greatly influenced by the etiology. Acute generalized peritonitis arising from perforative appendicitis, from perforation of a gastric ulcer, puerperal sepsis, or from external injuries, is usually of a violent form and ends fatally. Prompt operative intervention, however, is powerful in saving life in a small percentage of the latter class. When the disease is traceable to rheumatism or exposure, recovery may take place. A case occurred in my own practice in which acute sero-fibrinous peritonitis with considerable effusion was associated with acute articular rheumatism and organic lesions of the aortic segments; the patient recovered.

Peritonitis in Children.—Syphilitic peritonitis may be congenital, and septic peritonitis caused by an inflamed cord may be met in the new-born. In children the most common causes are trauma and appendicitis.

The *symptoms* differ from those presented in the adult. However severe the pain the child merely utters a short cry or whine. Constipation and vomiting are less conspicuous features. Meteorism is pronounced and fever high. Convulsions not rarely occur. The condition is extremely grave in young children.

LOCALIZED OR PARTIAL PERITONITIS.

(*Circumscribed Peritonitis: Visceral Peritonitis.*)

This is a localized form of inflammation of the peritoneum that is coextensive only with the serous covering of single organs, and involves a limited portion of the membrane. Hence, to the various forms of circumscribed peritonitis such terms as perihepatitis, perisplenitis, perinephritis are applied. The condition is found in its most important form in *appendicitis*, but the points that are characteristic of localization in this disease have been mentioned elsewhere (*vide* Appendicitis, p. 823). Localized peritonitis may also be caused by carcinoma.

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[1] *International Medical Clinics*, vol. iii., second series, p. 82.

Pyo-pneumothorax subphrenicus is the term applied to a circumscribed peritoneal abscess containing air, situated between the liver and diaphragm. The condition is described under the heading Acute Perihepatitis (p. 879).

Local pelvic peritonitis (perimetritis) is the most frequent variety, and is secondary, as a rule, to inflammation about the uterus, Fallopian tubes, and ovaries. The leading causes are tuberculosis, puerperal septicemia, and gonorrhea. F. Billings points out that when abdominal rigidity is absent in this form rectal examination will disclose rigidity of the pelvic muscles.

Symptoms.—The *local* clinical features do not differ from those described under the diffuse form, but their area of distribution is more or less strictly limited to definite regions. By eliciting the *physical signs* with care fluid collections are sometimes demonstrable. The *constitutional symptoms* are likewise similar in character, though less marked than those belonging to the diffuse variety. There may be *rigors*, and *pyemic symptoms* appear, together with the temperature-curve peculiar to this condition. The danger of involvement of the general peritoneal cavity as the result either of rupture or of an extension of septic inflammation is a constant menace. When the peritonitis remains localized these cases may pursue a subacute or even a chronic course, though grave constitutional disturbance finally develops.

Diagnosis.—In attempting to diagnosticate acute generalized peritonitis it is of great importance for the clinician to keep in remembrance the sthenic and asthenic forms of the affection. The character and gravity of the symptoms, both general and local, are such as to render the diagnosis of the sthenic form entirely easy. Especially valuable features are the *constant pain*, the *marked tympany*, the *excessive tenderness under pressure*, and the *vomiting at intervals of a greenish fluid material*. Of equal importance are the serious general disturbance previously depicted, and in particular the *cool, sharpened features* and the *ever-increasing weakness* and *rapidity of the pulse*. These clinical manifestations clearly foreshadow cardiac exhaustion or fatal collapse. When the cases are not seen until the advanced stage has arrived, however, the diagnosis presents many difficulties. Nothing is now more important than the consideration of the history from the time of onset, also of the previous history, with a view to determining the point of origin and the probable cause of the disease (usually some such primary disease as appendicitis or gastric ulcer).

The smaller number of cases belonging to the adynamic type are from the start extremely difficult of diagnosis. Here a history that is clearly indicative, the presence of moderate tenderness, and augmented tension of the abdomen, with profound collapse, would point to this condition. It must, however, be confessed that a positive opinion is often unwarranted, owing to the absence of the more characteristic clinical indications.

General Differential Diagnosis.—*Hysteric peritonitis* (so-called) simulates in every leading particular the genuine form so closely as to make the distinction an insurmountable difficulty, unless there be present other hysteric manifestations. In my experience the tenderness has been out of proportion to the gravity of the constitutional disturbance. The patient often complains bitterly before the abdomen has been touched; on the other hand, when his attention has been otherwise engaged firm and prolonged pressure can be made.

Acute generalized peritonitis occasionally supervenes on *typhoid fever.* In such cases it is caused either by perforation of the intestine or by a direct extension of inflammation from a deep typhoid ulcer. If consciousness be retained, sudden severe pain, tenderness followed by excessive tympany, and signs of collapse will establish the diagnosis. Peritonitis, however, develops more often in those grave cases of typhoid that are attended with coma, marked meteorism, and profound adynamia, and under such conditions it often remains unrecognized (*vide* Typhoid Fever, p. 35).

In *acute enteric catarrh* the meteorism and sensitiveness under pressure are usually less pronounced; the disease also lacks the marked constitutional symptoms of acute peritonitis. The pain is colicky, is characterized by exacerbations, and even intermits in entero-colitis, while it is constant in peritonitis. The pain in acute enteric catarrh is often followed by diarrheal stools.

Intestinal colic is distinguished from peritonitis by the flatulence, the borborygmi, and the wandering pain in the absence of all other phenomena.

Rheumatism of the abdominal muscles excites pain, which, however, is superficially located (the disease being an affection of the muscular layer), and is frequently associated with rheumatism in other parts of the body. There may also be a clear history of previous rheumatic attacks.

Tubal pregnancy (after rupture) has also been confounded with acute peritonitis, but its differential diagnosis is fully discussed and must be looked for in special works on gynecology and obstetrics.

Rupture of an abdominal aneurysm and *embolism of the superior mesenteric artery* are also conditions that give rise to peritonitic symptoms—meteorism, recurrent vomiting, and collapse—all appearing with explosive violence.

Acute generalized peritonitis in its symptomatology bears a close resemblance to acute intestinal obstruction, and the discriminating points have already been tabulated (*vide* p. 835).

Prognosis.—This is less grave than in the diffused form, and recovery may often be expected. Timely surgical intervention, particularly if a tendency to spreading be shown, may render the outlook encouraging or even lead to prompt recovery.

Sequelæ.—If recovery should take place, the inevitable result is the formation of adhesions and fibrous bands, the contraction of which may cause constriction of the bowels, bile-ducts, and other structures.

Treatment.—**Hygienic and Dietetic.**—The patient should be placed in the position that will give him most comfort, and should be kept absolutely undisturbed. The sick-room should be of good size and well ventilated; the temperature should be kept at from 65° to 70° F.

18.3°–21.1° C.). The diet demands careful attention. Pancreatized milk in accurate dosage (℥iv–vj—128.0–192.0—every two hours) should be administered, and if the stomach will not bear the introduction of nourishment, recourse should be had to rectal alimentation. Other liquid food-stuffs, as meat-juices and egg-white (diluted), may also be allowed. In asthenic cases alimentation must be generous, although solid articles of food are to be avoided.

Medicinal.—Formerly the opium method of treatment, first instituted by the late Alonzo Clarke, was the one followed by the bulk of the profession. His plan was to administer ½ gr. (0.0324) of morphin or its equivalent (gr. ij—0.129) of opium, and repeat the dose every two hours until the respirations were lowered to ten or twelve per minute. The pupils were then observed to be contracted, the pulse from 76 to 80, the pain relieved, and peristalsis arrested. This latter effect was obtained, even though in the case of some patients larger doses of opium than here indicated were necessary; in others smaller doses sufficed. The bowels were absolutely let alone. It is explained that in favorable cases the bowels moved spontaneously at the end of one week, and that the patient then entered upon convalescence. This method of treatment is at present adhered to only by the ultra-conservative element of the profession. Among those authors who recommend opium as the most efficient measure in the treatment of this disease many still advise against the immoderate dosage previously so generally administered, but employ just enough to keep the patient well under the influence of the drug.

The leading mode of treatment to-day consists in the use of *saline purgatives*, exhibited in divided doses in concentrated solution (ʒj–ij—4.0–8.0—every two or three hours) until the irritating intestinal contents, should any be present, are removed, and additionally several copious serous discharges occur daily. Purgatives do good when given in this manner principally by causing a rapid exosmosis of serum from the blood-vessels of the intestines, by removing the collateral edema, and by indirectly relieving the congestion of the peritoneum, thus promoting a rapid absorption through the latter membrane. By increasing the peristaltic movement they also diminish the danger of peritoneal adhesions. The remedies to be selected will depend upon two primary considerations: *first*, the etiology of the individual case (whether a communication has or has not been established between the peritoneal cavity and the bowel), or an intra-peritoneal abscess or abscess-cavity in one of the abdominal viscera; and *secondly*, the type of the case, whether sthenic or asthenic. If perforation is known to have taken place or the occurrence of this accident is strongly suspected, a prompt laparotomy, followed by the free use of salines, is the proper treatment. After the *primæ viæ* have been looked after by the surgeon, salines, for the reasons before stated, are to be used with a free hand. For a like reason they are most serviceable in peritonitis due to extension of the inflammation, and also in the puerperal form. If the patient be robust, with a full, tense pulse, we may begin the treatment by the use of mercury, the best preparation being calomel, exhibited in fractional doses (gr. ss—0.0324—every hour) until its purgative action is obtained; this is to be followed by the salines. The object of the *calomel treatment* is to defibrinate the exudations as well as the blood of the patient. Certain observers advocate the use of small doses of calomel, and seek to avoid any purgative action of the remedy. Indications demanding the *opium treatment* do not often present themselves. When, however, the vital forces are profoundly depressed, as shown by the symptoms of collapse, and there is not even a reasonable suspicion of perforation, then opium should be tried, but not in the heroic doses formerly advocated. Enough only should be given to obtain the physiologic effect of the drug in a moderate degree. Again, if the evidences of perforation into the general peritoneal cavity are complete and competent surgical skill is not at hand, large doses of morphin are imperative, with a view to relieving pain, keeping the patient at absolute rest, and sustaining the heart against the exhausting effect of shock. The bowels should now be relieved by simple large enemata. The value of serum-therapy in this disease is as yet uncertain (Fowler).

Local Treatment.—At the onset, if the patient be strong, from twenty to thirty leeches are to be applied to the abdomen. The ice-bag or ice-poultices are often of distinct service in the earlier stages. Later, in localized peritonitis, blisters may be useful, although objectionable in the event of surgical intervention becoming necessary. In cases in which meteoric distention is not great I have also made repeated trial of an ointment containing ung. ichthyol (℥j—32.0); ung. belladonnæ (℥ss—16.0); ung. hydrarg. (℥ij—64.0); this is applied to the entire abdomen thrice daily.

In order to relieve the *tympany* turpentine stupes are serviceable. I have also had favorable results from the insertion of the long rectal tube (soft esophageal) well up in the colon. *Large* high enemata should be used; and turpentine combined as follows may prove efficacious:

℞.	Turpentine,	ʒij (8.0);
	Ox-gall,	ʒij (8.0);
	Milk of asafetida,	℥iv (128.0);
	Warm water,	℥vj (192.0).

Puncturing the abdomen with a hypodermic needle in order to relieve tympany, as recommended by Loomis, may also be resorted to, though I have had no personal experience with this measure.

Pain.—No matter what general plan of treatment is pursued, the pain must be relieved by opium in some form. *Thirst* is to be relieved by chipped ice, over which a little brandy may be sprinkled. The *vomiting* is best treated by carbonated water exhibited in small quantities, or by iced champagne similarly administered. One-drop doses of creosote are also of value. For the systemic collapse, as well as for combating thirst and vomiting, I can warmly recommend saline infusion, to be repeated if needful.

CHRONIC PERITONITIS.

Definition.—Chronic inflammation of the peritoneum.

Pathology and Etiology.—The anatomic characters presented by different cases are greatly varied, though for convenience of study they may be considered under two divisions (as in the acute form): 1. *Local;* 2. *General.* The latter may be (*a*) **Adhesive,** when the peritoneal layers are inseparable and indistinguishable, with an obvious thickening, and the intestinal coils are everywhere seen to be grown together. The cause is usually a previous acute attack, and, doubtless with great relative frequency, the condition is produced by the *acute progressive form* (Mikulicz), which is *localized* at the start. Rheumatism is also an occasional factor, and a mild variety of adhesive peritonitis, confined, as a rule, to small circumscribed areas, may be engendered by the trocar used for tapping in ascites.

(*b*) **Proliferative Peritonitis.**—"The essential anatomic feature is great thickening of the peritoneal layers, usually without much adhesion" (Osler). It has been found to be associated with cirrhosis of the stomach, liver, and other abdominal organs. The amount of liquid effusion, varying in composition from serum to pus, is usually moderate, and it may, owing to adhesions, be loculated. The omentum is sometimes rolled up in the form of a massive cord, with its long axis in the transverse direction. In an autopsied case of chronic peritonitis apparently secondary to hepatic cirrhosis I observed in the thickened membrane numerous small hard nodules that were at the time regarded as being tuberculous in nature. It is to be pointed out, however, that a number of cases of pseudo-tuberculosis have been recently reported. In several of these an operative incision was followed by recovery, and this was put down as a cure of tuberculous peritonitis till the microscope showed the nodules to be fibrous. Among *etiologic factors* chronic alcoholism stands first. In one case that I saw, acute followed by chronic rheumatism seemed to be the only assignable cause. The condition is sometimes secondary to chronic nephritis, to syphilis, or a general fibroid process.

(*c*) **Cancerous Peritonitis.**—Quite often in connection with cancerous growths in the peritoneum a well-marked peritonitis is evident. There may be a liquid exudation, which is apt to be bloody and chylous.

(*d*) **Chronic Tuberculous Peritonitis.**—This is the most important variety. The inflammatory lesions are quite pronounced, as a rule, and lead to marked thickening of the layers—changes that are to the naked eye identical in appearance with those noted under the preceding forms, but which on histologic examination show the presence of tubercles and caseous degeneration. The amount of liquid effusion varies within wide limits, and is usually blood-stained. The frequent association of hepatic cirrhosis with tuberculous peritonitis should be remarked. From tuberculous peritonitis, tuberculosis of the peritoneum is also to be distinguished clinically; the latter may be acute or chronic, and the lesions consist in the deposit of various sized tubercles without much collateral inflammation. Acute and chronic tuberculosis of the peritoneum have received due consideration in their appropriate place (p. 312).

(*e*) **"Chronic Hemorrhagic Peritonitis."**—This term should be limited in its application to that form first described by Virchow, in which the peritoneum is at intervals partly covered by a membrane of new connective tissue that alternates, as it were, with layers of hemorrhagic extravasation. A similar condition results from the frequent use of the trocar for ascites.

Chronic Localized Peritonitis.—This is of frequent occurrence, and is confined most commonly to the serous covering of the spleen, liver, and certain portions of the bowel, particularly of the appendix. The condition results in the formation of *firm adhesions*, with matting of the intestinal coils and fibrous bands. It is usually the *sequel* of localized acute peritonitis occurring in connection with inflammatory diseases of the different abdominal organs.

Symptoms of the General Forms.—Whether chronic peritonitis follows the acute form or not, it always develops insidiously. Most cases remain quite obscure, and not a few are totally devoid of clinical manifestations. The patient may complain of disorders of the *alimentary tract*, and especially of *constipation*. On the other hand, *diarrhea* is observed in tuberculous peritonitis from associated intestinal ulceration. Rarely pressure, from the traction force of the adhesions, on the common duct or portal vein gives rise to obstructive *jaundice*, or *ascites*, as the case may be. I saw an instance recently in which compression of the veins leading to the lower extremities caused unilateral *edema*. *Subjective abdominal sensations*, as uneasiness, oppression, heat, and pain (often colicky in character), are experienced. Sometimes pain is entirely absent.

General symptoms appear, though they are quite vague as a rule. An irregular fever, hectic in type, is occasionally observed. Later, increasing general weakness, emaciation, and general nervous disturbance become rather prominent clinical features. Some of these phenomena, however, may be due to associated affections. When the peritonitis is tuberculous we frequently see clinical evidence of the causal disease in other parts of the economy (*vide* Tuberculous Peritonitis, p. 312).

Physical Signs.—*Inspection* usually shows the belly to be slightly, though unequally, enlarged. As in acute peritonitis, so here, we find the belly flat, or even concave occasionally, with great tension of its walls. Fluctuation is sometimes obtainable over limited areas only, since the fluid is not free, but encapsulated. The coiled-up and shrunken omentum may be *palpable* as a sausage-shaped mass, and thick bands of adhesion may also not rarely be felt, in different places, as hard, uneven masses simulating neoplasmata. The *percussion-dulness* varies considerably with the amount of effusion, its arrangement, the degree of peritoneal thickening, as well as with the character and locality of the fibrous bands. It follows that in some cases irregular areas of tympanitic percussion-resonance and of dulness are to be found side by side scattered over the abdomen. Obviously, too, changing the patient's posture would not give movable dulness, owing to sacculation of the fluid. A marked sense of resistance is experienced on percussion over the dull area. *Friction-fremitus* can sometimes be elicited, and less frequently *friction-sounds* also during forced breathing.

Symptoms of Chronic Local Peritonitis.—This condition is often entirely latent. When not so, the most characteristic indication is constant *pain*, distinctly colicky in nature and often quite intense.

The physical signs are negative, as a rule. Very rarely a resistant, ill-defined mass, corresponding with the seat of greatest pain, can be felt. A fibrous band may be so arranged as to form a snare through which a knuckle of bowel may pass, with resulting strangulation. Fitz's analysis of 295 cases showed 63 to be caused in this way.

Diagnosis.—That form of chronic peritonitis (serous or granular) most frequently seen in females at the commencement of puberty is hard to discriminate from *tuberculous peritonitis*, since the latter may be more or less latent. Tuberculous peritonitis is attended with fever, more pain and tenderness, and there is a more rapid accumulation of the exudate. Again, the general features, debility and loss of flesh, progress more rapidly than in granular peritonitis. The detection of conclusive evidence of the disease in persons closely related, or on physical examination of associated pulmonary or pleural lesions, would render the diagnosis of tuberculous peritonitis almost certain. In obscure cases the guinea-pig should be inoculated with the exudate (see Pleurisy, p. 545).

Course and Prognosis.—The milder varieties of simple chronic peritonitis may, though rarely, reach a favorable issue. In cases belonging to this category the disease takes a chronic course, and leads gradually to a condition of extreme debility, even if it does not, as is usually the case, materially shorten life. Tuberculous peritonitis has, until recently, been regarded as being almost uniformly fatal at the end of several months. Cures that must be attributed to the surgeon's work, however, are at present by no means uncommon. Rarely spontaneous cures also occur, particularly among children, in whom the disease is less serious than in adults.

Treatment.—The patient should be enabled to enjoy the benefits of good sanitary surroundings. Close attention is to be paid to the *diet*, the coarser vegetables and sweets being prohibited, since they increase the pain by exciting the production of gas. A change of air has improved the condition in several instances occurring in my own practice. The usual constipation may be relieved by simple enemata or by the use internally of the fluid extract of cascara sagrada. Tonics and alteratives, the latter with a view to promoting the absorption of the exudate, may also be employed, and I would recommend especially for this purpose the double iodids, as in the formula given in the discussion of Pleurisy (*vide* p. 560). In the early stages some degree of relief, or even a curative effect, may be secured by *local means*, as the application of equal parts of belladonna and iodin ointments until mild counter-irritation is produced. Ichthyol ointment is also serviceable. After all, however, little is to be gained from therapeutic measures, and it is to surgery that we must look for fresh triumphs in the treatment of this truly distressing complaint. Cases of chronic localized peritonitis with adhesions have been operated upon successfully by W. E. Ashton, H. A. Kelly, and others. Instances of chronic generalized peritonitis, whether tuberculous or not, in which the fluid effusion reaccumulates rapidly after repeated tappings, also furnish adequate indications for operative procedures.

ASCITES.

(*Hydrops Peritonæi; Dropsy of the Peritoneum.*)

Definition.—An accumulation of serum in the peritoneal cavity, resulting from stasis (obstruction) in the branches of the portal vein.

Pathology.—The quantity of liquid contained in the peritoneal cavity is quite variable, though it often amounts to several gallons. It is clear and transparent, or slightly opalescent, especially on standing, and the specific gravity ranges from 1010 to 1014. In color it often has a faint lemon-yellow tint; it may, however, be either distinctly yellow, brownish (in cirrhosis), bile-stained (as when jaundice is present), or slightly blood-stained. In reaction it is usually alkaline; very rarely it is either acid or neutral.

The ascitic fluid usually contains much albumin, resembling in this respect blood-serum, as would be expected from its source. The percentage of albumin may be approximately ascertained by noting the specific gravity of the fluid by the urinometer. Thus, in true ascites the specific gravity ranges from 1010 to 1014, and the variation in the percentage of albumin is from 1 to 2. In effusions due to *peritonitis* the percentage of albumin ranges higher (2.5–6 per cent.); hence the specific gravity ranges correspondingly higher (1015–1024). The standing specimen may show to the unaided eye a minute coagulum of fibrin. In the lowest layer of the fluid the microscope discloses leukocytes, red blood-corpuscles (in abundance when ascites is due to general venous stasis), fat-cells, endothelium, and cholesterin-crystals. In ascites the microscopic appearances of the peritoneum are usually normal, while in instances of peritonitis the membrane, including the subperitoneal fibrous tissue, is opaque and slightly thickened.

In the so-called *chylous ascites* the fluid resembles milk; it contains fat-droplets, a few lymphocytes, and sugar (Hodlmoser [1]). This condition may be associated with a collection of milky fluid in the left pleural sac, when there is thrombosis of the subclavian vein at the point at which the thoracic duct enters. The term *ascites adiposus* is applied to a milky fluid, in which the origin of the fat is the débris of degenerated epithelial cells, with few fat-droplets and no sugar (Quincke and Senator), to the exclusion of other morphologic elements.

In long-standing cases the abdominal and the thoracic organs become atrophied from pressure exerted by the dropsical fluid.

Etiology.—Among the chief causal factors are those that hinder or arrest the return of venous blood from the peritoneal membrane, as the following: (*a*) Pressure upon the branches of the portal vein within the liver, due to contraction of surrounding tissues, as in hepatic cirrhosis (including malarial atrophy—De Brun), syphilis of the liver, and cancerous infiltration. (*b*) Numerous conditions in the course of which pressure may be made upon the portal vein external to the liver, as enlargement of the glands in the fissure, carcinoma, hydatids, or abscesses connected with the liver. Tumors of any adjacent organs (*e. g.* pancreas) may produce it. (*c*) Thrombosis of the portal vein. (*d*)

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[1] *Wiener klin. Woch.*, 11 *Jahrg.*, No. 49.

Pressure upon the inferior vena cava after it receives the hepatic trunk (Roberts), or upon the latter itself, or the lymphatics. (*e*) The portal circulation is also impeded in chronic pulmonary affections (cirrhosis and emphysema) and heart-diseases (*e. g.* ascites due to "pericarditic pseudocirrhosis of the liver"—Pick). (*f*) A new growth in the peritoneum may compress the smaller veins lying in the membrane or the root of the mesentery. (*g*) Diminished resistance of the walls of the portal vessels, due to chronic affections that diminish the albuminous constituents of the blood and impair the nutrition of the peritoneum, as Bright's disease, carcinoma, syphilis, chronic malaria. (*h*) Chylous ascites is caused either by a leakage of the lacteals (due to ulceration, injuries, or the presence of filariæ) or by the obstruction of the thoracic duct (due to thrombosis, cicatrices, compression). Cases of lactescent ascites in which the fluid is milky (not chylous) have recently been reported. The nature of the fluid is as yet unknown. (*i*) Adipose ascites has for its direct cause fatty cellular degeneration, such as is found in carcinoma, tuberculous and other forms of chronic peritonitis.

Leyden has recently (1897) described an ameboid organism.

Symptoms.—Slight peritoneal dropsy gives rise neither to symptoms nor to abnormal physical signs. When the sac contains 1 quart (1 liter) of fluid or over, however, the first subjective *symptoms* that are due to the mechanical effect of the fluid appear. They are a sense of weight and fulness, with slight uneasiness. As the proportion of transuded serum becomes gradually increased these symptoms become more pronounced. There may in addition be a *dragging pain* in the loins, *gastro-intestinal disturbance* (meteorism, constipation), and dyspnea (owing to the resistance opposed to the descent of the diaphragm, resulting in compression of the lungs). The latter symptom is much increased upon exertion or on assuming the recumbent posture. Since the *heart* is displaced upward, an embarrassment of its action (rapidity and irregularity) would be expected. *Syncope* is not infrequent for similar reasons. Frequent *micturition* from pressure upon the bladder is common, and the kidneys, owing to compression of the renal vessels, secrete an *albuminous urine*, which is greatly lessened in amount.

Physical Signs.—After the serum has collected in considerable amount the physical signs afford characteristic evidence of the condition. From *inspection* we learn many valuable points: (*a*) The belly is uniformly prominent (the degree depending upon the amount of serum present), giving a rounded form. Changing the posture of the patient shifts the point of greatest pouching. (*b*) The skin is seen to be tense, smooth, and shining, and sometimes shows lineæ albicantes; the umbilicus commonly bulges forward; less frequently it is obliterated, and the surface-veins are often enlarged. (*c*) The thorax appears small, except at the base, where it is distended, and the ensiform cartilage is sometimes abruptly curled up. (*d*) The respirations are hurried and are of the thoracic type, the abdominal movements being slight or entirely wanting. As soon as the belly-walls become moderately tense *fluctuation* is elicited by placing the palm of the left hand vertically upon one side of the abdomen, and then, with the finger-tips of the right hand, tapping lightly the opposite side; impulses thus sent through the fluid will be distinctly felt by the hand in contact with the abdomen. When the dropsical fluid is small in quantity the patient should assume the erect posture during the examination. In palpating the solid organs (liver, spleen, abdominal tumors) when ascites is present, the tips of the fingers only are placed upon the skin, and then are suddenly "dipped," displacing the fluid, thus touching the solid organ or new growth. *Percussion* gives dulness, even to flatness, over all of that portion of the abdominal cavity occupied by the fluid. The upper level of dulness is not represented by straight transverse lines, but presents a concavity that is pointed to the head. The dulness is extremely movable, shifting as the patient's position is changed. When the decubitus is supine the most dependent portions of the abdomen give dulness. Again, if the patient be made to lie on either side, the opposite or uppermost flank will be found clear, the ascitic fluid always gravitating to the bottom of the sac. Tyson has observed that the flanks are tympanitic with considerable frequency in ascites, and my own experience has been similar, tympany over the head of the colon being almost constant, except in pronounced cases. Moreover, to obtain reliable results, if the layer of fluid be thin, the pleximeter finger is pressed lightly upon the surface, and the gentlest percussion only is allowable. In the cardiac region there is often percussion-resonance as high as the fourth rib, and occasionally a murmur may be heard at the base.

Diagnosis.—In order to arrive at a positive diagnosis a clear history of one or the other of the known causative conditions is requisite, joined with distinct evidence of the presence of fluid—viz. fluctuation and movable dulness. For the early diagnosis of ascites the patient should be placed in the knee-elbow position, when dulness can be readily elicited in the umbilical region.

The diagnosis of chylous ascites and ascites adiposus rests upon insecure ground unless aspiration be resorted to, although the presence of the causative conditions in the case may afford a basis for suspicions.

Differential Diagnosis.—Ascites is most apt to be mistaken for an *ovarian cyst*. The accompanying table presents the chief differentiating points:

ASCITES.	OVARIAN CYST.
Clinical	*History.*
General health is bad prior to the appearance of the enlargement.	General health is good before the development of the tumor; failure afterward.
History of disease of liver, lungs, heart, kidneys, or other organ.	Frequent history of dysmenorrhea, negative as to organic affections.
Swelling begins below and gradually extends higher; more noticeable when sitting than in the standing posture.	Swelling is unilateral at first, gradually becoming more central.
Physical	*Signs.*
Enlargement is symmetric, the abdomen being rounded and most prominent about the umbilicus; in the supine posture the abdomen flattens, with lateral bulging; the umbilicus is often pouched and thin.	Enlargement is asymmetric or irregular, unless the tumor be very large, when it may fill the entire abdomen. The greatest circumference is below the umbilicus, which never bulges.
Fluctuation is general from side to side and in a vertical direction.	Fluctuation is circumscribed, corresponding to the limits of the tumor.

Ascites.	Ovarian Cyst.
Physical Signs.	
No aortic pulsation felt.	Aortic pulsation is sometimes evident.
Vaginal examination often shows the uterus to be movable. A pouch may project into the vagina, but no cyst is detectable.	Vaginal examination shows the uterus to be displaced. A cyst may be felt and outlined in the pelvis.
When standing, the upper line of dulness is concave.	When standing, the upper line of dulness is uniform or convex.
In the supine position the flanks are especially dull, with tympany in front.	In the supine position dulness is still in front and the flanks are resonant.
Dulness is movable according as the position is altered.	The area of dulness is not varied by change of posture.
Character of the Fluid.	
Ascitic fluid has a specific gravity of 1010–1014, and is usually clear. It is of a pale straw-color.	Ovarian fluid has a specific gravity of 1018–1054. It is of a thick, turbid character, and the color is variable.

It should be recollected that large cysts may spring from other abdominal organs than the ovaries, as the *pancreas* and *liver;* the elimination of these latter conditions, however, does not, as a rule, offer marked difficulty. Ascites must be distinguished in practice from the exudation due to *chronic peritonitis*, and the points of differentiation have been arranged thus:

Ascites.	Chronic Peritonitis.
A previous history of organic disease of the liver, heart, kidneys, or other organ is obtainable.	There is a previous history of acute peritonitis, tuberculosis, or inflammatory diseases of the female pelvic organs; sometimes a history of injury.
No pain is experienced.	Pain is a prominent symptom.
The abdomen is symmetrically enlarged.	Abdomen is irregularly prominent, and rarely flat.
Fluctuation is general in the transverse or vertical directions.	Fluctuation is often limited to circumscribed areas due to loculation of fluid.
Palpation detects no hard masses of irregular prominence.	Palpation often detects resistant, uneven prominences.
Dulness is always movable upon altering the position of the patient.	Dulness often not changeable on varying the position, owing to adhesions.
The fluid consists of serum with few morphologic elements. It is limpid, with a specific gravity of 1010–1014, and is pale straw-yellow in color, often with a greenish tinge.	The fluid is either sero-fibrinous, seropurulent, or milky in nature. It is often viscid, and its specific gravity is 1018–1024. The color varies.

Over-filling of the bladder has also been confounded with ascites, and this organ has been tapped under the mistaken notion that the condition was one of dropsy of the peritoneum. If, however, the precaution be taken to catheterize the patient before tapping for supposed ascites, the error cannot, as it should not, occur.

Prognosis.—The duration of ascites may be for many months or even years. In most instances the prognosis is unfavorable, though modified by the character of the causal condition in individual cases. The immediate cause of death may be either syncope, asphyxia, pulmonary atelectasis from compression of the bases of the lungs by the diaphragm, or it may be the causal disease.

Treatment.—**Dietetic.**—The diet should be largely nitrogenous, light, nutritious, and given at stated periods with a view to maintaining the normal proportion of albuminous material in the blood.

Medicinal.—By means of therapeutic measures we should aim to accomplish two things: First, the improvement or cure of the original disease; and secondly, to relieve the chief symptoms by removing the ascitic fluid on which they depend. Though the causative affection is usually chronic and incurable, every effort should be made to remove or mitigate its pernicious activity in accordance with the principles laid down in appropriate portions of this work. Of medicines used to remove the transudation, hydragogue cathartics are most potent for good, and particularly when the ascites is due to cardiac or renal disease. Calomel and jalap in combination, or salines in full doses, administered after the Matthew Hay method, should be tried. Diuretics are also recommended, and English authors greatly praise copaiba and its resin as being among the best. The bitartrate and other salts of potash, either alone or in combination with juniper and digitalis, are of signal value. Sabrazles and Dion have recently recommended urea as an effective diuretic in ascites due to benign forms of atrophic cirrhosis. Equally important with the exhibition of the above remedies is the use of tonics, including hematinics, to promote the general nutrition of the patient. I have reported one instance, occurring at the Philadelphia Hospital, in which a cure was effected perhaps solely as the result of measures intended to assist the nutritive processes. In ascites due to cirrhosis of the liver recourse should be had to *paracentesis abdominis*, not as a last resort only, but "as a systematic method of treatment" (Roberts). A single tapping is rarely sufficient, and a repetition of the measure from time to time, until the collateral circulation is established, is to be advised and encouraged. In cases in which the transuded serum has rapidly re-formed after its removal by tapping, Southey's tubes, by means of which permanent drainage is secured, have been used with good results. Drummond affirms that ascites due to liver-cirrhosis can be cured, and has proposed an operation whereby adhesions between the abdominal contents and its parietes are secured, in which new blood-vessels are formed, thus establishing a collateral circulation (*vide* p. 899).

NEW GROWTHS IN THE PERITONEUM.

THE most frequent and important of the new growths of the peritoneum are (*a*) carcinoma and (*b*) tuberculous deposit and tuberculous peritonitis, the latter two having been already considered.

CARCINOMA OF THE PERITONEUM.

There occur the usual varieties—scirrhous, encephaloid, and colloid—the latter most frequently involving the omentum. Primary carcinoma of the peritoneum is rare. Primary endothelioma, however, is occasionally met with. It resembles true carcinoma in macroscopic as well as in microscopic appearances, though it is in reality to be ranked with the sarcomata on account of its origin. Carcinoma of the peritoneum is almost always secondary to carcinoma of the stomach, liver, or pelvic organs. The peritoneum may either be the seat of numerous small round miliary tumors, or, less commonly, of larger and distinctly nodular masses, the most extensive development being presented by the colloid variety. Cancerous peritonitis is commonly found to be an associated condition, and the retro-peritoneal lymph-glands not infrequently show cancerous development.

Etiology.—More cases occur in the female sex than in the male. Age has also a potent influence, most cases appearing late in life.

Symptoms.—When *primary*, carcinoma of the peritoneum is obscure during the early part of its course. Local *pain* and discomfort are complained of, and clinical evidences of the *cancerous cachexia* develop early, but these symptoms are not at first striking enough to be entirely characteristic. Later, however, the *nodules* can often be plainly felt (unless the liquid effusion be too marked), and the *ascites*, *loss of flesh*, *weakness*, and *anemia* are now sufficiently developed for. diagnosis. In the colloid variety ascites is often absent, the abdominal cavity being the seat of a large, semi-solid, non-fluctuating mass.

The *secondary form* usually follows carcinoma of the stomach or the ovaries, and the cachexia will have been developed before the peritoneum is secondarily involved in consequence of the presence of the primary growth. Hence, any symptoms referable to the general abdominal cavity are strongly suspicious. Among other *constitutional symptoms*, apart from those already mentioned, is fever (rarely absent), which may be due in small measure to the anemia, though in a greater measure to the associated peritonitis.

Physical Signs.—The abdomen protrudes if effusion be present or if the carcinoma be of the colloid form, though this cannot be set down as a uniform rule. Even when the tumor is large, dropsy of the peritoneum sometimes makes its detection impossible. On practising palpation after tapping, however, the nodules can be easily made out, either extending from side to side or being more or less localized and not adherent to underlying structures.

Differential Diagnosis.—It will be remembered that an oblong tumor lying in a transverse position below the stomach is met with in certain forms of *chronic peritonitis*. This offers the same physical signs that are presented by cases of peritoneal carcinoma, unless the tumor-masses in the latter affection be of considerable size. Carcinoma, however, is most apt to occur in persons past middle life, while nodular tuberculous peritonitis appears almost exclusively in children and young adults. Evidences of tuberculous disease elsewhere, past or present, and particularly suppuration about the umbilicus, would point to tuberculous peritonitis. Moreover, in all forms of abdominal carcinoma the inguinal glands are apt to be indurated and enlarged. *Proliferative peritonitis* usually gives a history of chronic alcoholism. The differentiation of *hydatid cysts* of the peritoneum from carcinoma depends upon the history of the case, the presence of hydatid fremitus, the finding of the hooklets in the fluid, the less rapid growth of the tumor, and the lessened amount of pain, fever, and cachexia in the latter disease. *Carcinoma of the intestine* may simulate somewhat the disease under consideration, but the signs of increasing stenosis, as evidenced by the colicky pain, the discharge of blood and pus with the stools, and the ribbon-like character of the feces, will serve to separate the conditions. *Retro-peritoneal tumors* (sarcomata) are discriminated with the greatest difficulty. As pointed out by J. D. Steele, in tumors behind the peritoneum the signs of intestinal obstruction, coupled with neuralgic pains or edema of the lower extremities from pressure upon their nervous and venous supply, are important discriminating features. Tumors of the peritoneum, however, whether of the omentum or mesentery, are movable, while those behind the peritoneum are generally fixed. In retro-peritoneal sarcoma, however, "the tumor may fluctuate and may move with respiration, or be movable by palpation." Omental tumors lie in front of the intestines (as can be shown by inflation of the bowel); mesenteric new growths sometimes have a coil of intestine in front of them. On the other hand, retro-peritoneal tumors are always crossed by loops of intestine (colon). Peritoneal tumors (particularly the omental) follow the movements of respiration, while the retro-peritoneal are, as a rule, immobile. The latter always cross to some extent the central long axis of the body, while the former may be confined to one or the other side. Finally, the only sure method of determining the character of tumors behind the peritoneum is by an exploratory celiotomy.

The **prognosis** is always unfavorable.

Treatment can accomplish nothing beyond a more or less complete relief from the distressing symptoms.

Other Tumors of the Peritoneum.—Primary sarcoma has been referred to above (p. 934). It produces larger or smaller areas of thickening of the peritoneum. Secondary sarcoma, the commoner variety, assumes the form of large nodular masses or of numerous miliary growths. The symptomatology has been given under Carcinoma of the Peritoneum.

Fibromata and *lipomata*—the former as fibroid nodules varying in size from a millet-seed to a split pea, the latter as localized overgrowths of fatty tissue showing great variation in their size—are among peritoneal and retro-peritoneal neoplasms. The lipomata, however, are the more frequent. Mr. Anderson points out that fibromata may merge, on the one hand, into the lipomata (*fibro-lipomata*); on the other, into the myomata (*fibro-myomata*). It is probable that lipomata usually spring from the retro-peritoneal tissue in the neighborhood of the kidneys and iliac fossa. Less commonly, however, they "originate in the subperitoneal tissues of the mesenteric or omental folds, where general fatty overgrowth in varying degree is frequently observed" (Allchin).

Peritoneal lipomata may be associated with extreme obesity, but this is by no means invariably the case. The *diagnosis* is rarely made, particularly in the female, owing to the close resemblance of these growths to ovarian cysts and other tumors found in connection with the female genitalia. They have been mistaken also for ascites, which is not rarely a symptom of fibromatous and lipomatous neoplasms. The *prognosis* is unfavorable, although, if early recognized, the tumors may be successfully removed.

PART VII.

DISEASES OF THE URINARY SYSTEM.

I. DISEASES OF THE KIDNEY.

MOBILITY OF THE KIDNEY.

(*Movable Kidney; Dislocated Kidney; Floating Kidney; Wandering Kidney; Ren Mobilis; Nephroptosis.*)

Definition.—A distinction is made between two common varieties of mobile kidney, according to the degree of displacement, as follows: (1) *Movable kidney*, the upper end of which can be felt during deep inspiration, and which can be pushed down in the retro-peritoneal space to the level of the umbilicus; (2) *Floating kidney*, which is freely movable below or beyond this point—*i. e.* possessing a larger arc of mobility. In the so-called *palpable kidney* the lower edge of the organ can barely be felt on deep pressure.

Etiology.—Although an anomalous position of the kidney is usually acquired, it may be congenital; in such cases the condition may be due to relaxation of the perinephric (peritoneal) tissues, the kidney thus having a mesonephron and floating freely about in the abdominal cavity. An abnormally long renal artery may also predispose to the development of a movable kidney.

Emaciation with a marked wasting of the fatty capsule in which the kidney is imbedded is a frequent underlying cause of movable kidney. Women are oftener affected than men, and relaxations from multiple pregnancies, tight-lacing and girdling, and traumatism (falls, heavy lifting, and the like) have frequently caused displacement and mobility of the kidney. Suckling[1] observed that a number of girls who served beer, and were therefore obliged to stoop and immediately stand upright with considerable frequency, were likely to have movable kidney. Heavy tumors of the organ, the pressure of adjacent tumors (as of the liver), and the traction of hernias may likewise cause the condition.

In *enteroptosis*, or Glénard's disease, in which there is a downward displacement of all the viscera, mobility of the kidney is often associated. Although either kidney, or even both kidneys, may be abnormally mobile, the right one is usually affected, probably owing to its anatomic position and to its relation with the liver above. Sometimes a floating kidney becomes fixed by peritoneal adhesions in an abnormal position, as in the right iliac fossa; an instance of this occurred in a seaman, under my care, admitted to the Medico-Chirurgical Hospital of Philadelphia. In this case the dislocation of the kidney was apparently caused while on shipboard by a prolonged and intense straining at stool after taking a large dose of castor oil.

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[1] *Edinburgh Med. Journ.*, Sept., 1898.

Symptoms.—Movable kidney may exist without any symptoms whatever. It may be discovered accidentally by physical examination, and not infrequently it is found *postmortem* in a similar manner, no history of trouble having been elicited during life.

The symptoms of movable kidney are local, reflex, and general, the *local* and *reflex symptoms* being the most prominent in the average case. The reflex symptoms, though usually abdominal, may become general. The local symptoms are most marked in extreme mobility of the kidney (floating kidney), while in moderate mobility the reflex symptoms usually predominate over the local.

Most frequently there is a troublesome dragging pain, or a sense of weight or pressure in the loins or abdomen, especially after long walking or standing or hard labor; this may, at times, be referred to the sacral region. Sometimes the pain may be quite sharp and colicky in nature. Pain in the kidney itself is seldom complained of, and then only in those comparatively rare conditions in which congestion is produced by pressure or traction upon the renal veins, obstruction of the ureter, or the like. The patient himself sometimes recognizes the kidney as a tumor, tender and distressing, as in a case under my own care.

Reflex gastro-intestinal disturbances are common. *Indigestion* is usually complained of, and occasionally nausea and vomiting are noted. Dilatation of the stomach may possibly be caused by a dislocated kidney pressing upon the duodenum, but the association of the two conditions is probably coincident rather than causative. Pressure-jaundice is also an unusual concomitant of the floating kidney. *Cardiac palpitation*, constipation, flatulence, and edema of the lower extremities (from pressure on the inferior vena cava) may attend, and disturbances of the pelvic viscera have also been noted occasionally (dysmenorrhea, abortion, and irritable bladder). Improvement is usually considerable in pregnancy. Some cases of displaced kidney are characterized by sudden and severe attacks of nephralgic or gastralgic pains, chills, fever, vertigo, nausea and vomiting, and general collapse. These attacks are often periodic, occurring sometimes at the menstrual period, and are known as "*Dietl's crises*" or "*incarceration symptoms.*" They may be excited, also, by a too free indulgence in eating and drinking, as in a case reported by Osler. It is most probable, as Dietl himself suggested, that these cases are due to a twisting or bending of the renal vessels or of the ureter, or, perhaps, to circumscribed inflammation of the mobile kidney. An acute hydronephrosis may thus develop, with diminished diuresis. The urine is concentrated, and may contain uric acid or oxalates in excess. After three or four days, as the attack subsides, micturition becomes free, the swollen and sensitive kidney becoming movable once more. When produced by movable kidney these attacks of *transitional hydronephrosis* may occur intermittently (*vide* Hydronephrosis).

Floating kidney associated with Glénard's disease (enteroptosis, splanchnoptosis), in which the transverse colon, pancreas, stomach, intestines, and other viscera are prolapsed, owing to looseness and weakness of the visceral attachments, gives rise to symptoms similar to hose stated above, only with the addition of greater discomfort and utritive and nervous disturbances. Sometimes there is albuminuria.

The *general symptoms* of movable kidney are those of "nervousness" eurasthenia, or hysteria. Mental anxiety, leading to melancholia, someimes follows the discovery by the patient of a movable abdominal tumor, which is persistently believed to be a "cancer." Cephalalgia, backache, mental irritability, paresthesias, neuralgias, nervous dyspepsia, and various hysteric manifestations may arise and prove a perpetual annoyance. In those less frequent instances in which men are affected with mobile kidney hypochondriasis may develop.

The **physical signs** of movable or floating kidney are highly important and diagnostic. *Palpation*, especially bimanual, as by Israel's method. The patient lying in a semi-recumbent position, counter-pressure (the left hand being placed over the lumbar region, the right next the skin in front, manipulating the abdomen from above downward) may detect a firm, movable tumor of renal size and shape in either flank (usually the right) just below the ribs (movable kidney), or in the inguinal or umbilical regions (floating kidney). Or, the patient may stand and, grasping the back of a chair, may lean slightly forward, while the examiner, at the patient's side, presses with one hand over the loin, and with the other feels over the abdomen below the ribs. Though comparatively easy to outline, the tumor is nevertheless hard to grasp; it is often, however, readily pushed into place. Deep breathing may affect a paipable or movable kidney, but has no effect upon one that freely wanders about the abdomen (floating kidney.) Pulsation of the renal artery may be felt in the last-named cases.

Inspection and *percussion* of the lumbar region in movable kidney are uncertain, and therefore unreliable. Visible depression here is rarer than a visible tumor anteriorly; the latter, however, is not common, although it is occasionally noted in cases of marked wandering of the kidney, as to the inguinal region. I have noted increased tympany over the affected side in several cases as compared with the same area on the opposite side.

A **diagnosis** is possible only after a careful and thorough physical examination. When this is made, an abnormally mobile kidney is usually discovered without difficulty. The size and shape of the organ, its right-sided position, and its mobility, associated with a train of local, reflex, or general nervous disturbances, especially in a thin, emaciated woman, are quite distinctive. A knee-elbow posture is sometimes more favorable than the recumbent position for determining a movable kidney.

Floating kidney is of course more easily diagnosticated than the movable type, and partly because of the fact that in instances of the latter, tumors of the gall-bladder especially, and wandering spleen must first be excluded. The absence of a well-defined splenic notch, the presence of pulsation of the renal artery, a tympanitic note over the usually intervening colon, and an unchanged area of splenic dulness will assist in the diagnosis; in addition there is the fact that wandering spleen is a comparatively rare affection.

Differential Diagnosis.—Tumors of the gall-bladder, as Henry Morris has shown, are frequently mistaken for movable kidney; occasionally the opposite error is made; sometimes both conditions may exist. They are both common to women; the right kidney is more often movable than the left; they both may present as tumors in the right hypochondriac and umbilical regions; they are more or less movable, firm, smooth, slightly tender, round or oval in shape, with variable percussion-signs, and dyspeptic symptoms; and either may give rise to paroxysms of severe colic, or to jaundice. Jaundice, however, is probably rare in movable kidney, while emaciation and general nervous disorders are more common; the floating tumor is also less easily palpated than the cholecystic, and may vary in size (hydronephrosis), the diminution being accompanied by a marked increase in the flow of urine. If the gall-bladder be filled with calculi, the consistence is firmer than that of the kidney, and fremitus may be felt. Moreover, the movements of the gall-bladder are usually lateral within a short arc of a circle, the center of which is a point beneath the edge of the right lobe of the liver; while those of floating or movable kidney may be either vertical, oblique, or lateral in arcs of a much larger radius. Again, tumors of the gall-bladder descend with inspiration, as is not the case with wandering kidney.

In some cases it may be necessary to distinguish between the attacks of pain known as "Dietl's crises" and renal, hepatic, or intestinal colic, acute intestinal obstruction, affections of the genital organs, and appendicitis; the symptoms peculiar to these conditions must be considered in forming a diagnosis.

Tumors of the ovaries and bowel are rarely confounded with wandering kidney.

Prognosis.—In uncomplicated cases life is never endangered, and a cure may be effected in a large majority of cases in which suitable combined medical and surgical treatment is pursued. The general nervous symptoms are usually very obstinate, but after relief is afforded from the local and reflex symptoms, whether by tentative or operative means, they subside or cease altogether.

Treatment.—Since emaciation and loss of perirenal fat is a frequent cause of wandering kidney, it is often advisable to resort to measures that will tend to increase the weight and fat of the body. The "rest-cure," with its forced feeding, may be all that is necessary in highly nervous subjects having but a slightly movable kidney. In all cases more or less prolonged intervals of rest (lying down) throughout the day aid markedly in ameliorating the symptoms. Other hygienic measures, as the avoidance of over-exertion, extreme bodily movements, straining—as at stool—and so forth, should also be enjoined.

For several years, and until recently, the operation for anchoring the mobile kidney has been advised as appropriate in nearly all cases. This is now perhaps wisely deprecated; and a reversion to the careful, patient, and constant use of suitable abdominal pads and binders in certain cases is meeting with much success in affording comfort and support, besides a marked reduction in the reflex, abdominal, and general nervous symptoms. In severe cases of renal displacements, in which recurring attacks of hydronephrosis, strangulation-crises, profound nervous and mental disturbances, or other grave renal complications occur, some such surgical procedure as nephrorrhaphy may be necessary. This often proves an effectual cure, although occasionally the anchorage may be torn loose by a sudden or severe physical effort. Total extirpation of the kidney (nephrectomy) is justifiable only in the gravest cases and after other means have failed. The hypodermic injection of morphin and atropin and the external application of heat are indicated in the crises of Dietl.

CIRCULATORY DISORDERS OF THE KIDNEYS.

ACTIVE HYPEREMIA.

(*Acute or Active Congestion.*)

Definition.—An acute, temporary engorgement of the vessels of the kidneys, with little or no exudation.

Pathology.—The kidney is swollen, deep-red in color, and engorged with blood, which flows freely on section. Microscopically, in severe congestion there may be seen cloudy swelling of the cortical epithelium.

Etiology.—Acute renal congestion is due mainly to the action of irritants present in the circulation, as in the acute infectious (especially the eruptive) fevers. The stimulating diuretics and certain poisonous drugs, as copaiba, squills, cantharides, potassium chlorate, and carbolic acid, also sudden contraction of the peripheral blood-vessels by exposure to cold while the body is overheated, act as causes. Post-operative acute hyperemia (ether ?) is frequently met with, particularly after abdominal sections. When prolonged the congestion passes into an acute nephritis. It may be caused in one kidney as a result of the nephrectomy of its fellow. Certain ill-defined centric and peripheral nervous influences and neuroses are held by some to cause an active hyperemia of the kidneys through a vasomotor paralysis of the renal arteries.

Symptoms.—There may be a dull pain in the lumbar region, with a slight elevation of the temperature and pulse-rate. The *urine* either is scanty, or, as in cantharides-poisoning, it may be altogether suppressed. It is dark, the specific gravity is increased, and it contains some free blood, a trace of albumin, and a few hyaline tube-casts.

Diagnosis.—The absence of a marked quantity of albumin, of the numerous and various casts, of dropsy, and of uremic symptoms distinguishes active hyperemia from *acute nephritis.*

The **prognosis** is quite favorable upon the removal of the cause. It must be borne in mind that a frequent repetition of the attacks may lead to a nephritis.

Treatment.—Absolute rest and a liquid diet should be ordered. Cupping over the loins or the use of hot fomentations should be practised. The free use of water and other diluents or mucilaginous drinks should be encouraged. Saline laxatives to freely open the bowels, and the use of hot air or a hot pack to promote sweating, are important aids in relieving the congested kidneys.

PASSIVE HYPEREMIA.

(*Chronic or Passive Congestion.*)

Definition.—A chronic venous engorgement of the renal vessels, generally secondary to diseases of certain other viscera.

Pathology.—There is in the later stages a characteristic condition of the kidneys called "cyanotic induration." Earlier in the case the organs are enlarged, firm, and of a dark, bluish-red color. The capsule is usually non-adherent. On section the medullary substance is seen to be darker red than the cortex and coarsely fibrous in appearance. Microscopic examination shows the capillaries (both glomerular and medullary) somewhat dilated and the walls thickened. The epithelium may either be unchanged or a little cloudy and swollen, or, later, even fatty; the interstitial tissue may be slightly increased, especially beneath the capsule of the kidney.

Etiology.—Most commonly the renal congestion is a part of a general venous engorgement due to chronic cardiac, pulmonary, or hepatic disease. It is found in mitral valvular disease with ruptured compensation of the heart; in pulmonary emphysema, fibroid phthisis, and chronic adhesive pleurisy; and in cirrhosis of the liver. The "*cardiac kidney*" is the commonest variety. Less frequent causes of congested kidneys are tumors, the pregnant uterus, and ascites, all of which bring about the condition through pressure upon the renal veins. Only rarely may passive renal congestion be due to thrombosis of the ascending vena cava or of the renal veins.

Symptoms.—These are accompanied by those due to the primary diseases that are manifested in the general venous congestion, as *edema* of the lower extremities. There may be a sensation of weight in the loins. The *urine* is diminished in quantity, of a higher specific gravity, and darker in color; it contains a little albumin, some blood-corpuscles, and a few hyaline casts and epithelial cells, depending upon the chronicity and intensity of the congestion. Urates may be deposited in the standing urine.

Diagnosis.—From *nephritis* passive renal congestion may be differentiated by the comparative absence of albumin, casts, general dropsy, and uremia, and by the undiminished quantity of urea.

Prognosis.—This depends upon the primary cause. Chronic congestion may pass into chronic nephritis.

Treatment.—Rest and a light and easily assimilable diet, together with cardiac tonics and diuretics, are indicated. The infusion of digitalis serves a good purpose by increasing the quantity of urine and clearing it of albumin. Basham's mixture is a useful adjuvant.

EMBOLIC INFARCTIONS.

Anemic and hemorrhagic infarctions of the kidney are of pathologic rather than of clinical significance. Cicatrices may result from these infarctions, giving rise to the "embolic contracted kidney." Very rarely the *sudden appearance* of a slight amount of *blood* in the *urine*, associated with cardiac disease and possibly with a sudden severe pain over the loin, may point to hemorrhagic infarction.

SPECIAL PATHOLOGIC STATES OF THE URINE.

HEMATURIA.

Definition.—The presence of blood in the urine.

Etiology.—(1) *Local* or *renal causes* of hematuria include congestion (including that due to torsion of the renal vessels in certain cases of floating kidney), acute inflammation of the kidneys, and acute exacerbations of chronic nephritis, embolic hemorrhagic infarction, renal calculi and pyelitis, tuberculosis, malignant renal disease, diffuse myxangiomatous condition of the pelvic submucous tissue (Myles), actinomycosis (O. Israel), hydatids, traumatism, and parasites (the *filaria sanguinis hominis* and *distoma hæmatobium* (Billharz).

(2) *Affections of the Urinary Tract.*—*In the ureter*, calculi or lacerations due to traumatism, as in protracted and complicated abdominal sections; *in the bladder*, calculi, malignant tumors, acute cystitis, ulceration and rupture of varicose veins at the vesical neck; and *in the urethra*, gonorrhea, calculi, parasites, and traumatism,—may all cause hematuria.

(3) *General Diseases.*—Acute specific fevers and certain blood-dyscrasiæ (purpura, gout, scurvy, hemophilia, malaria, and leukemia) may produce hematuria. Malarial hematuria in mild form is not an uncommon feature of paludism in the Middle States of this country, and may occur after the manner of intermittent malarial paroxysms. That due to the renal congestion of chronic heart-, lung-, or liver-disease is not a marked condition, and has not been of frequent occurrence in my experience.

Senator describes an interesting and unusual form of hematuria that is sometimes seen in young persons whose health may be quite fair, the blood often appearing paroxysmally and without apparent cause ("renal hemophilia"), or "renal epistaxis" (Gull). Hematuria may be also a manifestation of vicarious menstruation.

Endemic hematuria, so called, is that variety found in some of the tropical regions where the distoma hæmatobium (a trematode worm) abounds.

Diagnosis.—This has for its object the discovery (1) of blood in the urine, and (2) of the source of the hemorrhage. Bloody urine varies in color according to the quantity of blood present, to its condition (coagulability), disposition, and the length of time present in the urine. A light reddish tinge may indicate a slight quantity of blood. A dark coagulum may be at the bottom as a sediment, with small clots floating above in a deep-red, turbid layer, above which, again, the urine may show but the slightest tint of red. Or the urine may have a smoky-red or chocolate-hued appearance. Microscopically, the blood-corpuscles are readily discovered, establishing the diagnosis from hemoglobinuria, in which condition they are absent. When red corpuscles are associated with tube-casts, renal hemorrhage may be positively diagnosed. In ammoniacal urine or in urine of low specific gravity the corpuscles are very pale and shadowy (dissolved hemoglobin). After remaining in ordinarily acid and diluted urine they lose their disk-like shape and swell into spheres of a smaller diameter. Urine containing blood always shows the presence of albumin. According to Newman,[1] a ratio of albumin to hemoglobin in excess of 1 to 1.6 indicates not only an independent albuminuria, but also a renal affection as the cause of the hematuria.

Chemically, the blood-pigment may be detected by Heller's test, which consists in adding liquor potassæ, boiling the urine, and observing the flakes of precipitating phosphates, which become reddish-yellow or brown from the added hemochromogen. The guaiacum test is also used. The spectroscope is sometimes employed to discover the bands produced by the blood coloring-matter.

The *source of the blood* in hematuria is of great diagnostic and therapeutic importance. In *renal hemorrhage* the blood is thoroughly mixed with the urine, giving a uniformly red or brown color, as in hemorrhagic nephritis. Blood-casts and leukocytes may also be found. The disease causing hematuria may be traced sometimes by a study of the urine; thus, in cases of valvular cardiac disease the sudden appearance of hematuria would indicate *infarction* of the kidney. The discovery of a few red blood-corpuscles in a concentrated urine would point to renal congestion. In profuse renal hemorrhages clots representing moulds of the renal pelves and of the ureters may be discharged. Hemorrhage due to *calculus* is usually small in amount and appears at more or less prolonged intervals. Tubercular hemorrhages may occur very seldom.

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[1] *Lancet*, July 9, 1898.

Blood from the ureters is usually moulded in clots in the shape of curved cylinders, and appears like small dark worms in the urine. Casts from the ureters are often secondary to hemorrhages; in such cases the hematuria may alternate with the passage of clear urine, owing to temporary hemorrhages or to the blocking of the ureter on the diseased side. (See also Fibrinuria.)

Vesical hemorrhages may be quite copious. The blood and urine are not intimately mixed, and large clots settle on standing. The first portions of urine discharged may not be bloody, while the last portion may consist of pure blood.

Finally, *urethral* blood is discharged before the urine, and either comes away freely or may be "milked out" independently of urination.

The endoscope has been used successfully to determine the source of the hemorrhage (which kidney ?). It is especially useful in women.

Prognosis.—This varies with the primary source of the hematuria.

The **treatment** consists primarily in rest in bed. The application of dry cold to the loins is useful, and the hypodermic injection of ergotin is to be recommended for trial. Internally, such hemostatics as the extract of hamamelis virginica, the extract of hydrastis canadensis, gallic acid, lead acetate, calcium chlorid, ergot, and opium may be used. Cantharides tincture in 2- to 5-drop doses has been tried with good results in hematuria due to renal congestion. The good results following the use of a 10 per cent. solution (a pint daily) have attracted some attention recently.

HEMOGLOBINURIA.

Definition.—The presence of blood-pigments, especially methemoglobin, in the urine.

Etiology.—The direct cause of hemoglobinuria is a condition of the blood in which, as a result of the dissolution of the red corpuscles, the hemoglobin is set free and is excreted by the kidneys.

(1) The **causes of the hemolysis** are principally *toxic*, and include the following: (*a*) Poisons (carbolic and pyrogallic acids, potassium chlorate, naphtol, phosphorus, arseniuretted hydrogen, and carbon dioxid). (*b*) The ingestion of poisonous fungi or of tainted edible mushrooms (*Helvella esculenta*). (*c*) The poisons of certain infectious diseases (scarlatina, typhus and typhoid fevers, yellow fever, syphilis, scurvy, purpura). (*d*) Extensive burns, the absorption of hemorrhagic effusions, and the transfusion of animal blood. (*e*) Rarely it may be due to exposure to cold and to violent physical exertion. (*f*) The so-called *epidemic hemoglobinuria* (Winckel's disease) occurring in the newborn.

(2) **Paroxysmal hemoglobinuria,** a rare and interesting variety, may occur without any apparent cause in persons enjoying otherwise good health. It appears thus distinctly as an independent disease. Some of these cases, however, have been attributed to a peculiar susceptibility to cold (generally or locally applied) and to marked exertion; especially is this the case in adult white males. It is held by some to be a manifestation of Raynaud's disease, uricemia, and by others to be the result of syphilis.

(3) It appears as a symptom of *malaria* (which acts like the other infectious diseases) in the southern part of this country, where the pernicious varieties of malarial toxemia are most common. This is termed *malignant malarial hemoglobinuria* or *hemoglobinuric fever*. In Africa it is called *black-water fever*.

Symptoms.—These are generally the symptoms of the condition that accompanies hemoglobinuria. In paroxysmal hemoglobinuria the attacks are usually sudden, brief in duration, and sometimes *intermittent*, especially when of malarial origin. An anemic condition seems to be essential to the production of malarial hemoglobinuria. *Jaundice* may be an associated symptom. The hemoglobinuria seldom lasts for more than two days, though very grave cases take on the aspect of a pernicious malarial attack. There may be lumbar pains, chills and fever, and gastric disturbances. Urticaria and purpura have also been noted, as has anemia in cases in which frequent attacks have taken place.

Diagnosis.—This is made by an examination of the urine. Macroscopically, it is of a red-brown color, slightly turbid, with a reddish-brown or brownish-black sediment. The reaction is usually acid, and the specific gravity slightly lowered. The microscopic features that distinguish hemoglobinuria from hematuria are variable. In the former condition few or no red corpuscles are present, and the few that may be seen are usually colorless ("shadows") or fragmentary. Small flakes or granules of disintegrated hemoglobin are found, and are brownish-black in color. There may be also brown-tinged casts and epithelium. Chemically, the urine is found tc contain albumin, for the discovery of which Heller's and the guaiac tests for blood-pigment may be tried. The former has been described in the preceding discussion of Hematuria. The *guaiac test* consists in overlaying with urine a mixture of the tincture of guaiac and hydrogen peroxid or the oil of turpentine (equal parts). When the blood-coloring matter is present, an indigo-blue ring is formed above a white resinous deposit. When shaken a lighter blue color develops throughout the contents. By means of the spectroscope the three absorption-bands of methemoglobin may be seen (red, green, and yellow). The blood-serum in hemoglobinuria may be somewhat red-tinged on account of the dissolved hemoglobin. The hemoglobinuria is further marked by the aplasticity of the red corpuscles, by their pallor, by poikilocytosis, and by the presence of the irregular flakes of hemoglobin.

The **prognosis** of hemoglobinuria depends upon the cause. It is favorable in the ordinary paroxysmal form. Malignant malarial hemoglobinuria, however, is often fatal.

Treatment.—Hemoglobinuria is rather intractable. During the paroxysms external warmth is needed, along with hot drinks to encourage perspiration. In malarial cases quinin, and in syphilitic the iodids, should be administered; although by some it is believed that quinin may aggravate the syndrome in particular cases.

ALBUMINURIA.

Definition.—The presence of albumin in the urine.

Pathology and Etiology.—The immediate cause is the escape of the normal blood-constituents, serum-albumin and serum-globulin, from the vessels into the renal tubules. This transudation of albumin indicates either a transient and slight or a permanent and grave nutritional disturbance of either the epithelium lining the glomeruli or of that of the contained tufts of capillaries, or, possibly, of the *membrana propria* or the epithelium of the uriniferous tubules. These changes induce and offer an abnormal perviousness to the albumin of the blood.

The principal *causes* of albuminuria are—(1) Those associated with definite lesions of the kidney; nephritis, acute and chronic; renal congestions, active and passive (the latter being secondary to chronic liver-, heart-, and lung-disease, pregnancy, or tumors); and certain *toxemias* Among the last-named are included scarlet fever (scarlatinal nephritis) and gout. Other causes are—amyloid and fatty degeneration of the kidney, suppurative nephritis, and renal tumors (cystic kidney).

Albuminuria occurs also in conditions in which (2) the renal lesions are either slight or undemonstrable: (*a*) Thus, it is present in blood-changes, as in chronic lead-, mercury-, and arsenic-poisoning, scurvy, purpura, syphilis, leukemia, or extreme anemia, and in cases in which urobilin or bile-pigment and sugar (glucose) circulate in the blood. Again, slight albuminuria may be present in pregnancy (*kidney of pregnancy*), in saccharin diabetes, and after etherization. In certain affections of the *nervous system* albumin is found in small quantity, as after an epileptic paroxysm, in tetanus, injuries to the head, apoplexy, and exophthalmic goiter.

(*b*) The so-called *accidental* or *spurious* albuminuria is due to the presence of pus or blood; in such cases the condition is not a true renal albuminuria, since it is commonly associated with cystitis, pyelitis, urethritis, or is the result of hemorrhage from the pelvis of the kidney, from the ureters, bladder, or urethra.

(*c*) *Febrile* albuminuria is of rather frequent occurrence in diseases accompanied by pyrexia, especially when long continued. Among these are typhoid fever, small-pox, yellow fever, diphtheria, and even influenza, follicular tonsillitis, and pneumonitis. The renal changes in these cases are, I believe, merely a transitory cloudy swelling in the glomeruli, which, together with the albuminuria, rarely lasts longer than the fever.

(*d*) Other forms of albuminuria have been styled *physiologic* or *functional*, *transient*, *dietetic*, *neurotic*, *intermittent*, and *cyclic*: in these no definite lesions of the kidney are found, and are denied by some to exist. Recent observers are inclined to believe that trivial, non-progressive renal changes occur in these cases. Slight albuminuria certainly does occur in some cases after a heavy meal rich in albumin, after marked and prolonged muscular exertion, intense emotion, and cold bathing.

(*e*) *Cyclic albuminuria* has come to be of greater interest and importance in later years, particularly as it bears upon the prognosis and upon life-insurance risks. In this variety there are a periodic appearance and absence of albumin in the urine. The albuminuric paroxysms are very variable, recurring usually after meals or on exertion, according to some, largely the result of the assumption of the upright posture upon rising from bed, but generally being absent during rest at night. The albumin is present in but small quantity, and only rarely are casts (hyaline) found. The urinary features are otherwise normal, and the accompanying signs and symptoms common to nephritis are absent. Cyclic albuminuria is most common in adolescent anemic males, of poor nutrition (gastro-intestinal auto-intoxication?), dyspeptic, neuralgic, often neurotic, and even hysteric. Under careful management these cases ordinarily recover. There is, however, a class of cases in which the albuminuria is persistent, though but a mere trace of albumin may be detected, and neither can tube-casts be found nor are symptoms complained of. However, an insidious degeneration of kidney-structure may manifest itself many years later. According to Ranault, albuminuria may rarely be even *hereditary*. Slight *senile albuminuria*, without evidence of renal disease, is not uncommon.

Diagnosis.—This rests upon the discovery of albumin by means of any one or more of the reliable tests shortly to be described. Since albuminuria is no more synonymous with nephritis than is glycosuria or polyuria with diabetes mellitus, it becomes important and necessary, both for prognostic and therapeutic reasons, to differentiate between the so-called functional albuminuria, the cyclic variety, and those associated with coarse and definite anatomic lesions of the kidney.

Differential Diagnosis.—Inquiry and careful inference concerning the etiology of a given case must be made. *Renal albuminuria* is persistent and of considerable quantity, except in chronic interstitial nephritis. Tube-casts are usually present. *Functional albuminuria* is slight and inconstant. Tube-casts are either absent or exceedingly few in number in the latter. Again, in the former variety general symptoms, as dropsy, cardiac hypertrophy, anemia, and uremic prodromes, are present. It is true that slight edema is sometimes found in cyclic albuminuria, but this is probably due to the marked anemia that is so often seen. When the quantity of albumin is disproportionately large in *spurious albuminuria*, a suspicion of coexisting renal albuminuria should be aroused (Strümpell).

Tests for Albumin.—Two samples of urine, one of the morning before any food is taken, and one of the evening before the patient retires, should be examined. Care should be taken that there be no contamination of the urine with the menstrual, leukorrheal, or urethral discharges. The smallest quantity can be detected only by its coagulum rendering the urine turbid; hence any turbidity present before the given test is made should be removed by filtration, unless this turbidity be due to urates, when a little warming of the tube will render the urine clear.

(1) *Boiling Test.*—This is the commonest and I think the most reliable practical test for albumin. The tube is filled about two-thirds full of urine. If alkaline or neutral in reaction, a drop of acetic or nitric acid is added; an excess of acid must be carefully avoided, lest the albumin (if present) be converted into a non-coagulable form. The tube, held aslant, is then applied to the flame, and slowly revolved with the fingers, so that the upper portion of the column of urine is brought to the boiling-point. A comparison of this with the lower portion of the

urine is made. Any turbidity is due to albumin or phosphates. If albumin, adding a few drops of nitric acid will increase and thicken the coagulum; if phosphate, the opaqueness will be cleared at once.

(2) *Heller's Nitric-acid Test.*—This is easily performed, and is both delicate and satisfactory. About 1 c.cm. of nitric acid is poured into a tube, and some urine is allowed to flow slowly from a pipet and settle upon the acid. The presence of albumin is indicated by a white ring at the point of contact of the two liquids. Uric acid, urates, and certain urinary coloring-matters form a pink or deep-red ring or zone; this forms, as a rule, above the juncture of the acid and urine. Hemialbumose also gives a white zone, but does not respond to the boiling test as does serum-albumin.

(3) *Johnson's Picric-acid Test.*—To filtered urine in a test-tube are slowly added a few drops of a saturated watery solution of picric acid. Immediate turbidity indicates albumin. Some authorities prefer that a dram or two (4.0–8.0) of the yellow fluid be placed gently on the surface of the urine, when, if albumin is present, a white zone at once is apparent, together with a haziness that spreads downward with the diffusion of the liquids. Heating emphasizes the evidence of the test, which is extremely sensitive.

(4) *Roberts' nitric-magnesium test* is also very delicate. It consists in using the following mixture, just as in Heller's test: one volume of concentrated nitric acid, added to five volumes of a saturated solution of magnesium sulphate.

(5) *Trichloracetic-acid Test.*—This will discover minute traces of albumin, but has the disadvantage that it responds to nucleo-albumin as well as to serum-albumin. A few crystals may be dropped into the urine, or a saturated solution may be used after the "contact method," when, if albumin be present, a white coagulum forms. This and the Geisler test-papers (Vierordt) constitute portable and handy tests.

(6) The *acetic-acid and potassium-ferrocyanid test* is also valuable and minutely sensitive. The urine is first made decidedly acid with acetic acid. A few drops of a freshly prepared solution of potassium ferrocyanid are then added, and if either albumin or hemialbumose be present, it will be precipitated.

(7) *Quantitative Test—Esbach's Albuminometer.*—This consists in using a graduated test-tube, into which definite amounts of urine and a reagent composed of 10 parts of picric acid, 20 of citric acid, and enough water to make 1000 parts are carefully mixed by reversing several times the stoppered tube. After allowing this to stand about twenty-four hours, the height of the precipitated albumin is read off on an etched scale, which will indicate approximately the parts per thousand. Not less than 0.5 parts per thousand can be estimated correctly, however. Should there be a hematuria, if the percentage of albumin by Esbach's method, divided into the number of red cells per cubic centimeter of urine, is less than 30,000, it suggests a purely hematuric albuminuria; if greater, it suggests an independent albuminuria (Goldberg).

Prognosis.—Etiologic considerations bear heavily in this matter. Functional abuminuria is of favorable import, as a rule. The febrile, hemic, cyclic, and paroxysmal varieties usually clear up with convalescence and with advancing years (in the latter case). The persistence of albumin in these cases, however, even in slight amounts or at variable periods, should cause suspicion, since there must be some glomerular renal change to account for the disorder, the tendency of which is to progress steadily and insidiously. Especially is this true when there is associated a gradually increasing arterial tension. The presence of tube-casts is conclusive of structural change in the kidneys, marked by degenerations and by exudative and productive inflammation, either acute or chronic.

ALBUMOSURIA.

True peptone (Kühne) has never been demonstrated in the urine. The so-called peptones discovered by Devoto's and other methods are really *albumoses*. These albumoses (proto-, deutero, hemi-) are found in acute fevers, and in acute suppurations or when resolution of inflammatory effusions is going on, as in lobar pneumonia. They may be found also in acute rheumatism, scorbutus, myxedema (Fitz), primary multiple sarcoma of bone,[1] and in certain forms of metallic and ptomaïn or bacterial poisoning. Albumosuria may be suspected when, after negative results with the boiling and nitric-acid tests, cold acetic acid produces a cloudiness. This suspicion may be confirmed by the *biuret* test, as follows: Any albumin that is present must first be coagulated and removed. Then, after placing some Fehling's solution in a test-tube, an equal quantity of urine is allowed to come in contact with it, when, if albumoses be in the latter, a rose-pink zone or halo appears at or near the point of contact.

Hemialbumose is formed in the urine in osteomalacia, in chronic suppurations, and in sarcomatous disease of the spinal cord, though the clinical significance of the substance has not been fully determined.

INDICANURIA.

Definition.—The presence of a pathologic quantity of indican in the urine.

Indican occurs in the urine in health in very small quantities as a colorless compound, and is, chemically speaking, indoxyl-potassium sulphate.

Pathology and Etiology.—Indican is increased abnormally in the urine by any disorder whereby large quantities of albuminous matters are decomposed. Thus, it occurs in intestinal obstruction, especially when the caliber of the small bowel is diminished from any cause so as to produce a stagnation of the contents and a consequent decomposition from bacterial action, especially the action of large numbers of the common colon-bacillus. Under such circumstances indol and phenol are formed. The former, being absorbed and oxidized into indoxyl, finally appears in the urine in combination with potassium sulphate. Acute peritonitis, obstinate and chronic constipation, wasting diseases, and cachectic conditions in which there is a considerable destruction of albuminoids (as in Addison's disease, neoplasmata, cholera Asiatica, and empyema) usually have an associated indicanuria. An increase of the aromatic sulphates in general, or an increase in proportion to the fixed sulphates, is especially significant of intestinal putrefactive processes. Since the pancreatic secretion peptonizes the proteids from which arise leucin and tyrosin, and these in turn are decomposed into skatol, indol, and phenol, it is stated (Piseuti) that any obstruction preventing the flow of the pancreatic juice into the bowel would be reflected in a diminished quantity of indican in the urine. On the other hand, any epigastric tumor suspected of pressing upon the small intestines would be accompanied with indicanuria.

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[1] De Holstein: *Sem. Méd.*, March 5, 1899.

Diagnosis.—This depends upon the demonstration of indican by adding strong oxidizing agents, which decompose this product and set the indigo or pigment free. At times sufficient oxidation of the indican has taken place in the urine before any chemical test is applied, so that a bluish tinge is given thereto. This may be seen in urine that has been standing for some time, the sediment giving a bluish reflection, or there may be a blue-turbid film on the surface. In the urine of indicanuria, moreover, where putrefaction is marked, a pronounced blue-black color may be present.

Tests.—*Jaffe's* well-known test consists in mixing equal volumes of urine and hydrochloric acid, and then adding, drop by drop, a concentrated solution of chlorinated lime, shaking the tube after each addition. A strong indigo-blue color appears if there is much indican.

A good modified test is the use of fuming nitro-hydrochloric acid and urine (equal parts) and a saturated solution of chlorinated potash, used as in the above method. A blue-black cloud or ring appears below the surface. If a few drops of chloroform are then added and the mixture is agitated slightly, a blue color settles at the bottom, owing to the chloroform carrying with it the oxidized indican.

PYURIA.

Definition.—The presence of pus in the urine.

Etiology.—Pyuria is due to (1) suppurative inflammation along some portion of the genito-urinary tract, or (2) to the rupture of adjacent abscesses into the tract.

Pyelitis and Pyelo-nephritis.—Pus from the pelvis of the kidney may be due to calculous, tuberculous, or other irritation. It is associated at times with the "railed" or transitional epithelium usually seen early in the case. In pyelo-nephritis casts may indicate renal involvement, although it should be borne in mind that in abscess of the kidney pus may be discharged continuously without the appearance of any casts in the urine whatsoever. One such case came to necropsy under the observation of H. S. Anders, in which small uratic calculi were discharged now and then for several years. Later, several larger stones were removed from the bladder by Willard by suprapubic cystotomy, in the hope that by drainage and irrigation of the bladder the marked pyuria might subside or cease. The abdominal opening healed in a few months, and, whilst bladder-symptoms were absent after removal of the calculi, pyuria persisted. Death having occurred suddenly from coronary artery disease and interstitial myocarditis, it was found *postmortem* that a large abscess occupied the lower third of the left kidney, which was filled with small, dark, and irregularly-shaped calculi. A thick pyogenic membrane surrounded the purulent and calculous contents. No casts were found at any time during life, though repeated examinations were made, and, remarkable as it seems, renal symptoms were altogether absent.

The pyuria is sometimes *intermittent*, one ureter becoming temporarily occluded (on the side of the disease), the clear, normal urine from the healthy kidney passing until the ureteral obstruction is relieved, when pus again appears. Purulent urine from the kidney is usually acid in reaction, except when the pyelo-nephritis is secondary to cystitis, when it is more apt to be alkaline and to contain a decided quantity of mucus. *Cystitis.*—Pyuria in this affection is fetid in most cases. Bladder-symptoms are marked. The urine is alkaline, and a stringy, tenacious muco-pus comes with the last portions. Triple phosphates are often found. The pus and urine are not so intimately mixed as in pyelonephritis.

Urethritis.—The pus is in small quantities, is passed in advance of the urine, and can be "milked out" from the male urethra. There is usually a history of gonorrheal infection, and the gonococcus may be demonstrated in most cases.

Rupture of contiguous abscesses into the urinary tract is accompanied usually with a sudden discharge of a large quantity of pus in the urine, preceded by symptoms of abscess elsewhere, as in the pelvis or right iliac fossa (suppurative appendicitis) or perinephric abscess. The pyuria disappears as abruptly as it came on, or lasts but a few days, lessening gradually until there is a complete cessation. The *strongylus gigas* in the pelvis of the kidney causes pyuria as well as hematuria.

Diagnosis.—Pus gives a greenish-yellow or yellowish-white tinge to the urine and sediment, the latter very often becoming very-tenacious or jelly-like from the presence of mucus. It may resemble a phosphatic precipitate, as in cystitis; the latter, however, is white, lighter, more granular, and not so thick or tenacious. Microscopically, a positive diagnosis is made by the discovery of pus-corpuscles (or leukocytes) with their granular protoplasm, which has the faculty of clearing up and showing one or more nuclei upon the addition of acetic acid. The corpuscles are either more or less swollen and clear, or opaque, granular, or even nucleated, according to their number, the length of time in the urine, and the degree of alkalinity or acidity of the latter. The greater the change in the urine, the more marked the change in the corpuscles. A few phosphatic crystals may be seen, and epithelium more or less characteristic of the seat of suppuration is present.

Chemically, there is slight albuminuria, a marked amount of albumin usually indicating renal disease. Nephritis may be diagnosed in connection with pyuria by the discovery of casts. On the addition of liquor potassæ to urine containing pus the latter is converted into a clear gelatinoid substance; mucus, on the other hand, becomes thin and flocculent. Mucus may also be distinguished from pus by its failure to react to cold nitric acid, whilst the albumin of purulent fluid coagulates.

CHYLURIA.

Definition.—The presence of chyle in the urine.

Etiology.—This interesting condition may be either *parasitic* or *non-parasitic* in origin. The former type is more common in the tropics, and is caused by an engorgement and rupture of the bladder or renal lymph-vessels, due to obstruction of the larger branches of the thoracic duct or in the duct itself, by the *filaria sanguinis hominis* (*vide* Filariasis). The latter form, the pathology of which is not definitely known, is occasionally found in temperate regions. It is held to follow injuries to the lymphatic ducts, and may be associated with pregnancy.

Diagnosis.—The urine is increased in quantity, and has a milky turbidity (*galacturia*) due to the emulsified fat. After standing for a time a light coagulum settles to the bottom and a creamy pellicle of fat rises to the surface. The sediment contains also the fibrin of the chyle. Sometimes as much as 2 or 3 per cent. of fat is present (*lipuria*); this may be tested by agitating a portion of the urine with ether, whereupon the turbidity disappears. Owing to the serum-albumin in the chyle, the various tests for that substance would show traces of its presence in chyluria. Hematuria may be associated with chyluria, especially in parasitic cases, in which case the blood comes from ruptured veins and tinges the urine accordingly. Microscopically, chyle-containing urine resembles milk in its millions of fine granules and fat-droplets.

Prognosis.—Chyluria is intermittent in its appearance, corresponding to the times of rupture of the vesical lymphatics, and may last for years. The prognosis of non-parasitic chyluria is good as to life, but unfavorable as to cure.

CHOLURIA.

Definition.—The presence of bile-pigment in the urine.

Etiology.—Choluria may be caused by any disease, local or general, in which *jaundice* is a symptom.

Diagnosis.—Bile-stained urine has a color varying from a greenish-yellow to a brownish-green or brown-black, resembling porter. When shaken its foam assumes a characteristic yellow or greenish-yellow color. White filter-paper dipped in the urine is stained yellow.

Tests.—The *chloroform* test consists in adding this substance to the urine and allowing it to settle to the bottom of the tube. If bile or pigment be present, the gravitated chloroform will be colored yellow.

Gmelin's test is most commonly employed, though it is not the most delicate. A few drops of urine and nitric acid are allowed to run together on a white porcelain plate; if bile-pigment (bilirubin) be contained in the urine, a play of colors ensues, the green predominating, followed by the blue, violet, and red, each shade representing a new form of pigment. The first color noticed (green) corresponds to the biliverdin or normal bile-pigment of herbaceous animals. This oxidation of bilirubin into biliverdin is better accomplished by nitric acid containing a little nitrous acid. Hence, the test may be improved by adding enough fuming nitric to ordinary nitric acid to form a yellow trace of the nitrous acid. This may be placed in a test-tube or wine-glass, and some of the urine added gently from a pipet. Bile-pigment will be indicated by successive rings of green, blue, violet, and red from above downward; this occurs, however, only when the bile-pigment is present in considerable quantities.

Rosenbach's test is a modification of Gmelin's, and is more distinct. The urine is first filtered, and a drop or two of the nitric-nitrous acid is then poured upon the filter-paper, when the characteristic colored rings will appear if bile be present. According to Penzoldt, the Gmelin-Rosenbach test is made more distinct by acidulating the filtrate with acetic acid and pouring a thin layer into a white shallow dish. The acetic acid assumes a greenish-yellow, and later a green, or even a blue-green, shade if bile be in the urine. This reaction is quickened or intensified by the application of heat to the liquids.

In the *Marechal-Rosin test* a mixture of one part of the tincture of iodin and ten parts of alcohol is spread in a deep layer over the suspected urine in a test-tube or glass. A grass-green ring forms at the point of contact in choluria.

Bile-acids.—These are principally the glycocholic and taurocholic acids. Traces are found in normal urine, and their clinical significance or diagnostic importance, as far as is known, is practically *nil.*

When testing for bile-acids the Stranburger modification of Pettenkofer's method may be used, as follows: "After isolation cane-sugar is added to the extract, which is then filtered. A drop or two of strong sulphuric acid is spread on the dried filter; a violet or purple color appears" (Musser).

Other constituents of the urine in choluria of long standing are slight quantities of albumin and icteric or yellow bile-stained hyaline or finely-granular casts.

A point in **differential diagnosis** should be noted in connection with the fact that *certain drugs*, as rhubarb and santonin, when given internally, may produce a discoloration of the urine similar to that caused by the presence of bile. On agitation, however, there will be no yellow foam and no reaction to the tests for bile, while the addition of liquor potassæ causes a red color.

UROBILINURIA.

Definition.—The presence of pathologic quantities of urobilin in the urine. Urobilin is the principal coloring-matter of the urine, and hence is present in normal urine in small quantity. It is derived from hematoidin or bilirubin as a product of the reduction of these substances in the tissues and blood-vessels.

When present in large quantities urobilin gives to the urine a red-brown color. This is seen in fevers, varying in depth of shade according to the degree of pyrexia; also in diseases of the liver, after hemorrhagic effusions (due to resorption), in the hemorrhagic diathesis, in purpura, and in progressive pernicious anemia.

When deposited in the tissues it gives rise to a form of jaundice—in which there is a brownish discoloration of the skin—called *urobilin-icterus.*

Diagnosis.—The presence of urobilin is best detected by a spectroscopic examination. A marked absorption-band between Frauenhofer's lines (f and b), fading off from the green into the blue, is characteristic. Chemically, the addition of a few drops of a watery solution of zinc chlorid to the urine will cause the peculiar red-green fluorescence of urobilin to appear.

GLYCOSURIA.

Definition.—The presence of sugar (glucose) in the urine. Normally, a trace of sugar is present in the blood (glykemia), but it may be doubted whether any is excreted in the urine in health, except after the ingestion of an excess of food rich in saccharine or starchy substances. Uric acid may give the same reactions as glucose in the urine.

Etiology.—The causes of glycosuria may be enumerated as follows: (1) Diabetes mellitus—the most common. (2) Certain diseases, like gout (*intermittent glycosuria*), cholera, typhoid, typhus, and scarlet fevers, whooping-cough, diphtheria, malaria (*paroxysmal glycosuria*), tetanus, phthisis, hepatic cirrhosis, and organic nervous diseases, especially those affecting the medulla and involving the floor of the fourth ventricle. Glycosuria may also result from psychic causes, as excessive mental exertion, extreme emotional activity (grief, worry, and shock), from injuries and after operations[1] as cerebral concussion and hemorrhage, and fracture of the skull, from apoplexy, cerebro-spinal meningitis, and after epileptic paroxysms. (3) Pregnancy. (4) Certain toxic agents cause a transient glycosuria, among these being carbon monoxid, morphin, atropin,[2] hydrocyanic acid, amyl nitrite, curare, chloral, alcohol, mercury, arsenic, turpentine, copaiba (Bettman), phloridzin, and various coal-tar derivatives, as salicylic acid and salol. This source of glycosuria has been experimentally demonstrated in dogs by Paul Gibier, of the New York Pasteur Institute. (5) Obesity and thyroidismus may cause a temporary glycosuria (lipogenic). (6) Pancreatic disease (chronic interstitial pancreatitis and, less commonly, pancreatic calculi, carcinoma, and cysts). (7) Glycosuria may occur in exopthhalmic goiter, and, according to Lyman, may be present for a short time in (8) diabetes insipidus. (9) Heredity probably plays a part in predisposing to glycosuria in certain cases, particularly in the permanent affection. (10) Dietetic or alimentary glycosuria may at times be noted, as in beer-drinkers.

Diagnosis.—The daily quantity of the urine of typical glycosuria—*i. e.*, when masking saccharine diabetes—is greatly increased (60 fluidounces—2 liters—and over *per diem*); it is of high specific gravity (1025 and over), of a clear, pale-yellow color, a "ripe-fruit" odor, a sweetish taste, and an acid reaction that is intensified on standing, owing to the fermentation of the sugar. Albuminuria may be associated with glycosuria, and the albumin should be removed before testing for sugar. Again, since urine of high color, heavy density, and marked acidity often contains uric acid, and since this substance responds to the sugar tests, care must be exercised lest a false conclusion be drawn.

Tests.—The most important of these depend mainly upon the peculiar property of glucose in reducing the blue oxid of copper to the orange or red suboxid. It must be remembered that other metallic substances are similarly decomposed.

(1) *Fehling's Test.*—Two solutions are used, equal parts being mixed to form the Fehling's solution, as follows:

Solution I. contains 34.64 gm. of cupric sulphate, dissolved in enough water to make 500 c.cm. Solution II.: 173 gm. of Rochelle salt are dissolved in 480 c.cm. of sodium hydroxid (sp. gr. 1.14); this is then diluted with water up to 500 c.c.

Application: Dilute 1 c.c. of Fehling's solution (about 10 drops of each of the above solutions) with about 1 dram (4 c.c.) of water in a test-tube, and heat to the boiling-point. If the clear blue color remains, the solution is ready for use; should it change color, however, the solution is unfit for use and should be discarded. The suspected urine is added, drop by drop, heating occasionally, when, if glucose be present, the blue color will be discharged by a yellow turbidity, which increases until finally a deep-yellow or orange red precipitate falls. Bluish-white flakes and a greenish discoloration of the mixture simply indicate cupric hydroxid, and not glucose. This test serves for the detection of .001 per cent. of glucose (Wormley).

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[1] T. R. Brown, *Johns Hopkins Hosp. Bull.*, May, 1900.

[2] F. Raphael, *Deutsche med. Woch.*, July 13, 1899.

(2) *Trommer's Test.*—To about 5 c.c. of urine in the tube add one-third or one-half its volume of potassium or sodium hydroxid, and then, drop by drop, add a 10 per cent. solution of cupric sulphate. If a bluish-white precipitate falls, either filter or agitate the liquid until it assumes a slight and uniform turbidity; then heat, and, if sugar be present, a yellow or red deposit of cuprous oxid falls: .01 per cent. of glucose may be detected in this way.

Besides uric acid, there are certain other substances which when present in urine make the copper tests fallacious by reducing the cupric to cuprous oxid. Among these are mucin, lactose, pyrocatechin, hydrochinon, bile-pigments, glycosuric acid, the products of elimination after the ingestion of chloral (urochloric acid), and benzoic and salicylic acids.

(3) *Böttger's Bismuth Test.*—This may be performed as a counter to the copper tests. Albumin, however, interferes with the test on account of the contained sulphur, which forms a black bismuth sulphid: hence, if present, it must first be removed. This may be done by acidulating the urine with acetic or nitric acid, boiling, and then filtering. Böttger's test is then made by adding to the non-albuminous urine or to the filtrate from one-half to an equal quantity of liquor potassæ and a few grains of bismuth subnitrate. Boil for several minutes, and if glucose be present black metallic bismuth will be precipitated.

(4) *Nylander's reagent* may be employed. This consists of 2 parts of basic bismuth nitrate and 4 parts of sodium tartrate to 100 parts of an 8 per cent. solution of caustic soda. One part of the reagent is boiled with 10 parts of the urine for a few minutes, when a change from the original to a brown or black color will indicate the presence of glucose. This test is quite distinct, but has the fallacy that is common to all the bismuth tests, of forming a black precipitate with the sulphur compounds.

(5) *Fermentation Test.*—Though not always convenient to apply, this is, nevertheless, a most reliable test. It depends upon the action of yeast in breaking up glucose into alcohol and carbonic-acid gas (carbon dioxid). It is performed easily by adding a small piece of compressed yeast to the urine in a test-tube, inverting the latter in a dish of the same, and standing aside for twelve to twenty-four hours, the temperature being kept at about 80° to 100° F. (26.6°–37.7° C.). The evolution of gas resulting from the fermentation of the sugar takes place, with a consequent reduction of the specific gravity of the urine. The yeast may be tested simultaneously for its purity and strength by placing one portion in a test-tube containing about two-thirds mercury and filling with normal urine, and a similar portion in a second tube with mercury and a thin, watery solution of sugar or glucose; the fermentation test of the suspected urine may be made at the same time, and all three tubes inverted over a dish of mercury. Obviously, the first

tube should not show the presence of carbon dioxid if the yeast was free from sugar; but the second tube should show this gas to be present or the yeast was inert.

Other tests, such as *Moore's liquor-potassæ-and-boiling test*, *Johnson's picric-acid test*, and the *phenyl-hydrazin test*, are more intricate and in no way more reliable.

The *quantitative estimation of sugar* may be made with Fehling's solution in two parts, as recommended above for the qualitative test. This method is based upon the fact that the cupric oxid of 1 c.c. of Fehling's solution will be reduced by not less than 0.005 gm. of glucose. Place 1 c.c. of the solution in a test-tube and dilute with 4 c.c. of water (5 c.c. dil. sol.). Heat to the boiling-point, and add 1 c.c. of urine, and heat the liquid again. If reduction has taken place, 0.005 gm.—0.5 per cent. or more—glucose is present; if no reduction has occurred, less than 0.5 per cent. is present. If 2 c.c. urine are used before the color of the Fehling solution is discharged, there will be 0.25 per cent. glucose. If $\frac{1}{2}$ c.c. is used, 1 per cent. is present. If $\frac{1}{10}$ c.c. urine is all that is required (about 2 drops), then 5.0 per cent. of glucose is present.

Roberts' differential-density method depends upon a loss in the specific gravity of the urine, due to the fermentation of glucose. According to Roberts, each degree in specific gravity lost is equivalent to 1 grain of glucose in 1 imperial fluidounce (437.5 gr.) of urine, or one degree represents 0.23 per cent. glucose. (See works on Urinalysis.)

Circumpolarization.—Finally, sugar may be determined by the saccharimeter or polariscope. Glucose polarizes light to the right. The percentage may be calculated by reading the vernier scale indicating the degree of reflection, and multiplying the number read by the factor of the apparatus used, after making any required corrections.

ACETONURIA, DIACETONURIA, AND OXYBUTYRIA.

Acetonuria, diacetonuria, and oxybutyria are so closely allied with glycosuria, and especially with diabetic coma (acetonemia), that they may be considered together. In the first-named condition the urine contains acetone; in the second, diacetic or aceto-acetic acid; and in the last, oxybutyric acid. Diacetic and oxybutyric acids are products of the decomposition of acetone, and hence the importance previously ascribed to the latter, when detected in the urine of diabetics, has given place to the two former, the oxidation of which yield acetone.

Acetonuria may exist to a minute degree in health, the acetone being a product of the normal metamorphosis of albumin. It may be present also in—(1) diabetes; (2) carcinoma; (3) febrile conditions; (4) inanition; (5) psychoses; and (6) auto-intoxication, especially with fatty acids in the stomach. Urine that contains acetone in pathologic quantities has a fruity (apple-like) odor or one resembling that of chloroform.

Tests.—(1) *Gerhardt's* original test consisted in the addition of a few drops of the tincture of the chlorid of iron, which produced a Burgundy-red color with acetone, or rather with the aceto-acetic acid.

(2) *Nitro-prussid Test.*—To a fluidounce (32.0) of the urine add a

dram or two (4.0–8.0) of a solution of sodium nitro-prussid (gr. v to ℥j —0.324 to 32.0) and a few drops of strong aqua ammoniæ. On standing a rose-violet color appears. According to Legal, proportionately smaller quantities of urine and the reagent may be used, and strong liquor potassæ. A bright-red color develops, and fades rapidly, but upon adding acetic acid this changes to purple or violet-red (Vierordt). This is a better test.

(3) Perhaps the most accurate and, at the same time, satisfactory test for acetone is the following: Distil the urine with a little phosphoric acid, and add to the distillate a few drops of sodium hydroxid and of Lugol's solution. If acetone be present, yellow crystals of iodoform will form, with the characteristic odor.

Diacetonuria and **oxybutyria** never occur normally. They are often associated with acetonuria in diabetes, and sometimes in fever, or occur as an independent disease (V. Jaksch). Moreover, it is believed that diacetic and oxybutyric acids are the causes of diabetic coma, and not acetone, as was held formerly. Stadelmann affirms that of like value with the recognition of oxybutyric acid in diabetes is the determination of a marked and increasing amount of ammonia in the urine (1 gram—gr. xv—and more *per diem*), as indicating the imminence of diabetic coma. Diacetonuria is found to occur in certain acute diseases of children, accompanied with convulsions.

Tests.—The presence of diacetic acid is demonstrated by the chlorid-of-iron reaction, as in the case of acetone, except that the urine is boiled previously. This is done to avoid fallacy, since in unboiled urine acetic, formic, and oxybutyric acids may strike a Burgundy-red also; in urine that has been previously boiled these do not react, while the diacetic acid does, if present. Diacetic acid is usually present simultaneously with acetone. If a portion of the urine is mixed with sulphuric acid and extracted with ether, diacetic acid may be inferred to be present if the extract shows a chlorid-of-iron reaction that fades within twenty-four hours (V. Jaksch).

LITHURIA.

Definition.—A persistent excess of uric (lithic) acid and urates (lithates) in the urine.

Normal urine contains about 0.4 part of uric acid to 1000 parts of urine (about gr. x—0.648—*per diem*), or it exists in the proportion of about 1 to 45 of urea, the principal solid constituent.

Etiology.—The causes of lithuria, as seen in certain conditions in which this metabolic change occurs, may be put down to be chiefly as follows: (1) Lithemia (uricemia; uric- or lithic-acid or gouty diathesis); (2) gout and rheumatism; (3) fever; (4) leukemia and pernicious anemia; (5) pulmonary affections in which the interchange of gases is interfered with; (6) a highly nitrogenous diet. Certain other conditions of the urine may diminish its power of dissolving the uric acid shortly after voidance, and may cause a deposit that should not be mistaken for an excess. Such are—(*a*) temporary increase in the quantity of uric acid from an over-indulgence in nitrogenous food; (*b*) temporary high acidity; (*c*) deficiency in mineral salts.

Diagnosis.—The urine has a high specific gravity, a deep red-yellow color, and a marked acid reaction, although, rarely, uric acid is formed in neutral or alkaline urine (Vierordt). Albumin may be present in small amount at the same time. On standing the uric acid is deposited in yellowish-red or "Cayenne pepper" grains, composed of microscopic uric-acid crystals. Chemically pure uric acid is colorless, but that deposited from urine has always this yellowish-red appearance both to the naked eye and under the microscope. Examination with the latter shows a great variety of rhombic prisms—"whetstone-shaped," "crosses," "lozenges," and other many-shaped and sized crystals—single and in agglomerations.

Test.—The *murexid reaction* may be obtained by evaporating a little urine in a watch-glass or porcelain dish, adding a few drops of strong nitric acid, and heating to dryness again; this is allowed to cool, and a drop of liquor ammoniæ added, when a beautiful purple shade of murexid will appear if uric acid be present.

Urates.—These are increased in pathologic conditions that give rise to uric acid in excess, and are usually present with the latter in some quantity. It is not rare, however, in healthy individuals for a deposit of urates to occur in concentrated urine exposed to a cool atmosphere. Urates appear also in the scanty urine due to the profuse perspiration and diarrhea of renal congestion, in fever, from renal calculi, and after a meal rich in albuminous elements.

Urates occur principally as acid sodium urate, calcium urate, and ammonium urate. They appear macroscopically as a flesh-colored or "brick-dust" (lateritious) sediment; this is usually abundant and very finely granular in appearance, while the urine above is cloudy. It is quite characteristic that upon heating such urine it becomes clear, the urates being completely dissolved. Microscopically, the sodium and calcium salts of uric acid occur as needle- or dumb-bell-like crystals or as fine, dark, amorphous granules. Ammonium urate is found in alkaline urine, often with triple phosphates when some putrescence has ensued. It is seen in dark-brown or green spiculated spherules; these are sometimes called "hedge-hog" or "thorn-apple" crystals. On the addition of a drop of hydrochloric acid under the cover-glass uric-acid crystals may be seen to develop.

OXALURIA.

Definition.—A persistent excess of calcium oxalate in the urine. A few crystals may occur in normal urine that has been standing for a long time.

Transient oxaluria may follow the ingestion of sub-acid fruits, as pears, or of vegetables containing oxalates, as rhubarb, tomatoes, sorrel, spinach, asparagus, and cauliflower.

Pathology.—Oxaluria has been described by some English physicians as an independent disease or special diathesis in which marked dyspepsia and hypochondriasis or neurasthenia are associated. The condition is better explained, probably, as one of a disturbed metabolism—particularly of the fats and carbohydrates—in which the oxaluria and the nervous symptoms are manifestations analogous to the lithuria and the irregular gouty symptoms of lithemia. Oxalates and lithates are not infrequently found together in the urine of those subject to the gouty habit. Uric acid may be oxidized to oxalic acid. The ultimate source seems to be the nucleins and nucleo-albumins. Oxaluria is also present in wasting diseases, as in tuberculosis and diabetes mellitus, and in the cancerous cachexia; it may appear in catarrhal jaundice, spermatorrhea, also with the "mulberry calculi," and in general paresis of the insane. Slight albuminuria is not infrequently associated.

Diagnosis.—Oxalate-of-lime crystals appear in the urine in two forms—most commonly as minute, regular, highly-refracting octahedra, or, more rarely, as hour-glass- and dumb-bell-shaped crystals.

The octahedral crystals have two crossed axes, giving a star or envelope-like appearance. Oxalates sometimes give a glittering and scintillating effect to floating mucus in urine that has undergone fermentation.

The **prognosis** is usually favorable.

In the **treatment** of oxaluria the nitro-hydrochloric acid in 2-drop doses is a useful agent.

PHOSPHATURIA.

Definition.—A persistent excess of phosphates in the urine.

Phosphoric-acid salts may be precipitated in normal urine that has become temporarily alkaline. These acid sodium and potassium phosphates in normal acid urine are derived from the alkaline phosphates (neutral sodium and potassium phosphates) of the blood. In normal urine 1.2 parts of alkaline phosphates per 1000 and 0.8 part of earthy phosphates are appreciable.

Etiology and Pathology.—Conditions that produce an alkaline fermentation of the urine cause a deposit either of *amorphous earthy phosphates* or of *crystalline phosphates*. They are also found in the decomposing urine of chronic cystitis, of phosphatic vesical calculi, of paralysis, and in undue retention of urine. In this alkalinity, due to the ammoniacal fermentation of urea, ammonium carbonate reacts with the phosphates of magnesium to form the triple ammonio-magnesia phosphatic crystals, the commonest variety of phosphaturia. Here the phosphates are deposited before or immediately after the urine is passed, giving a milky appearance to the last portion. Deposits of phosphates, and especially of triple phosphates, by no means, however, indicate an actual phosphaturia. This must be determined by chemical analysis. *Amorphous carbonate of lime* in small quantity may be present also if the urine is strongly alkaline and ammoniacal (Beale). The *calcium phosphates* are generally more abundant than the magnesian, and may be found in cases of nervous or atonic dyspepsia, neurasthenia, melancholia, and other debilitated conditions. Whether or not a marked precipitate of phosphates means any detriment to nervous tissue alone has not been determined precisely as yet.

A quantitative estimation of the daily output of phosphates shows a decided increase in wasting diseases, as tuberculosis, leukemia, chronic articular rheumatism, and acute yellow atrophy of the liver. The phosphoric acid is not increased, however. The so-called "phosphatic diabetes" is characterized by polyuria, excessive phosphaturia, thirst, emaciation, and nervous disturbances (Tessier).

Diagnosis.—Phosphatic urine has usually a stale, ammoniacal odor, a whitish turbidity, and a copious light-colored granular sediment falls on standing. Microscopically, the calcium phosphate crystals appear singly as "knife-blade," "arrow-head," or "slender wedge-shape," or in stellate clusters. Acetic acid dissolves them. The ammonio-magnesian phosphate crystals are transparent rhombic or triangular prisms, large and small—"coffin-lid-shaped." These also are soluble in acetic acid; oxalate-of-lime crystals are not so.

On heating phosphatic urine an increased cloudiness is produced that simulates albumin, but on acidifying, as with a drop of nitric acid, this is cleared up at once.

LEUCINURIA AND TYROSINURIA.

Definition.—The presence of leucin and tyrosin in the urine. These are strictly pathologic substances, and are usually found together. They are products of the decomposition of albumin, intermediary to the formation of urea, and are most apt to be found in the urine, along with biliary matters, in certain hepatic conditions.

Etiology.—The principal causes of leucinuria and tyrosinuria are acute yellow atrophy of the liver, acute phosphorus-poisoning (in both of which fatty degeneration of the liver is pathologically conspicuous), specific infectious diseases, as typhoid fever, small-pox, and yellow fever, and pernicious anemia. Leucin and tyrosin have also been found (Kirkbride) in the urine of a patient with erysipelas, during two days of the decline.

Diagnosis.—Of the two substances, leucin is the more soluble, and hence is rarely found in the urinary sediment. Tyrosin, on the other hand, may be discovered sometimes as a fine greenish-yellow deposit. Bile-pigment may be found not infrequently in urine containing leucin and tyrosin. A trace of albumin also may be present, while the urea is, as a rule, markedly diminished. Leucin and tyrosin may be detected by evaporating a few drops of urine on a glass slide and examining microscopically. Leucin appears in the form of slightly glistening, greenish-yellow spheres that may show radiating lines and concentric rings. Tyrosin is recognized by the slender tufts of fine, needle-like crystals arranged in star- or cross-like fashion.

If the residuum after evaporation be heated with a drop of nitric acid, slowly evaporated to dryness, and then touched with a drop of sodium hydroxid, the leucin, if present, will assume a yellowish-brown hue.

Tyrosin becomes red in color when boiled with Millon's reagent of mercurous nitrate, or it is demonstrated by a violet color when carefully warmed with a little sulphuric acid, and then treated with a drop of the solution of phenic chlorid.

CYSTINURIA.

Definition.—The presence of an excess of cystin in the urine. Cystin in minute quantity may be found sometimes in normal urine. It contains sulphur. The *causes* of cystinuria have not been well made out, though *hereditary influences* seem to have an important bearing on the etiology. Insufficient nitrogen metabolism, as occurs similarly in such allied conditions as gout and obesity, seems to give rise to cystinuria.

On account of the insolubility of cystin any marked quantity would be deposited in the urine. Cystin calculi sometimes result, though cystinuria may exist without the presence of a cystin calculus.

Brieger points out a probable significance in the discovery of the associated presence of ptomaïns with cystinuria. Thus, in certain infectious diseases, as intestinal mycosis, a ptomaïn-cystinic product is supposed to be formed, then to be absorbed, and finally decomposed in the urine, thus setting free the cystin. Cystitis may be caused by ptomaïns.

Diagnosis.—The sediment is light, and not very unlike that of the amorphous urates. It is not dissolved by heat, however, though soluble in ammonia. Under the microscope cystin occurs in the form of thin, transparent, hexagonal crystals. Care should be exercised in forming a diagnosis of cystinuria that a contamination with iodoform be excluded, since the microscopic appearance of that substance is similar to that of cystin. On account of the sulphur contained in cystin, a test may be employed by which hydrogen sulphid is liberated, as by boiling the suspected urine with a solution of lead oxid and sodium hydroxid, black lead sulphid resulting from the reaction if cystin be present.

VARIOUS OTHER CONDITIONS.

Urea.—This occurs in solution in the normal urine as a product of the perfect decomposition of the nitrogenous elements of food and tissues. In 1000 parts of urine about 20 parts are constituted of urea (2 per cent., equivalent to about gr. 450—30.0—daily). The quantity of urea is *increased* in the urine after the ingestion of a considerable quantity of proteid food; sometimes after exertion; in acute inflammation and in fevers—either relatively or absolutely, as in pneumonitis; in diabetes and other morbid conditions in which metabolism is accompanied by an increase in the tissue-waste. In febrile states its excretion increases or diminishes with the exacerbations and remissions of temperature respectively.

Urea is *diminished* in quantity in all forms of nephritis, and markedly so in uremia; in organic liver-diseases; in cachectic and anemic states; and in dropsy, inanition, and allied conditions.

The quantitative estimation of urea may be made according to one or more of several methods: Fowler's hypochlorite test (with Labarraque's solution) is perhaps the most practical for ordinary clinical purposes; the hypobromite and Liebig's methods, both requiring special apparatus, are better adapted for the laboratory.[1] Fowler's method is based upon the loss of specific gravity upon the liberation of the nitrogen of the urea. The mean specific gravity of a mixture of 1 part of urine and 7 parts of the solution of sodium hypochlorite is taken while quiescent, and is then subtracted from the specific gravity of the mixture taken after agitation several times during about two hours. The difference which is due to the liberation of the nitrogen (as is shown by the effervescence), multiplied by the factor 0.77, gives the approximate percentage of urea in the urine. This test, however, has a considerable range of error.

Urine evaporated to a syrupy consistence and then treated with nitric acid shows crystalline quadratic plates of urea nitrate.

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[1] See works on Urinalysis.

Chlorids.—About 10 parts of the chlorids of sodium and potassium in 1000 parts of urine are excreted daily. They are *increased* in the urine after muscular exertion, during the resorption of mechanical or inflammatory transudations and exudations, and in intermittent fevers, owing to the destruction of the red corpuscles.

Pathologic *diminution* in the quantity of chlorids occurs in fevers, in the nephritides, in cachectic conditions, and especially in such diseases as pneumonitis, pleuritis, and rheumatism. In the last-named class the chlorids diminish as exudation continues, and may even totally disappear from the urine in extensive pneumonic consolidations, to reappear again with the resorption of the exudate.

Test.—The chlorids may be detected, after first removing any albumin that may be present, by acidulating with a few drops of nitric acid (to keep the phosphates in solution), and by then adding, drop by drop, a strong solution of argentic nitrate. According to the abundance of the resultant white, curdy precipitate of argentic chlorid a rough estimate may be made of the total quantity of chlorids in the urine.

Lipuria is a term applied to the presence of fat in the urine. It may result from the steady use of cod-liver oil or of fatty food, or it may be found in pyonephrosis (Ebstein); in phosphorus-poisoning; in prolonged suppuration; in the lipemia of diabetes mellitus; in the "large white kidney" with fatty degeneration of chronic Bright's disease; in beer-drinkers; and in chyluria. Fatty urine becomes clear upon agitating after the addition of ether.

Lipaciduria, or urine containing volatile fatty acids (acetic, butyric, and propionic), is as yet without diagnostic significance.

Melanuria, or urine containing the pigment melanin, is found in cases of melanotic sarcoma. The urine is dark, either just after being voided or after some exposure and oxidation.

Hematoporphyrinuria (*Urospectrin*).—This term implies the presence of hematoporphyrin (iron-free hematin) in the urine. It occurs after long-continued use (even in small doses—Müller) of certain coal-tar products (sulfonal, trional). Stockton found it in a case of acute ascending paralysis.[1] In addition to the gastric and nervous *symptoms* in poisoning from these substances is a cherry-colored or dark blue-red urine, the abnormal appearance of the latter being due to the presence of hematoporphyrin resulting from the destruction of the red blood-corpuscles. The condition has proved fatal in several cases in which the kidneys were diseased. The urine is always quite acid. According to Garrod, hematoporphyrin is a scanty though constant ingredient of normal urine. He extracts it by adding 100 c.cm. of urine to 20 c.cm. of a 10 per cent. solution of sodium hydroxid. This precipitates the phosphates, which are washed with water and redissolved with rectified spirits. After acidulation with hydrochloric acid the solution shows spectroscopically bands of acid hematoporphyrin. The *treatment* consists in the withdrawal of these drugs and the administration of alkalies.

Pneumatinuria, or gas-formation in the bladder, rarely occurs. Heyse[2] records a case of myelitis in which this condition was present.

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[1] *Amer. Jour. Med. Sciences*, July, 1900.

[2] *Zeit. f. klin. Med.*, 1894, xxiv. p. 130, quoted in *The American Year-Book of Medicine and Surgery* for 1896.

Fibrinuria.—In certain conditions of the genito-urinary tract, particularly pyelitis and ureteritis, fibrinous (and mucous) casts are found in the urine. Fibrinuria may follow nephro-lithiasis (v. Jaksch).

Typhoid bacilluria occurs probably in about 25 per cent. of the cases of typhoid fever (Horton Smith, Gwyn).

Bacteriuria.—There are probably few specimens of urine that do not contain bacteria. Engel has found a great variety of organisms in the nephritides, one of which (a micrococcus of characteristic growth and properties) was present in 17 out of 31 cases, hence regarded by him as the specific cause of some of the cases in this category of diseases. This organism was found in mild types of nephritis, and Engel believes it to be responsible for many instances of the sort beginning as mild forms of "*bacterial albuminuria.*" Warburg[1] reported a case of chill and fever with turbid urine due to large numbers of the Bacillus lactis aërogenes.

Lactosuria.—Lactose is found in the urine of some puerperæ.

Inosituria.—Inosite occurs in the urine in diabetes insipidus.

Alkaptonuria.—Alkaptone is an obscure substance (so called by Bredeker) that is sometimes found in the urine of phthisical cases, or at times in that of patients without any apparent local or general disease. Alkaptonuria seems to be congenital in a few cases. On exposure the urine darkens in color; also upon the addition of liquor potassæ. It gives the sugar-reaction with Fehling's solution (Osler).

Urine as affected by the administration of various drugs—as carbolic acid, salol, antipyrin, and potassium iodid—responds to certain chemical tests, for the study of which the reader is referred to works on urinalysis and clinical diagnosis.

Cholesterinuria has been found in cases of pyonephrosis, hydronephrosis, renal hydatids, epilepsy, and severe dyspepsia.

THE NEPHRITIDES.

BEFORE considering the several varieties of nephritis, and especially the clinical history peculiar to each variety, it may be well first to describe certain general manifestations of renal diseases that are more or less common to all. Reference to these symptoms under the different forms of nephritis will, it is hoped, thus make possible a clearer apprehension of their significance and clinical importance, as well as render unnecessary any further elaboration.

One of these pathologic conditions has already been described—viz. (1) *Albuminuria.* It remains, then, to speak of (2) the *Morphologic constituents* of the urine in nephritis, (3) *Edema* (*anasarca*, *dropsy*), and (4) *Uremia.*

THE MORPHOLOGIC CONSTITUENTS OF THE URINE IN RENAL DISEASE: CASTS, EPITHELIUM, ETC.

1. **Tube-casts.**—These are undoubtedly the most important morphologic elements in the urine of a nephritic. Albuminuria is coincident-

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[1] *Münchener Med. Wochen.*, July 18, 1899.

ally present, and the occurrence together of these two pathologic constituents furnishes indisputable evidence of renal disease. Although, on the other hand, as shown by Martin and Jones,[1] hyaline and granular casts may occur in many pathologic states minus albuminuria. According to the nature and quantity of the casts also may be determined the character and variety of the affection of the kidneys in most instances. Casts, as their name implies, are simply cylindric bodies moulded in the renal tubules, and composed essentially of the coagulable substances in the blood-serum. The coagula of the tubules are mostly albuminous. Other morphologic elements may be mixed with casts—epithelium, red blood-cells, pus-cells, and the granular matter and fat-droplets due to degeneration of the renal epithelium.

Singly, the casts are invisible to the naked eye, but in acute nephritis they may be so abundant as to form a cloudy sediment.

(*a*) Microscopically, the *unmixed* or *hyaline* cast—the commonest—appears either long or short and narrow or broad, of a clear, transparent, homogeneous substance, delicate in outline, and often showing ends with a cheesy—or wax-like—fracture. They may be straight or slightly curved and tortuous, with fine short transverse lines here and there at the borders of the cast. Rarely, a cast may be found equal to a millimeter in length. The so-called *narrow casts* are about equal in width to the diameter of a leukocyte, while the *medium* or *broad casts* are from three to four times this size. They will take either the carmin or gentian-violet stain. Hyaline casts are usually associated with other varieties of casts in nephritis, though in fevers, congestion of the kidneys, chronic interstitial nephritis, and in amyloid kidney they may occur unassociated with other forms of casts (Vierordt).

(*b*) *Granular* casts are nothing more than hyaline casts with fine or coarse granules superadded. The granules represent minute, opaque particles of urates, albumin, fat, cellular débris, and even bacteria (*bacterial casts*). It should be remembered, however, that granular casts may be simulated by casts of coagulated albumin covered with particles of hematoidin, especially in acute nephritis. The hematoidin can be recognized, however, by the brown-yellow coloration.

(*c*) *Epithelial* casts are hyaline casts more or less covered with renal epithelium, indicating an acute desquamative nephritis. The epithelial cells may show evidence of granular or fatty change.

(*d*) *Blood-casts* consist of soft hyaline casts having red blood-cells imbedded in them. These are present in renal hemorrhage and in acute hemorrhagic nephritis.

(*e*) *Waxy* casts are similar in appearance to hyaline casts, though better defined, broader as a rule, and of an opaque, slightly yellowish tint. They often show broken ends. They do not necessarily indicate amyloid disease of the kidney, as was formerly held. They may, however, sometimes show the amyloid reaction with iodin and potassium iodid, and are always suggestive of serious renal disease.

(*f*) *Fatty* casts are such as have left upon and in them fat-droplets or granules, which, if abundant, are indicative of fatty degeneration of the kidney. Cells showing granulation (fatty change) may also be seen.

Rolled casts or *pseudo-casts* (made by sliding a cover-glass over a specimen of urine) of urates, epithelial cells, blood-corpuscles, and débris should not be mistaken by beginners for genuine tube-casts. The same may be said of cylindroids, mucous cylinders, and foreign substances. Nephritis may exist when the casts are always to be found, varying in numbers only, while albuminuria may be inconstant or intermittent.

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[1] *Phila. Med. Journ.*, Sept. 8, 1900.

2. **Epithelium.**—Renal cells are found in the urine of those forms of nephritis that are characterized by a catarrhal or desquamative and exudative process in the tubules. Epithelial cells from the kidney are polygonal or spheric in contour, with an indistinct cell-wall; they have a large oval nucleus, and are either abundantly granular or show a fatty change. These cells are about the size of the white corpuscle.

3. **Leukocytes.**—Only when attached to casts can it be positively affirmed that leukocytes are of renal origin (Strümpell). The pus-cells are frequently seen to be without nuclei in marked or chronic pyuria.

4. **Red Blood-corpuscles** (*vide* Hematuria, p. 938).—In acute hemorrhagic nephritis and in severe renal congestion free red blood-corpuscles are generally to be found.

5. **Fat-globules and granular, fatty-degenerated cells** are seen especially in the subacute and chronic forms of nephritis with fatty degeneration of the proliferated epithelium, or in the fatty stage of large white kidney.

DROPSY OF RENAL DISEASE.

Since, as in other conditions, renal dropsy or edema is an abnormal accumulation of watery fluid transuded from the blood-vessels into the cellular tissues and lymph-spaces, the question arises, "What is the rationale of its development in nephritis?" On the ground that the renal secretion consists principally of water, and that in most forms of nephritis the urine is diminished, it was formerly held that the dropsy was due to the saturation of the tissues with the water that was not excreted by the kidneys. This theory is not fully tenable, however, for there are some cases of edema unaccompanied by any diminution in the daily quantity of urine; on the other hand, certain instances of renal disease in which there is a state of almost anuria show no evidence of dropsy whatever. The failure of any one theory to explain the etiology and pathology of edema has justified the proposal of another and undoubtedly a more plausible one by Landerer—viz., that the relaxation of the tissues (which may be caused by the increased transudation of stasis, or by hyponutrition from hydremia), and their consequent loss of elasticity, prevent that forcing of the lymph into circulation that exists in the normal state, and as a result a watery infiltration of the tissues is permitted. The loss of elasticity or power of resistance in edematous tissues is quite apparent under the skin, and affords a positive means of diagnosis in the pitting produced by the pressing finger.

The dropsy of the nephritides may be either slight or marked, local or general (anasarca), and sudden or slow in onset. It is purely renal in origin perhaps only in acute Bright's disease or in the earlier stages of chronic Bright's disease. In all forms of chronic nephritis the dropsy may be due, in part, to the venous stasis of cardiac incompetency. In chronic interstitial nephritis, especially, edema is slight, and usually is the result of weakness and dilatation of the heart, increasing *pari passu* with the latter.

. I desire to mention here those rare cases of dropsy that simulate Bright's disease in which no satisfactory causative lesion is apparent or discoverable, and also those cases, rarer still perhaps, that have a peculiar family or congenital origin.

Physical Signs.—The recognition of edema is made possible by both *inspection* and *palpation.* Renal dropsy is manifested first by puffiness of the skin of the face, and especially of the eyelids. At other places where there is loose subcutaneous cellular tissue, and in particular where the parts are dependent, dropsy is most apt to be seen early, as under the malleoli of the ankles, the dorsum of the foot, and the scrotum. Later, the limbs and the lower part of the back become swollen, and even the whole body is involved in severe cases. The skin has a peculiar waxy pallor and a glossy appearance. When evident vascular or cardiac changes exist, so as to permit of increased dropsy from engorgement, as in cirrhotic kidney, a cyanotic or muddy color of the skin may prevail.

Pathologic Features.—Dropsy is most constant and most persistently decided in the large white kidney of subacute or chronic nephritis: it is most uncommon and irregular in chronic interstitial nephritis (contracted kidney). The familiar pitting on pressure over edematous tissues is a true indication of fluid under the skin. There is also a doughy or putty-like consistence. In very marked cases of dropsy the deeper parts, such as the muscles, become affected. The serous cavities also in general anasarca show evidences of effusion, and thus give rise to hydro-thorax, hydro-peritoneum, and hydro-pericardium. Less frequently there may be edema of the larynx, uvula, conjunctiva, and other mucous membranes. Edema of the brain, either local or general, may be the cause of grave uremic symptoms in chronic nephritis, or of unilateral convulsions or paralysis and apoplectic seizures. The dropsical liquid is chemically similar to a diluted blood-serum. A minute quantity of albumin and urea is present.

UREMIA.

Definition.—Uremia is the term applied to a group of manifestations, mainly nervous and either acute or chronic, resulting from a toxemia due to the retention in the body of certain products of urinary or renal origin.

Although most common in Bright's disease, uremia may arise also in other diseases, as in gout (gouty kidney), scarlet fever (scarlatinal nephritis), typhus fever, yellow fever, and cholera, in which the kidneys and blood may be seriously affected. Kidneys which, on account of marked structural changes, fail to eliminate the normal quantity of solid constituents are directly or indirectly responsible for an association of the morbid conditions known as uremia.

Our present knowledge of the **pathology** and **etiology** of uremia, as of renal edema, is based solely upon theoretic views. The theory that attributes uremic symptoms to the retention of the excretory products appears to have the strongest proofs to support it; but the positive nature of these substances, or which is the most toxic, or whether several are concerned in the causation or not, remains to be determined. Since the urea and uric acid have been found in increased quantities in the blood of uremic patients, and since these products are diminished in the urine of nephritis, they also were at first supposed to be the cause. Ammonium carbonate, it was alleged by Frerichs, operated in the same manner after it accumulated in the blood in sufficient quantity as a result of the decomposition of the urea by a ferment.

Not only some of the solid urinary constituents accumulate in the blood in uremia, but the water also is only partly eliminated, and its presence in the blood renders the latter hydremic and of lower specific gravity. Notwithstanding the fact that most cases of uremia may be traced to a marked simultaneous diminution in the quantity of urine passed, there remain still certain instances of renal disease in which uremic symptoms appear without any such perceptible diminution. Even more frequent perhaps are those perplexing cases of anuria now and then reported in which no uremic symptoms appear. In the latter instances it is probable that the elimination of products normally excreted by the kidneys may be accomplished through other channels, as by the skin and bowels; in the former it is still likely that the solid urinary constituents are retained, even with an undiminished quantity of water excreted.

Traube's theory of the cause of uremia, particularly of the nervous or cerebral manifestations, was that it is an acute edema of the brain—local or general—with cerebral anemia. This would seem to explain certain cases of nephritis, as already mentioned, in which a fair amount of urine and solid constituents are passed; also cases of anuria due to urethral obstruction in which no uremic symptoms appear; and certain cerebral disturbances. But with our present knowledge of the chemico-pathology and of the clinical cause of the uremia of nephritis in all its forms there is, I think, no doubt that most cases are caused by the toxemia produced by the retention of the mass of excrementitious substances due to an abatement of the renal functions.

Delafield, however, attributes the sudden violent motor symptoms of acute uremia to a contraction of the arteries from some unknown cause other than blood-contamination.

The **symptoms** of uremia may be either acute or chronic in onset, severity, and course. In **acute uremia** the *severest nervous symptoms* come on suddenly; they last but a comparatively short time, and terminate fatally, with convulsions and coma, dyspnea, feeble cardiac action and pulse, fever, and pulmonary edema. These acute symptoms, however, are not infrequently preceded by mild *uremic prodromes*, as headache, somnolence, nausea, malaise, slight dyspnea, and uneasiness.

Chronic uremia is characterized by the *absence* of the marked symptoms referred to above, the milder manifestations alone appearing and lasting over a considerable length of time. Here the general prostration, the feeble cardiac and arterial states, the occasional stupor and delirium, transient dimness of vision, anorexia and nausea, irregularly hurried breathings, and muscular twitchings, indicate the grave condition of the patient. To gain a more thorough knowledge of this interesting and serious complication of renal disease a divisional study of the symptomatology is necessary.

Cerebral Symptoms.—These vary from a slight headache, tremors, and the restlessness of anxiety to the most violent maniacal delirium and convulsions; from somnolence, low muttering, and mental stupor to profound coma; and from slight visual disturbances to complete amaurosis. The onset of a noisy delirium, and less commonly of a marked mania, is often abrupt, and may be the first manifestation of Bright's disease in an individual. Delusional insanity (*folie Brightique*) is seen in a few cases. Bischoff has observed only two cases of purely uremic psychoses among 3000 cases of insanity, and believes that a neuropathic tendency, chronic alcoholism, and pregnancy are the most important predisposing causes. Melancholia and the delusion of persecution, with suicidal and homicidal tendencies, may thus occur. The most characteristic symptom of uremia, however, is the convulsion (uremic eclampsia). *Uremic convulsions* are epileptiform in type, although they may be either unilateral or local—of the Jacksonian form of epilepsy. They are supposed to be due to a local or general edema of the brain, and are probably allied to the *apoplexia serosa* of early writers (Osler). The convulsions of uremia may come on suddenly or may be preceded by headache, vertigo, dropsy, nausea, and vomiting. As in the epileptiform convulsion, after the early tonic rigidity there may follow at short intervals the clonic spasm, with cyanosis, fever, and contracted arteries, and the intervening periods of unconsciousness, shallow or noisy respiration, and slow, hard pulse. *Coma* may come on gradually as well as during the convulsive attacks. It may be preceded by headache, apathy, and insomnia, and continue progressively to deepen for a long time. A *typhoid state* not infrequently accompanies uremic coma. The temperature is usually lowered, and moderate dilatation or contraction of the pupils may be evidenced.

Uremic Amaurosis.—Blindness may follow uremic convulsions, or, rarely, it may come on without motor disturbances. It is of purely centric origin (the cortex of the occipital lobe), and its duration is short, lasting but a few days in most instances. *Uremic deafness*, which is probably also of centric origin, is a less common manifestation. Other nervous phenomena, as hemiplegia, monoplegia (from cerebral or spinal congestion or edema), contractures, aphasia, pruritus, paresthesiæ, and cramps in the calf-muscles are not so frequent in occurrence.

Circulatory Disturbances.—The pulse is moderately slow, tense, and full in uremia, but with the onset of acute and severe symptoms, as convulsions, it usually becomes accelerated, small, and feeble. The heart's action is labored and feeble.

Respiratory Symptoms.—Renal dyspnea, which is sometimes called "uremic" or "renal asthma," is a marked, rather constant, and often an early symptom of uremia. The respirations are deep and often stertorous in coma, or they may be irregular, accelerated, and shallow, sometimes assuming the Cheyne-Stokes type. Dyspneic attacks are especially apt to occur at night. In chronic uremia slight dyspnea may be continuous for a long time. Again, alternating paroxysmal exacerbations may arise. The uremic dyspnea is probably due in most cases to the toxemia affecting the respiratory nervous centers. It may, however, be the result of cardiac weakness or of dropsy or pulmonary edema.

Gastro-intestinal Symptoms.—Uremic stomatitis is generally seen. The breath is foul, the tongue, lips, and gums are red, swollen, and painful, and the saliva is increased. Uremic vomiting is also usually of centric origin, though it may be provoked by the irritation of the gastric mucosa, caused by the vicarious elimination of the urea and the decomposition of the latter into irritating ammonium carbonate. The vomiting may come on suddenly and be persistent. Uncontrollable hiccough and sometimes uremic diarrhea may be associated. The irritant action of the ammonium carbonate on the intestinal mucous membrane may produce a catarrhal or diphtheritic inflammation, and ulceration even (Grawitz). Uremic diarrhea may also exist apart from any marked gastric disturbances.

General Symptoms.—The *skin* of the face is usually pale in uremic coma. *Urea* may be excreted by the sweat-glands, and may be seen as minute glistening crystals in some of the cutaneous furrows after the evaporation of a free sweat. The skin is often harsh and dry, as in chronic interstitial nephritis. Uremic pruritus is probably the result of the peripheral irritation of the cutaneous nerves by crystals of urea. The *temperature* is generally lowered, but uremic fever frequently accompanies the convulsions or they may be preceded by "uremic chills." In some cases the temperature rises to 105°–107° F. (40.5°–41.6° C.) just before death, whilst in other cases, characterized by a profound and lasting coma that deepens into collapse, the temperature may be so low as 91° or 93° F. (32.7°–33.8° C.).

There is not infrequently an *ammoniacal odor* about a uremic patient. The *urine* is diminished in quantity, is generally highly albuminous, and deficient in urea. A previous dropsy is sometimes markedly reduced upon the appearance of acute uremic symptoms.

Duration and Prognosis.—Acute uremia is manifested by coma and convulsions, seldom lasting more than a few days. Chronic uremia, in which milder nervous symptoms, nausea and vomiting, and dyspnea are more prominent, may persist, however, for many weeks. While a grave condition, uremia, even in its most acute and violent forms, is not at once necessarily fatal, for under proper treatment—as by venesection, for instance, followed by judicious hygienic measures—life may be considerably prolonged. Sooner or later, however, barring a possible death from some intercurrent affection, a fatal result is inevitable.

Diagnosis.—Uremia may be recognized by the history, the marked arterial tension, and the accentuated second sound of the heart; also by the albuminuria (the urine has to be withdrawn), the temperature, and the odor of the breath. The presence of dropsy in some cases is a valuable indication of the nephritic origin of uremic manifestations.

Differential Diagnosis.—Uremic unconsciousness coming on suddenly, as in chronic interstitial nephritis, may simulate *alcoholism*, *cerebral hemorrhage* (*apoplexy*), *cerebral tumor*, or *meningitis*. The points of dissimilarity between the first two conditions and uremia are here tabulated (Herrick):

CEREBRAL HEMORRHAGE.	ALCOHOLIC NARCOSIS.	UREMIA.
Pupils unequal or dilated.	Pupils contracted or dilated; eyes injected.	Pupils generally dilated; albuminuric retinitis.
Stertorous, puffy breathing, and flapping cheek.	No stertorous breathing.	Sharp, hissing stertor.
No odor.	Odor of alcohol.	No odor, unless urinous.

CEREBRAL HEMORRHAGE.	ALCOHOLIC NARCOSIS.	UREMIA.
Paralysis ; hemiplegia.	No paralysis, usually.	No paralysis.
Unconsciousness absolute.	May be aroused.	May or may not be aroused.
Pulse slow and strong or irregular ; arteries often atheromatous.	Pulse frequent and feeble.	Pulse at first strong, later weak and rapid ; tension strong ; arterio-sclerosis.
Coma sudden and deep.	Coma gradual.	Coma gradual or sudden.
Convulsions late ; may be unilateral.	No convulsions.	Preceded by general convulsions, headache, etc.
Urine generally negative.	Urine generally negative.	Urine albuminous.
Apoplectic habit ; heart may show hypertrophy.	Red face and nose, heart often weak, dilated, myocarditic.	Edema and pallor ; heart hypertrophied.

In *meningitis* the mode of onset, the rigidity of the neck, incoherence or mild delirium, photophobia, and pronounced fever point to the distinction.

Uremic coma must also be differentiated from *opium-poisoning* and *diabetic coma*. Chronic uremia must not be confounded with the asthenic state of *typhoid fever* and *acute miliary tuberculosis*. In *opium-poisoning* the pupils are contracted and do not respond to light. Again, in opium-poisoning the respirations are slow, deep, and full, and the patient may answer rationally when aroused. In uremic coma, it will be remembered, consciousness is abolished. In *diabetic coma* the history must be learned, the harsh, dry skin and emaciation noted, and especially are the ethereal odor and the Burgundy-red reaction of the urine (acetone) with the tincture of the chlorid of iron to be observed; sugar is also present.

The **prognosis** is grave, but guarded; it is even favorable in many cases, so far as immediate results are concerned.

Treatment.—This will be detailed in the discussion of the various forms of nephritis. Suffice it to say that the supreme indication is the prompt elimination of the poisons in the blood. When diaphoresis and catharsis fail either in promptness or efficiency, venesection should be employed ; the latter measure is also probably the most reliable in urgent cases of uremic convulsions or coma. The counter-injection (intravenous) of normal salt solution may be indicated in cases of profound weakness threatening collapse.

Bozzoli recommends the subcutaneous injection of sterilized serum because of the gratifying results secured in a number of cases of uremia.

AMYLOID KIDNEY.

Definition.—Amyloid (waxy or lardaceous) degeneration of the kidneys ; it is usually coexistent with a similar degeneration of other viscera.

Pathology.—Macroscopically, the amyloid kidney appears pale, greenish or yellowish-white, firm, and uniformly enlarged, and the surface is smooth, glistening, and often mottled, owing to the prominence of the stellate veins. On section a homogeneous, anemic, or "bacon-like" surface presents itself, particularly in the cortical region. The cortex is wider than normal; the pyramids may be red in color and slightly infiltrated; and the glomeruli may show an infiltration by the glistening, translucent amyloid (albuminoid) material. On the application of Lugol's solution of iodin to the amyloid areas a mahogany-red color is produced. Brushing over the amyloid substance with a solution of iodin, and then with dilute sulphuric acid, gives a blue or violet tint. Similarly used, a 1 per cent. solution of methyl-violet strikes a red color. The capsule of the kidney is thickened, though not always adherent.

Microscopically, the amyloid change is generally found in the early stages to affect the walls of the capillaries of the Malpighian tufts. The walls are swollen with the homogeneous material and the vessel-lumen is diminished or obliterated. The straight uriniferous tubules are also infiltrated later perhaps, the deposit occurring primarily in the membranæ propriæ. A diffuse nephritis is nearly always an associated condition. The tubules generally contain hyaline casts. Fatty degeneration of the epithelium, glomerulites or waxy glomeruli, and a thickening of Bowman's capsule are common in markedly amyloid kidneys. In advanced cases most of the secretory structure becomes atrophied. Amyloid infiltration of the smaller granular kidney is less common than of the large white kidney, with intense parenchymatous changes.

Hypertrophy of the heart is not always present in amyloid disease of the kidneys. Amyloid infiltration of other organs, however, as of the liver and spleen, is usually associated with waxy kidneys.

Etiology.—The causes of amyloid kidney are those of the amyloid change affecting (either simultaneously or nearly so) other organs, as the spleen, liver, and intestines.

Commonly, amyloid disease is marked also in the other solid organs named above; it is secondary to wasting diseases, cachexiæ, and the like. Perhaps the most frequent cause of the waxy kidney is tuberculosis, especially of the lungs ("chronic ulcerative phthisis"): tuberculosis of the intestines also is often associated and aggravates the amyloid infiltration. Next in order are the prolonged suppurations, particularly of the bones, as in osteitis of the vertebræ and hips (usually tuberculous). Chronic empyema, intestinal ulcers, vesico-vaginal fistulæ, and other purulent affections, chronic in nature also, have the same etiologic effect.

Amyloid kidney is often present in syphilis, especially in the tertiary stage, when ulceration of the mucous surfaces and of the bones is present. Rarely, gout, malaria, leukemia, cancer, and chronic valvular endocarditis with insufficiency seem to produce amyloid disease.

Symptoms.—These vary greatly according to the extent to which the amyloid degeneration has encroached upon the normal kidney-structure, and may be overshadowed partially or completely by those of the dominant causal affection.

The *urine* is pale yellow, clear, and variable in quantity, and the amount passed in twenty-four hours is sometimes normal or may be slightly diminished. More frequently, perhaps, it is increased, and especially in marked or advanced cases. The specific gravity is apt to be low (1015–1005), and there is seldom any sediment.

Serum-albumin and *globulin* may both be present in the urine; but a highly significant condition, and one that is seemingly diagnostic, is the high proportion of globulin as compared with the serum-albumin (Salkowski, Senator). *Tube-casts* may be found, but their presence may be only temporary; they are usually wide hyaline or fatty and granular, and are few in number (Fig. 64). The amyloid reaction may be elicited with the hyaline casts; symptoms referable to the kidney are often absent in comparison with those of the nephritides. *Dropsy* is not invariably present, and when present is but moderate in degree and generally in the legs only. It is proportionately prominent with the increase in the anemia, circulatory depression, and wasting of flesh and strength. The latter manifestations, constituting a cachectic appearance, are quite commonly observed in amyloid kidney.

The associated enlargement and the firm, sharp outlines of the liver and spleen are of diagnostic significance. *Marked diarrhea* may be due to coexisting amyloid infiltration of the intestines or to tuberculous intestinal ulcers, and is often seen in advanced cases.

Diagnosis.—This can seldom be made upon the urinary manifestations alone. Important and often necessary adjuncts are the histories of causation and of the associated symptoms and physical signs. Thus, there will be evidenced in most cases tuberculosis, chronic bone-suppurations, or syphilis, while coexisting hepatic and splenic enlargements, wasting, and cachexia are usually present. In any of the diseased conditions mentioned amyloid kidney may be diagnosticated with reasonable certainty upon the development of an increased quantity of pale clear urine of low specific gravity and containing a large amount of albumin, or even with slight albuminuria.

From *parenchymatous nephritis* amyloid kidney is to be differentiated by the history, by the more marked and generally distributed dropsy, and by the albuminuric retinitis that characterize the former. In *chronic interstitial nephritis* there are less marked albuminuria and dropsy, and there are present arterio-sclerosis, cardiac hypertrophy, and a pronounced tendency toward uremic symptoms.

Prognosis.—This varies with the cause. Incipient bone-disease or tuberculosis, with only slight evidences of amyloid change in the kidneys, may be controlled. As a rule, however, the structural alterations are so far advanced, and the constitutional powers of resistance so much enervated, before the amyloid infiltration can be distinctly apprehended that in the majority of instances the prognosis is entirely unfavorable. In decided cases death ensues in from several weeks to as many months.

Treatment.—This also depends upon the causal affection. Hygienic and dietetic measures are always useful, however, with a view to improving the general nutrition. The iodid of iron has been recommended as an alterative, and easily assimilable and palatable fats and tonics may also be tried. Tuberculous cases require creasote or allied preparations; syphilitics require mercurials and iodids; while malarial subjects do best under the systematic use of arsenic, iron, and quinin.

NEPHROLITHIASIS.

(*Renal Calculi; Pyelitis Calculosa; Renal Colic; Gravel.*)

Definition.—A condition characterized by the formation of fine or coarse concretions in the kidney-substance or in the renal pelvis by the precipitation of certain of the solid urinary constituents.

Varieties.—According to their size, renal concretions are variously termed—(1) **Renal sand,** of which the particles are fine and pulverized; (2) **Renal gravel,** consisting of coarse grains or even of pea-sized concretions; (3) **Renal stone, or calculus,** when larger masses than the preceding exist, either more or less rounded or as stony casts or moulds of the pelvis of the kidney, its infundibula, and calyces (*dendritic* or *coral calculi*).

According to their composition, the chemical varieties of renal concretions are—(1) *Uric-acid* calculi, the most frequent in occurrence. Urates are often associated in the calculus with uric acid, thus producing stratification. These concretions may occur as sand, gravel, or large stones: they are usually quite hard, reddish-brown or black in color, and have a smooth though irregularly-shaped surface. The fracture is crystalline, and in the larger calculi often shows the alternating layers of uric acid and ammonium urate. Pure uratic stones may occur in children.

(2) *Calcium-oxalate* concretions occur more rarely in the kidney. They constitute the so-called "mulberry calculi," from a fancied resemblance to the mulberry, owing to their dark-brown or black color and very irregular and nodulated or prickly appearance. They are also quite dense; lamination, however, is not common, although they are sometimes formed about a uric-acid nucleus.

(3) *Phosphatic* calculi of the kidney are still less common than the oxalate, but they are more common in the bladder. They may consist of calcic phosphate or ammonio-magnesic phosphate, and may possibly be associated with calcic carbonate. Phosphatic salts are most often deposited secondarily about uric-acid or oxalate calculi in the alkaline urine of a cystitis set up by the irritation of the true renal stones. Phosphatic calculi are grayish-white in color and are comparatively soft.

(4) Renal stones composed of *cystin*, *xanthin*, *carbonate of lime*, *fatty or saponaceous matters* (urostealith), *indigo*, and *fibrin*, though of extreme rarity, have been occasionally reported. Cystin calculi have a pale-yellow color and a waxy luster.

Pathology.—The anatomic changes of the kidney vary with the degree and persistence of the irritation, the size of the calculi, and their passage or retention. Sometimes numerous granular and pea-sized concretions are found in the renal pelvis, with desquamated epithelium and a turbid urine. Interesting cases are those in which a dendritic stone occupies a great portion of the atrophied kidney-substance, as well as the entire pelvis of the organ. In one of my own patients the left kidney was, apparently, nearly twice the normal size, owing to the presence of a large coral-calculus (uric acid and urates), connected by an isthmus with a rounded stone in the inferior portion quite as large as a large walnut. The pelvis of the right kidney also contained a dendritic calculus.

Secondary Lesions.—Perhaps the most usual result of renal concretions is a pyelitis: this may be simple catarrhal, diphtheritic, or purulent, with or without hemorrhages, depending upon the intensity of the mechanical irritation. A pyelo-nephritis may follow in severe cases, as may even a general suppuration (pyonephrosis) or perinephric abscess and perforations. Renal pus-cavities are sometimes found *postmortem* containing numerous small stones. Hydronephrosis is another important pathologic sequel, in which the cause is to be attributed to the blocking of the ureter by an erstwhile passing stone or by the closing of the aperture of

a ureter from within the pelvis. Pressure-necrosis and perforation may thus be induced. Owing to the prolonged pressure of a dendritic calculus, there is commonly a distinct and marked atrophy of the renal parenchyma, resulting in chronic diffuse nephritis with little or no exudation.

Etiology.—The definite causation and the exact manner of formation of renal concretions are still unestablished. We may infer not a little, however, with some good reason, since the *predisposing causes* are rather distinct. Thus, in children and in advanced life (before 15 and after 50 years of age—Purdy) the occurrence of calculi is most common, the uratic variety being most frequent in the former and the uric acid in the latter. Men are subject to nephrolithiasis more often than are women. The uric- or lithic-acid state (lithemia), gout, and the various influences that induce these conditions, as an excessive meat (proteid) diet or a sedentary life, seem to predispose to stone. Heredity, I believe, plays a prominent part in many cases.

Broadly speaking, any habit of the system that encourages the precipitation of insoluble abnormal ingredients or of normal ingredients in excess, owing to chemical changes in the urine, tends to the formation of calculi. It should be stated, however, that the *primary causes* of calculus-formation is the presence of some substance in the urinary tract that affords a nucleus about which the successive layers of crystals may deposit and adhere, such as bits of mucus, colloid material, epithelial shreds, parasitic ova, bacteria, blood-clots, and tube-casts.

It is generally believed that the requisite conditions for the formation of a uric-acid renal calculus are—a highly-acid urine, an excess of uric acid, a low percentage of salines, and deficiency of the normal urinary coloring-matters.

Symptoms.—These may be slight, progressive, and chronic, or they may be intensely acute and comparatively short in duration, though subject to repetition—*i. e. renal colic.* It is not unusual for patients to pass uric-acid sand and gravel for years without much complaint. A sudden blocking of a ureter, however, or a slowly-passing stone of distending dimensions produces great agony at times. A smooth, snugly-fitting dendritic calculus in the pelvis may not cause any symptoms for years until the destruction of tissue by its weight and mechanical irritation ensues; there is then a progressive failure of health, a constantly increasing pain in the back, *occasional hematuria*, tenderness on pressure over the diseased kidney, both anteriorly (deep) and posteriorly, and finally *uremia* and death.

The characteristic symptoms of stone in the kidney appear as an attack of *renal colic.* This happens when a calculus in its passage down the ureter acts as a mechanical irritant, or when it is caught and stopped in the passage. The large "gravel" or pea-sized and more or less rough stones usually cause the attack, which comes on, as a rule, quite suddenly, although it may be preceded by a chill and some general uneasiness or by slight pain in the region of the kidney. It may be excited by a sudden muscular effort. The pain is tearing in character, and rapidly reaches an agonizing maximum of severity, starting from the lumbar region and extending down along the ureter into the groin, and often into the testicle and inner side of the thigh. The paroxysm may appear in the form of a diffuse abdominal and lumbar pain in some instances. There is local tenderness on pressure, and nausea and repeated vomitings are frequent. The patient is often collapsed, and perspiration, a rapid, small, and feeble pulse, trembling, anxiety, bodily twistings about, convulsions even, and syncope may ensue. There may be moderate fever. The *urine* is scanty or may be suppressed for a time, and is often bloody. Frequent and painful attempts at urination are made, with the passage of but a few drops at a time, owing perhaps, in part at least, to a reflex spasm of the vesical sphincter (vesical tenesmus). The presence of pus and of pelvic epithelium in the urine indicates a pyelitis. When a large quantity of clear urine is passed, as sometimes happens, it may be looked upon as having come from a healthy kidney.

The *paroxysm* of renal colic ends when the impacted stone passes out of the ureter. This may occur within a few hours or it may take several days; in instances of the latter type the attacks of renal colic may be intermittent.

Recovery is not always complete immediately upon the evacuation of the stone. The previously retracted testicle may be painful and swollen for a little while, and there are apt to be aching and soreness over the affected kidney and ureter.

In certain severe cases of mechanical irritation the symptoms of pyelitis, pyelo-nephritis with abscess, or hydronephrosis may be superadded. Anuria and uremia result from simultaneous obstructive suppression of the urine upon both sides.

Nephrolithiasis as a *chronic affection* may exist for many years, with recurring paroxysms of renal colic. I observed a case for five years that had extended over a period of thirty years, until it finally came to necropsy. Between the attacks of colic the patient may be entirely comfortable, save perhaps an occasional burning in the urethra on micturition, owing to a highly-concentrated, acid urine or to the passage of minute uric-acid granules. There are apt to be pain and tenderness over a kidney containing a large imbedded stone. A smoky-hued urine, due to slight hematuria, is also sometimes present in long-standing cases of renal calculus, particularly after exertion.

A *renal intermittent* fever, simulating malarial paroxysms, may occur in nephrolithiasis, and is analogous to the hepatic intermittent fever of cholelithiasis.

Pyelitis—simple or purulent—with late involvement of the kidney-parenchyma (pyelo-nephritis) is a frequent concomitant of chronic nephrolithiasis. The presence of pus in the urine is constant, with an absence of renal epithelium in cases of an abscess-cavity of the kidney. In ordinary pyelitis the pyuria is often intermittent.

The general health of patients with nephrolithiasis is, as a rule, remarkably good. Anorexia is not only seldom present, but such persons are habitually free and good livers. Persistent headaches with nausea, however, should warn one of uremia. Splenic and hepatic enlargement may be found with prolonged suppurative pyelo-nephritis, indicating amyloid disease.

Diagnosis.—This resolves itself into a study of the diagnostic characters of (*a*) the attacks of renal colic, (*b*) of the underlying systemic condition in general, and (*c*) the renal condition in particular that renders these attacks possible. The latter can be discovered only by a careful and continuous study of the clinical history and urinary manifestations as outlined in previous paragraphs.

Nephrolithiasis may be positively diagnosed in a case in which, after sudden, agonizing, colicky pain, referred to either lumbar region and radiating down the ureteral course to the testicle, a concretion is found to have passed with the urine. It is therefore necessary in a suspected case of renal colic to pour the urine through a fine sieve as soon as passed. The more recent improvements in the operative technic for producing the Roentgen rays enable us to detect renal calculi with considerable accuracy as to their number, size, and relative position.

Differential Diagnosis.—Renal colic must not be taken for *biliary or intestinal colic.* The antecedent history is of great value in arriving at a diagnosis. In biliary colic there may be jaundice, and pain referred to the upper rather than to the lower abdominal zone, both of which symptoms are absent in renal colic; while in the latter the disturbance of micturition and the character of the urine, especially the hematuria, are characteristic.

In intestinal colic the griping pain is usually most intense in the umbilical region, is often relieved by pressure, and is associated with tympanites and constipation; it has usually a dietetic origin, while the renal and urinary symptoms are absent. The exclusion of *lumbodynia* and *lumbo-abdominal neuralgia* is not so difficult. The differentiation of the varieties of calculi from the symptoms is not positive. It has been suggested, however, that the oxalate stones usually cause the sharpest pains and the hematuria. Right-sided ureteral pain felt over the lower abdominal region may be confounded at first with *appendiceal colic.* Musser has found the pain of renal colic to be more paroxysmal and less uniform in location than in the latter however. Early *renal tuberculosis* (*vide*) with its hematuria and pyuria must be differentiated from renal calculus also.

Prognosis.—This should always be guarded, owing to the possible dangers and complications that frequently attend nephrolithiasis in all of its forms. Thus the passage of gravel without marked symptoms tends to persist or recur—in both events an unfavorable tendency, since subsequent formations are apt to be larger and cause serious symptoms. An attack of renal colic may itself be fatal. Large latent calculi (dendritic), of long standing, are nearly always incurable, and in most instances lead to such grave complications as pyelo-nephritis, pyo- and hydronephrosis, perinephric abscess, and uremia.

Treatment.—Paroxysms of renal colic call for prompt relief. This is best afforded by hypodermic injections of morphin and atropin, coupled with hot baths or fomentations applied to the loins. The free use of hot drinks, as lemonade, soda, or plain water, is also helpful in promoting the passage of the stone. Drinking large quantities of glycerin mixed with water has proven of service in some cases. Cases of excessive suffering require the inhalation of chloroform.

The treatment of the nephrolithiasis without or between attacks of renal colic is most important. First to be considered are the **hygienic** and **dietetic** measures, for in mild and uncomplicated cases much can be done to prevent the aggravation of the disorder, and at least the formation of larger concretions may be delayed. When the tendency is to uric-acid gravel (the commonest variety), the patient should live a regular, calm, steady, and temperate life. Exercise should be so managed that it may be taken rather moderately in the open air, and with a view to preventing additional weight in persons of fair nutrition and to promoting a reduction of weight in the obese. In short, the exercise should be sufficient to thoroughly use up all nitrogenous food, so that the formation and elimination of urea may be increased to normal and the quantity of uric acid diminished. Hence I would strongly advise a clinical study of the percentage of urea in the urine (*vide* p. 961).

Over-indulgence in food, particularly in red meats (liver, sweetbread, and similar nuclear food), should be prohibited, owing to the ready formation of uric acid from the latter. Alcohol should be taken seldom, or, better, not at all. On the other hand, since the urine is apt to be scanty and highly acid, the patient should be encouraged to drink freely of plain and alkaline waters, artificial and natural. The value of various pure spring-waters as diluents is undoubted, the Buffalo, Londonderry, and Otterburn Lithia, the Saratoga, Bedford, and Poland waters, all being distinguished for their purity. More marked and more generally useful for their alkalinity are the Carlsbad, Vichy, and carbonated waters. In cases characterized by occasional hematuria the Rockbridge alum-water may be tried. Plain soda-water and lemonade may be used as adjuvants.

The **medicinal** treatment of nephrolithiasis is aimed to secure a solvent and disintegrating action upon the stones; it is symptomatic. It is extremely doubtful whether stones once formed in the pelvis of the kidney and remaining there are ever dissolved, though certain drugs would seem to have had an eroding effect in some instances, and they are to be recommended as useful in preventing the formation of new deposits. Lithium citrate or carbonate in 5-grain (0.324) doses in tablet form, three or four times daily, has been generally employed for the purpose. Sodium phosphate and the vegetable salts of potash, as the citrate, acetate, and tartrate, are useful. Much water, especially the carbonated, should be drunk, along with doses of the above, in order to facilitate the solvent action, and in this way relieve, in a measure, the local distress and pain. Recently piperazin, lycetol, and urotropin have been brought forward as uric-acid-calculus solvents by some clinicians, and that they have such action as is claimed has been proved beyond a doubt in certain cases. Whilst they deserve a further trial in nephrolithiasis, it is too much, however, to expect to look for positive and successful results in every case.

Recently, Van Noorden and Strause have recommended calcium carbonate (gr. x–xv—0.648–0.972—or more thrice daily). The theory is that the calcium unites with the acid phosphates in the intestines, and thus reduces the deuterophosphates in the urine, leaving the protophosphates to dissolve the uric acid. They report excellent clinical results.

The reaction of the urine must be tested at stated intervals and kept faintly acid. Should the urine become alkaline, the alkaline treatment must be suspended for a while, or a secondary deposit of phosphates about the uric-acid stone may be induced. Nagging lumbar pains may be relieved by occasional doses of such analgesics as phenacetin, belladonna, hyoscyamus, codein, and indirectly by the sweet spirits of niter, buchu, and uva ursi. Renal hemorrhage may be controlled effectually by the

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ACUTE NEPHRITIS.

Synonyms.—*Acute Bright's Disease; Acute Diffuse Nephritis; Acute Parenchymatous Nephritis: Catarrhal, Tubal, Desquamative, and Glomerulo-nephritis of Acute*

Definition.—An acute inflammation of the kidneys, more or less
It may be either of a mild, severe, or grave character. Delafield describes three varieties of acute renal inflammation under the common synonym of acute Bright's disease, as follows: (1) *acute degeneration of the kidneys,* (2) *acute exudative nephritis,* and (3) *acute productive nephritis.* This division is of etiologic and pathologic importance and interest, rather than of clinical necessity or practical value.

Pathology.—The anatomic changes in, and the appearances of, the kidneys vary considerably in different cases according to the degree of involvement. From the very mild to the gravest cases of nephritis there is an intermediate series of continuously more marked pathologic changes in the renal tissues. These depend greatly on the amount of poisonous material circulating in the kidneys and eliminated by them, as well as upon the intensity and duration of its noxious action.

In the mildest cases the *macroscopic* appearances of the kidneys may present nothing distinctly abnormal. As a rule, however, the organs are slightly enlarged, swollen, and somewhat softened. These conditions are more evident when the interstitial exudation is abundant and when inflammatory edema is evident. The kidneys may be reddened and congested and appear bloody on section, or they may be pale and mottled. In examples of the former, hemorrhages may be formed beneath the cap- (*hemorrhagic nephritis*), though it is more common to see red ...remia alternating with opaque, whitish portions on both cut-surfaces of the kidneys. The cortex especially is pale, or slightly congested in the mildest cases, and

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Acute degenerative changes are frequently found in the acute
s diseases or when inorganic poisons have been introduced into
ly. In phosphorus-poisoning actual fatty degeneration of the
lium may be found, this either proceeding from the cloudy swell-
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h in severe cases.

True acute nephritis is not only characterized by changes of the renal epithelium (the parenchyma), but the inflammatory exudate (serum, leukocytes, and erythrocytes) is found between the tubules. The kidneys show different stages of the process in different portions. In some places there is only a slight cellular infiltration of the intertubular tissues; in others, besides the desquamation of necrotic epithelial cells and the presence of hyaline casts in the tubules, the interstitial tissue is swollen by the coagulated sero-fibrinous exudate, abundant leukocytes, and some red blood-corpuscles. It should be stated that the inflammatory exudate collects also in the Malpighian bodies and tubules. The epithelium lining the latter, especially the convoluted portion, is often flattened, and the tubules themselves may be dilated and choked with degenerated cells, or, more frequently in the straight tubules, with hyaline casts. The white blood-cells that are found infiltrating the stroma of the kidney are not usually equally diffused, but are collected in foci in the cortex.

The glomerular epithelium of the capsule, and especially that covering the outside of the capillaries of the tufts, is swollen and opaque, and the outlines of the individual capillaries are lost. In most cases of diffuse exudative nephritis new epithelium appears, and a restoration of the glomerular function takes place. In the *productive variety* of acute diffuse nephritis, however, according to Delafield, the lesions consisting of a cellular growth in the capsules and of connective tissue around thickened arteries—are more permanent in character from the first, and hence the increased gravity of the disease. In the more intensely acute cases the new tissue between the tubules is largely cellular; in those of a subacute type it is relatively dense and fibrous.

Anasarca and pleural, pericardial, and peritoneal dropsy are also found in those dying of acute Bright's disease. Cerebral edema, meningitis, and lobar pneumonia are to be mentioned as complicating conditions that are sometimes seen *postmortem*.

Etiology.—Acute nephritis may occur at any time of life, though it use of the fluid extract of ergot, or by alum in 10- or 15-grain (0.648 or 0.972) doses, or by gallic acid in 20- or 30-grain (1.29–1.94) doses.

Efforts to acidify the urine are indicated when the calculus happens to be composed of phosphates or of calcium carbonate. This is more difficult of accomplishment than when it is necessary to reduce the acidity. Saccharin in 2- or 3-grain (0.129–0.194), and benzoic and boric acids in 5- to 15-grain (0.324–0.972) doses, in capsules, seem to be most useful for this purpose. It is claimed for calcium carbonate, again, that it diminishes the phosphates without making the urine alkaline.

The question of surgical interference must be decided in not a few cases; thus, it may be briefly stated that in protracted and obstinate cases of calculous renal disorder, with persistent local pain, a gradually decreasing capacity for work, and evidences of severe pyelitis, pyelonephritis, or, worse, of perinephric abscess, the surgeon must operate. In the simplest cases a nephrotomy or nephro-lithotomy may be performed and the stone removed. Where the renal structure is much damaged it may be necessary to do a nephrectomy. To avoid the increased perils of the latter operation, however, it were better that a nephrotomy were done as early as consistent with the diagnosis of incarcerated pelvic stone and the condition of the patient.

ACUTE NEPHRITIS.

(*Acute Bright's Disease; Acute Diffuse Nephritis; Acute Parenchymatous Nephritis; Exudative, Catarrhal, Tubal, Desquamative, and Glomerulo-nephritis of Acute Course.*)

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Microscopically, alterations may be discovered that are not visible to the naked eye in the very mild cases referred to above. There is simply a cloudy swelling or a granular (parenchymatous) degeneration of the epithelium of the Malpighian tufts, Bowman's capsule, and of the cortical uriniferous tubules. This is not true acute nephritis, however, in the absence of exudative changes in the interstitial tissue. The acute parenchymatous degeneration may be almost exclusively limited to the glomeruli, as in some cases of scarlatina, and hence the term *glomerulo-nephritis*. The cells are swollen, opaque, and irregular in shape, while the cell-contents are granular (albuminoid or fatty). A further advance in the process is seen in cellular coagulation-necrosis or disintegration, desquamation of the cells, and hyaline degeneration of masses of them in the tubules. Acute degenerative changes are frequently found in the acute infectious diseases or when inorganic poisons have been introduced into the body. In phosphorus-poisoning actual fatty degeneration of the epithelium may be found, this either proceeding from the cloudy swelling or developing independently. A rapid necrosis of cells is also met with in severe cases.

True acute nephritis is not only characterized by changes of the renal epithelium (the parenchyma), but the inflammatory exudate (serum, leukocytes, and erythrocytes) is found between the tubules. The kidneys show different stages of the process in different portions. In some places there is only a slight cellular infiltration of the intertubular tissues; in others, besides the desquamation of necrotic epithelial cells and the presence of hyaline casts in the tubules, the interstitial tissue is swollen by the coagulated sero-fibrinous exudate, abundant leukocytes, and some red blood-corpuscles. It should be stated that the inflammatory exudate collects also in the Malpighian bodies and tubules. The epithelium lining the latter, especially the convoluted portion, is often flattened, and the tubules themselves may be dilated and choked with degenerated cells, or, more frequently in the straight tubules, with hyaline casts. The white blood-cells that are found infiltrating the stroma of the kidney are not usually equally diffused, but are collected in foci in the cortex.

The glomerular epithelium of the capsule, and especially that covering the outside of the capillaries of the tufts, is swollen and opaque, and the outlines of the individual capillaries are lost. In most cases of diffuse exudative nephritis new epithelium appears, and a restoration of the glomerular function takes place. In the *productive variety* of acute diffuse nephritis, however, according to Delafield, the lesions consisting of a cellular growth in the capsules and of connective tissue around thickened arteries—are more permanent in character from the first, and hence the increased gravity of the disease. In the more intensely acute cases the new tissue between the tubules is largely cellular; in those of a subacute type it is relatively dense and fibrous.

Anasarca and pleural, pericardial, and peritoneal dropsy are also found in those dying of acute Bright's disease. Cerebral edema, meningitis, and lobar pneumonia are to be mentioned as complicating conditions that are sometimes seen *postmortem*.

Etiology.—Acute nephritis may occur at any time of life, though it more often makes its appearance before than after middle life. Males are more susceptible than females, and particularly when engaged in occupations requiring exposure to cold and wet. The habitual use of alcoholics also is generally a predisposing cause of acute Bright's disease.

The principal exciting causes of acute diffuse nephritis are the following: (1) *Those acting on the skin*, as cold and dampness, extensive burns, and chronic skin-diseases. In many cases it is difficult to estimate whether the influence of alcoholic intemperance predominates or the exposure incident to it. Thus, acute intoxication from beer-drinking itself may cause an attack of acute nephritis, but it is likely that in most instances the direct exciting cause is cold acting upon the individual in his exposed and maudlin condition. The disease may also be attributed at times to exposure to cold and wet irrespective of alcoholic indulgence. It may be presumed with reason that in such cases there is some inherent or acquired weakness or a susceptibility of the kidneys, rendering them the weak links in the visceral or systemic chain.

(2) *Biologic Toxic Agents.*—These embrace the poisons of the acute infectious diseases, though in the majority of cases scarlet fever is the primary affection. Nephritis may supervene during the height of scarlatina, but more often it occurs in the second or third week of convalescence. Other infectious fevers may also cause acute nephritis (smallpox, typhus, typhoid, relapsing fever, epidemic influenza, cholera, diphtheria, yellow fever, measles, chicken-pox, erysipelas, septico-pyemia, acute lobar pneumonia, cerebro-spinal meningitis, dysentery, acute articular rheumatism, and tuberculosis: syphilis is rarely a cause). Acute infectious nephritis may also occur as a primary disorder, and the brunt of the affection may fall either upon the kidney, rather than upon any other part, or upon the organism as a whole, as in the fevers. Mannaberg, among others, has described such cases, and demonstrated streptococci in the urine.

(3) *Chemical Toxic Agents.*—Among the principal irritants of this class are turpentine, cantharides, carbolic and salicylic acids, iodoform, the mineral acids, potassium chlorate, and such inorganic poisons as phosphorus, lead, arsenic, and mercury. The excessive ingestion of highly-acid, spiced, or adulterated foods (as from salicylic acid and lead chromate) may in certain individuals cause acute renal inflammation.

(4) *Pregnancy.*—Here the nephritis (*gravidarum*) comes on in primiparæ, usually in the last months of pregnancy. It is probably caused by renal engorgement due to mechanical pressure, as well as to nutritive disturbances in the kidney, owing to the altered blood-condition.

(5) Finally, latent and insidious *chronic nephritis* may be the cause of an onset of a manifest acute nephritis.

Symptoms.—The *onset* varies with the cause of the nephrit though generally it is rather sudden. Chilliness, nausea and vomiti pain in the back, and, within twenty-four hours, dropsy, are seen in so cases. Children may be seized with *convulsions* (uremic), and adults not less liable to them in severe attacks. Fever may be present, althou it is neither constant nor high. The characteristic symptom is the ear appearance of *edematous puffiness* of the eyelids and face, with pallor the skin. Soon (and sometimes at first, even) a swelling is noticed abo the ankles and legs, and in marked cases the whole body becomes dro sical, so that pitting on pressure may be observed pretty much all ov the bodily surface. In such instances the scrotum and penis or the labia may become enormously distended, the skin having almost a translucent appearance.

Local symptoms, as pain and tenderness in the lumbar region, are often wanting and are never marked. There may be a desire to micturate often, accompanied by slight burning and vesical tenesmus, due to the concentrated urine. In very severe dropsy the tense, dry skin, as of the limbs, may be sensitive or even painful to the pressing finger. Movements of the body are often difficult, painful, and distressing in marked anasarca. Intense headache and backache may precede the onset of uremia.

In mild cases the renal condition may be overlooked unless a urinary examination is made. Prostration may be unnoticed, and the patient feel nothing more than a general malaise.

The characteristics of the urine in acute nephritis are all-important. The total *quantity* passed in twenty-four hours is diminished, and may be very scanty, sometimes amounting to not more than from 5 to 25 ounces (150–740 c.c.). Suppression occurs in some cases of toxic origin, when an acute degeneration or necrosis of the renal epithelium takes place, and in the most severe exudative inflammations. The *specific gravity* is increased to 1025 or more early in the case ; later it may be as low as 1010 or 1015. The *color* is darker than normally, and is usually smoky-red or reddish-brown, according to the amount of blood passed. If the abnormal morphologic constituents are present in great quantity, a more or less abundant flocculent sediment appears on standing.

Microscopically, some red blood-corpuscles and renal epithelium are found, along with the characteristic *blood*, *epithelial* and *granular tube-casts* (Fig. 59). Chemically the urine is *acid*, and on boiling a *thick, curdy precipitate of albumin* forms. The percentage of the latter by weight varies from ¼ to 1 per cent. The urea and gross solids are diminished.

Other symptoms may develop during the course of acute Bright's disease. If *great general edema* is present, physical signs of hydrothorax, ascites, and hydropericardium may be elicited. The first-mentioned condition is bilateral and causes dyspnea; the second increases the dyspnea by pressing the diaphragm upward; and the last impairs the heart's action. Strümpell describes a form of pneumonia—a "stiff inflammatory edema"—midway between lobar pneumonia and broncho-pneumonia, that sometimes develops in severe cases of acute nephritis. Edema of the conjunctivæ, soft palate, and larynx may also occur. Recently, Lapinsky reported a fatal case of acute parenchymatous nephritis in which severe bilateral sciatic neuritis was associated.

The *pulse* is often hard and tense, and, though slow at first, it may become accelerated later. Cardiac hypertrophy of a slight degree may be detected. The aortic second sound is accentuated. Epistaxis is an occasional symptom, and subconjunctival hemorrhages are sometimes seen as a result of uremic convulsions that may not have been witnessed. A very constant symptom is the dry, anemic skin. *Uremic manifestations* may ensue at any time during the course of the disease. They appear early in the most severe cases, with intense headache and backache, vomiting, and convulsions.

The clinical course in other cases differs somewhat from the above, which may be considered as the common form resulting from exposure. Acute nephritis *occurring as a complication of the infectious fevers*, except scarlatina, may be characterized by the very slight degree, or even by the absence, of dropsy. Albuminuria, hematuria, anemia, and uremia supervene in the graver affections. In *scarlatinal nephritis*, however, anasarca is common, and slight edema at least is quite constant. During the period of convalescence tube-casts (granular or fatty granular) may be found in the urine (Fig. 60). In mild affections simply a little albumin and a few hyaline casts reveal the parenchymatous degeneration. In cases of *degenerative nephritis* due to mineral poisoning the subsidence of the acute toxic symptoms may be followed by the *typhoid condition*. In the so-called *nephro-typhoid condition*, where typhoid fever begins with pronounced symptoms of acute nephritis, hematuria may be marked. The *nephritis of pregnancy* is usually gradual in its onset. The albumin increases in amount from month to month, and reaches a high percentage during the eighth and ninth months. Some hyaline or faintly granular casts are found (Fig. 62), and erythrocytes rarely appear in the urine. *Danger of eclampsia* is constant until the child is delivered; but recovery is rapid after the birth of the child as a rule.

That variety of *acute (productive) nephritis* in which there is a tendency to the formation of patches or wedges of fibrous tissue is characterized by higher fever, by cerebral and circulatory disturbances of a typhoid nature, and by anemia, dropsy, and a highly-albuminous urine, even though blood may be absent and casts may be few. The dropsy is most apparent in the legs. Dyspnea, vomiting, diarrhea, and a progressive and rapid loss of flesh and strength ensue until convulsions or coma, sometimes preceded by acute maniacal excitement, end in death. Milder cases, lasting from two to four weeks, apparently get well, albumin and casts persisting, however, until, after an interval of weeks or months, another and similar attack occurs. In short, the first acute attack is liable to chronic repetition until a fatal one takes place.

Diagnosis.—The condition cannot be overlooked when the urine is carefully examined both chemically and microscopically. The dreaded *eclampsia gravidarum* can, however, be recognized only by repeated urinary examination, especially during the last months of pregnancy. Acute Bright's disease should be suspected, and the urine examined in every case showing pallor of the skin and puffy eyelids, whether general prostration of the health is apparent or not. The characteristic symptoms of acute exudative nephritis, as commonly seen when the condition is due to cold or occurs in scarlet fever, are the following: headache, restlessness, muscular twitching, nausea and vomiting, a tense pulse, moderate fever, dropsy, and anemia. Tube-casts and albuminuria are constant. It should be borne in mind that slight albuminuria occurring in the course of pregnancy or during any of the fevers, *without casts*, is not a true nephritis, although the latter may be a more or less remote consequence of the glandular degeneration of the renal epithelium associated with the febrile albuminuria. In addition to the presence of albumin and hyaline and cell-casts, however, a diminished quantity of sooty-looking urine and the discovery of red and white blood-corpuscles will render the diagnosis positive. The history of the case and the causal factors are also to be taken into consideration.

Prognosis.—The *duration* of ordinary exudative nephritis follow-

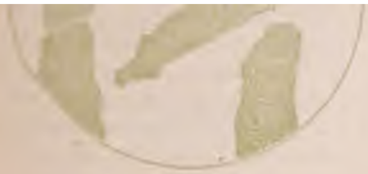

FIG. 59.

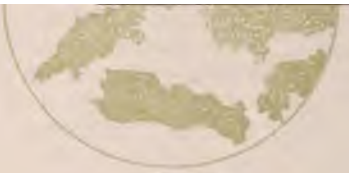

FIG. 60

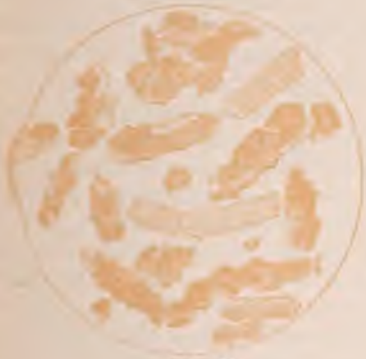

FIG. 61.

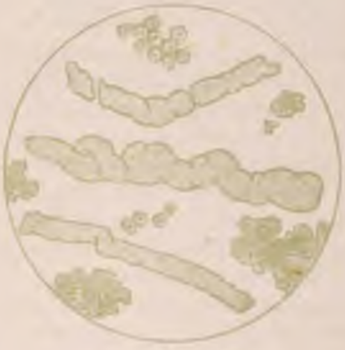

FIG. 62.

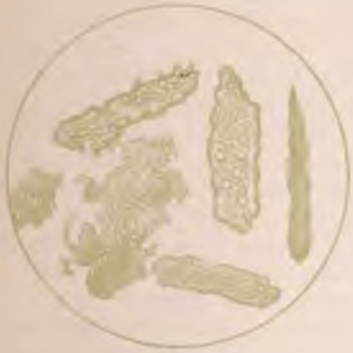

FIG. 63.

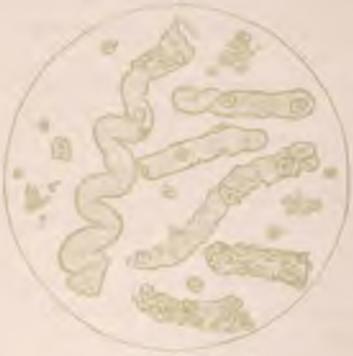

FIG. 64.

FIG. 59.—A. G., aged fifteen, male, suffering from acute nephritis. Urine showing granular casts (Queen obj. $\frac{1}{8}$; eye-piece ij.).

FIG. 60.—C. A., aged nine, male. Scarlatinal nephritis, third week of convalescence. Urine showing granular casts (Queen obj. $\frac{1}{8}$; eye-piece ij.).

FIG. 61.—J. D., aged fifty-four, male, suffering from cancer of the common duct and head of the pancreas. Urine showing bile-stained casts (Queen obj. $\frac{1}{8}$; eye-piece iv.).

FIG. 62.—B. J., aged twenty-two, female, suffering from puerperal eclampsia. Urine showing large, finely granular casts (Queen obj. $\frac{1}{8}$; eye-piece ij.).

FIG. 63.—S. A., aged fifty-eight, male. Urine showing granular and fatty casts; post-mortem showed chronic parenchymatous nephritis (Queen obj. $\frac{1}{8}$; eye-piece iv.).

FIG. 64.—C. C., aged forty-two, female, suffering from septicopyemia with amyloid kidney. Urine showing epithelial and (so-called) amyloid casts (Queen obj. $\frac{1}{8}$; eye-piece ij.).

[L. Napoleon Boston.]

ing exposure to cold and wet varies from a few days to three, four, or six weeks. The albuminuria steadily decreases, and with the casts finally disappears, while the daily quantity of lighter urine increases, as does the daily excretion of urea. The prognosis depends much upon the primary disease or causative condition, and also upon the intensity and character of the renal inflammation. Scarlatinal nephritis is less likely to be recovered from than nephritis due to exposure to cold after alcoholic excesses. The acute parenchymatous degeneration that accompanies typhoid fever, diphtheria, and other infectious fevers, as well as pregnancy, is usually a mild affection and recovery takes place easily. But in acute yellow atrophy, yellow fever, cholera, and in severe phosphorus- or mercurial poisoning death may occur from the intense and widespread necrosis of renal epithelium. In favorable cases of ordinary exudative nephritis the dropsy and albuminuria gradually diminish, while the color of the skin and the quantity of urine and urea increase, so that in the course of from three to four or six weeks recovery is established. After the disappearance of the dropsy the albumin may persist for some time, and then slowly disappear; but rarely, in unfavorable cases, even when dropsy has disappeared, albuminuria may continue and the affection become a chronic parenchymatous nephritis.

Serious and often dangerous symptoms of acute nephritis are—severe general edema, dropsical effusions into the serous sacs (as hydrothorax), uremia (especially when beginning with cerebral manifestations, as coma or convulsions), and finally inflammation of the internal organs, as pleuritis, pneumonitis, pericarditis, peritonitis, and meningitis. In the absence of uremia recovery in cases of marked general dropsy is quite common. Suppression of urine, however, lasting more than twenty-four or forty-eight hours, is usually a fatal symptom. The prognosis is unfavorable also in cases in which the nephritis has a productive character. Life may, on the other hand, be prolonged for several years.

Treatment.—I shall not include here the management of the primary affection of which the nephritis may be either a complication or consequence.

Since the renal function is diminished by the congestion and inflammation, the first object in the treatment is to relieve these conditions and thus restore the excretory function. The single or combined use of diaphoretics and cathartics is practised, therefore, not that the skin and bowels should be made to perform the work normally done by the kidneys, but in order to restore the functional equilibrium by the antiphlogistic effect produced.

Absolute rest in a warm bed and in a warm room is of primary importance. Woollen underwear and blankets should be provided, so as to promote a constant free action of the sweat-glands. These hygienic measures should be carried out both in the mild and in the severer cases.

Bland liquid foods only should be allowed in the diet, and the patient should be encouraged to drink freely of water (plain, distilled, or carbonated), lemonade, skimmed milk, or buttermilk; these are especially valuable when hot. Later, thin meat-broths may be allowed, although a strict milk diet is better.

Local bloodletting, as by leeches or cupping over the loins, I seldom employ; in rare cases, however, when much pain is complained of, it may be useful, although hot fomentations may be more so. Diminution of the edema and the elimination of urea and other urinary constituents that may be retained in acute nephritis are best obtained by exciting a profuse perspiration. The hot-air or hot-water bath and the hot wet-pack may be used to accomplish these results, and in most cases the last-named method suffices. It is easily applied by wringing a blanket out of hot water, wrapping the patient in it, and then with a dry blanket, and finally a rubber-cloth cover, surrounding all. This furnishes a steam-bath in which the patient may remain until copious sweating has lasted an hour or so, according to the condition, Children suffering from scarlatinal nephritis may be treated thus, or quite readily also by immersion in hot water, for twenty, thirty, or forty minutes; the skin should then be lightly dried, and the child wrapped in warm sheets or blankets and warmly covered in bed. Hot vapor or air may be generated alongside the bed, and transferred under the raised or cradled bed-clothes by means of a tin funnel and pipe. The sweating will be aided by the drinking of hot lemonade or soda-water or of water containing spirit of Mindererus. Should the skin fail to respond to these measures, as in uremia, perspiration may be started by a hypodermic injection of pilocarpin (gr. $\frac{1}{8}$ to $\frac{1}{6}$—0.008 to 0.0108), after which it will continue to pour out on the application of heat. The heart and pulse should be watched after the injection of pilocarpin, as serious collapse sometimes attends its use. The sweatings should be repeated until the dropsy disappears and as often as the patient's strength will permit. A useful adjunct to the above is the administration of hydragogues, as the saline cathartics, elaterium, and compound jalap powder. Elaterium extract (gr. $\frac{1}{6}$–$\frac{1}{4}$—0.0108–0.0162) is prompt in action, and magnesium or sodium sulphate (ʒj—4.0), given in hot concentrated solution every hour, or a calomel purge, may be recommended. It may be necessary to aid in relieving the tension and distress of extreme edema by multiple punctures or by the use of a small trocar and canula, with a drainage-tube (Southey) attached to the latter after the trocar is withdrawn. Aspiration must be performed if either hydro-thorax, hydro-pericardium, or ascites assumes serious proportions. Half-ounce (16.0) doses of the spirit of Mindererus (liq. ammon. acetat.) in water may be added to the diaphoretic treatment; this, combined with aconite, aids in controlling the fever that may be present and in preventing the vaso-constriction that is often premonitory of uremic symptoms.

Uremic convulsions that do not soon yield to prompt diaphoresis and catharsis should be treated by venesection. As much as a pint or two (.5–1 liter) of blood may be withdrawn and life saved thereby. Sometimes chloroform-inhalations are needed to subdue the very violent convulsive seizures, as in eclampsia. Their return may be prevented by rectal injections of potassium bromid and chloral, consisting of 1 dram (4.0) of the former and $\frac{1}{2}$ dram (2.0) of the latter. Contraction of the arteries with increased tension and beginning muscular twitchings calls for the use of nitroglycerin, chloral hydrate, or, possibly, morphin.

Diuretics other than the simple diluent drinks mentioned have very little use in the therapy of acute diffuse nephritis, at least early in the disease. Later, as adjuvants to the diuretic properties of water, potassium bitartrate or acetate, sodium benzoate, and cardiac stimulants, as caffein citrate and the infusion of digitalis, may be given, well diluted.

During convalescence care must be exercised that the patient does not catch cold. The diet must not be increased to solids too suddenly nor too rapidly, and particularly in the matter of meats. Light watery vegetables, fruits, and cereals may be gradually added to the diet-list, although milk should be mainly used. Ferruginous tonics are indicated for the anemia, and Basham's mixture is an admirable preparation at this stage.

A change of locality to a warmer, drier, and more equable climate, and careful habits of dress, diet, and exercise, are necessary in cases of recovery from the very serious forms of nephritis, in which the renal parenchyma is shown to have been somewhat damaged by the persistence of slight albuminuria at intervals.

ACUTE INTERSTITIAL NON-SUPPURATIVE NEPHRITIS.

("*Lymphomatous Nephritis*"—*Wagner.*)

Increasing interest and importance attach to the etiologic and pathologic features, principally, of this affection.

Definition.—"An acute inflammation of the kidneys, characterized by cellular and fluid exudation into the interstitial tissue, accompanied by but not dependent upon degeneration of the epithelium; the exudation is not purulent in character, and the lesions may be both diffuse and local" (Councilman).

Pathology.—The kidney is enlarged, pale, and somewhat mottled. The essential lesion consists in an acute proliferation of the cells in the intertubular tissue, with but little change in the parenchyma. The proliferation takes place mainly from the venous and capillary endothelium. The cells are found chiefly in the intermediate zone of the kidney, between the pyramids and the cortex. Howard[1] noted the occurrence of the following: (1) plasma-cells, lymphocytes, polymorphonuclear leukocytes in the exudation; (2) lymphocytes and plasma-cells in the dilated vessels; (3) mytosis and evident ameboid activity of plasma-cells in both blood-vessels and tissues. A special observation was the discovery of large numbers of typical eosinophilic leukocytes in the interstitial exudation and in the blood-vessels.

Etiology.—Most of the cases of acute interstitial nephritis occurred in children suffering from diphtheria and scarlet fever. The first case described, by Biermier in 1860, occurred in a case of scarlatina. Other acute infectious diseases, as typhoid fever, lobar pneumonia, measles, and epidemic cerebro-spinal meningitis, may have acute interstitial nephritis present. The Bacillus coli and pus streptococci have been found in some of the kidneys; and a general streptococcus infection following abortion has been noted in several cases of this form of nephritis; but Councilman concludes that bacteria play no part in the etiology of this affection, and that the powerful toxins of the mixed infection commonly met with are responsible for the chemotactic cellular proliferation.

No satisfactory *clinical histories* are obtainable. Many cases were moribund when first seen, and no special attention was given to urinary examination, since there were no prominent symptoms, such as edema, to indicate renal disease.

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[1] *Amer. Journ. Med. Sciences*, Feb., 1901.

CHRONIC NEPHRITIS (EXUDATIVE).

(*Chronic Bright's Disease; Chronic Parenchymatous Nephritis; Chronic Diffuse Nephritis with Exudation; Chronic Tubal and Chronic Desquamative Nephritis; Chronic Glomerulo-nephritis; Large White Kidney; Secondary or Fatty and Contracted Kidney.*)

Definition.—A chronic diffuse inflammation of the kidneys, attended with epithelial degeneration, exudation from the blood-vessels, and permanent connective-tissue changes in the stroma. According to Delafield, this is the *chronic productive* (*or diffuse*) *nephritis with exudation*—one of two varieties of chronic Bright's disease.

Pathology.—Although there are several types of pathologic kidney in this disease, the anatomic differences depend upon the causation and duration of the nephritis.

The first type of kidney to be mentioned is the *large white kidney* (without waxy degeneration). It is either enlarged or normal in size, and pale or yellowish in color. The surface is smooth, and the capsule is easily stripped off. On section the cortex is broader than normally, yellowish-white throughout, or it may present opaque yellowish or whitish areas with mottlings of red. The pyramids are congested in some cases. Microscopically, the following changes are commonly observed: the renal epithelium is swollen, hyaline, granular, or fatty, and more or less disintegrated or flattened; the glomeruli are enlarged from the growth of the capsule-cells and of the cells covering the capillaries, and in some cases, owing to the connective-tissue thickening of the capsule, the tuft of capillaries is found to be atrophied. The interstitial tissue shows some thickening of the arterial walls and a moderate growth of connective tissue in patches around the glomeruli and tubules; the latter contain hyaline and granular casts.

The *small white kidney*, or secondary contracted kidney, in most instances is probably a later stage of the preceding, in which the degeneration of epithelium is more advanced and the growth of connective tissue and resultant cicatricial contraction are prominent features. The kidneys are about normal in size (shrinkage of the large white kidney), the surface is slightly granulated, and the capsule is proportionately adherent. While this kidney is usually grayish or yellowish in color (*pale, granular kidney*), there may be some mottling due to red spots. The consistence is firmer than that of the large white kidney. The cut-surface shows yellowish-white foci of the fatty degenerated epithelium in the somewhat narrowed cortex, and hence the term that is sometimes used of "small, granular, fatty kidney." Under the microscope we find extensive degeneration and disintegration of the epithelium of the glomeruli and convoluted tubules, with atrophy of the parenchyma, and a corresponding increase of the interstitial connective tissue. Waxy degeneration may be associated.

Another variety is the *large red or variegated kidney of chronic hemorrhagic nephritis*. The organs are usually enlarged, swollen, red, and congested-looking or mottled, and frequently "bumpy" or slightly bossellated. The capsule is slightly adherent to the depressions between the bosses. Red spots, due to small hemorrhages, may be noticed on both the outer and cut-surfaces of the kidney. The section shows also congested portions and gray or yellow spots corresponding to the anemic and fatty degenerated portions. Small cortical hemorrhagic areas or striations, brownish-red in color, are distinctive of the kidney. The microscopic appearances are those of the large white kidney plus those of acute nephritis. Or, there may be inflammatory edema and cellular infiltration of the intertubular tissue, and dilated tufts of capillaries with surrounding cellular hyperplasia.

Etiology.—The disease may follow either the acute diffuse nephritis, as of scarlet fever or pregnancy, or simple chronic congestion and chronic degeneration of the kidneys. More often it arises insidiously, in a subacute manner and without any previous acute manifestation. Males are more frequently subject to this form of chronic Bright's disease than females. Children affected with the disease have usually had scarlatinal nephritis. Young adults are more commonly affected, however, with the usual variety, developing subacutely. Drinkers of beer and other malt and alcoholic intoxicants seem to be liable to the disease. It is not improbable that some toxic or infectious agency, acting slowly and persistently, may in the insidious cases be the cause of the nephritis, although manifestations elsewhere may be absent. I have observed it in certain individuals living in malarial regions. Persons working under exposure to cold and wet, and those living in humid and low, marshy localities, are more liable than those who are better protected from climatic vicissitudes. Tuberculosis, syphilis, and chronic suppuration may give rise to the "parenchymatous" form of chronic Bright's disease, and it is usually combined with amyloid disease (waxy degeneration).

Symptoms.—There may be a persistence, in a lesser degree, of the symptoms of an acute parenchymatous nephritis, particularly the anemia, dropsy, and the albuminuria, until the affection becomes chronic. In most cases, however, the disease develops slowly and gradually, in a *subacute* manner, though the earlier symptoms seldom indicate any renal derangement. There may be simply a general impairment of health and strength, loss of appetite, nausea, and attacks of indigestion, headache, dulness, and perhaps some pallor. Soon there is puffiness of the eyelids or swelling of the feet or ankles, or both, and the complexion takes on a blanched appearance. The *edema* gradually extends up the legs, and is often worse as the day grows, while on rising in the morning it may be found to have disappeared during the night's rest and recumbency. The *quantity of urine* is diminished in the majority of cases, though in the later stages it may be nearly or quite normal, and even slightly increased in long-standing instances of pale contracted kidney or when absorption of the dropsy is taking place. Superadded acute nephritis may cause a very scanty or a suppressed secretion of urine. The specific gravity is, of course, increased in scanty urine, and *vice versâ*. *Albuminuria* is often quite marked. The amount of albumin may be from one-fourth to three-fourths of the volume of the urine, or from 1 to 3 per cent. by weight, so that the daily loss of albumin may be considerable. The urea is much diminished. The color of the urine is turbid, sometimes smoky-yellow, and urates, casts, red and white blood-cells, epithelial cells, granular débris, and fatty granular cells are found in the usually abundant sediment. The *tube-casts* are of different varieties, but narrow or broad hyaline, fatty granular (Fig. 63). and epithelial casts are commonly observed.

The *edema* is prominent and persistent. It gradually extends all over the body, so that pitting can be obtained on the limbs, chest, abdomen, and back. The loose subcutaneous tissues, as of the penis, scrotum, and eyelids, are particularly distended. In chronic hemorrhagic nephritis, only, the edema may be absent or very slight. The *pasty, pallid complexion* and *anasarca* are most characteristic of chronic exudative nephritis, especially with large white kidney. The dropsy may be moderate and about stationary for several months; then, despite all treatment, it becomes insidiously worse, death ensuing in a month or two.

Dropsy of the serous sacs, with its attendant distressing symptoms, may be present in serious cases, and edema of the larynx and lungs may come on suddenly and cause death. Dyspnea may be toxic and nervous, as well as mechanical or cardiac in origin. *Cardiac dyspnea*, due to failure of the heart's action, as seen in many cases, is usually worse on lying down. It may be provoked by vaso-constriction, and is then a danger-signal of uremia. Catarrhal bronchitis may be associated with cough and expectoration.

The heart is often affected with *moderate hypertrophy of the left ventricle*, and later by dilatation and weakness of both ventricles. The *aortic second sound is accentuated* and the *pulse-tension increased*.

Uremic symptoms are frequently manifested, except the convulsions which are common to chronic nephritis without exudation. Headache, vertigo, sleeplessness, nausea and vomiting, diarrhea, and stupor, coma, or delirium, may develop and precede a fatal termination.

Albuminuric neuro-retinitis, as evidenced by dimness of vision and field-defects, occurs in quite a number of cases. The skin of the legs becomes subject to a red eczematous eruption in some cases of great dropsical distention. In the absence of complicating inflammations, such as pericarditis, endocarditis, pneumonitis, and ulcerative colitis, which are rare, the temperature is practically normal.

The **course** of chronic exudative nephritis may either continue from bad to worse, until death ends all in a year or two, or anemia, dropsy, and albuminuria may attack one who for years previous has had apparent good health, after a first attack the second proving fatal within a few months. Again, some patients, having a little pallor, slightly diminished urine of high specific gravity, with albumin, may complain of nothing for years, until decided attacks, lasting for several months, may occur, with intervals during which the dropsy, dyspnea, etc. may be absent, although some albuminuria persists.

The average *duration* of the disease varies from one and a half to three years. The duration of chronic hemorrhagic nephritis may be somewhat longer (eight months to two years) than that of the large white kidney (six to eighteen months), but it is shorter than the secondary, contracted kidney, which lasts from one and a half to three or even five years.

Diagnosis.—The diagnosis of the disease itself is not difficult, but of the stage or the variety of kidney it is almost impossible to tell correctly in some instances. The urinary examination, coupled with the symptoms of dropsy and anemia, is sufficiently diagnostic of chronic diffuse nephritis (with exudation). The fact that, as shown by Czyhlarz and Donath,[1] methylene-blue is retarded in its elimination from the kidneys in nephritis, may have diagnostic value in some cases.

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[1] *Wien. klin. Wochen.*, June 15, 1899.

In cases of *large white kidney* the urine passed is less in quantity and of higher specific gravity than in the small, pale, and contracted kidney. Edema is usually greater in the former also, while in the latter cardio-vascular changes are more marked. The transition of the disease from the earlier to the later stage may be thus noted. The casts in the latter may also be narrower and more darkly granular than in the large white kidney. The existence of *hemorrhagic kidney* may be inferred from the history of alcoholism and the persistent presence of red blood-corpuscles and blood-casts in the urine.

Chronic parenchymatous is distinguished from chronic interstitial nephritis by the following points of difference:

CHRONIC PARENCHYMATOUS NEPHRITIS.	CHRONIC INTERSTITIAL NEPHRITIS.
Occurs in early or middle life.	Occurs later in life.
There is a previous history of an acute attack of scarlet fever, or perhaps of acute alcoholism.	A previous history of gout, chronic lead-poisoning, syphilis, excessive eating and drinking (spirits), nerve-strain.
The onset is gradual or markedly manifest.	The onset is very slow, insidious, and indefinite.
Dropsy is a constant symptom.	Dropsy is rare.
Vascular changes and cerebral symptoms are comparatively uncommon.	Arterio-sclerosis, cardiac hypertrophy, and cerebral symptoms are common.
Marked albuminuria, with tube-casts.	Very slight albuminuria and few casts.
Urine but little increased in quantity, often diminished; specific gravity is increased or slightly diminished.	Urine of very low specific gravity, and excessive in quantity.
Anemia occurs earlier and is more distinct.	Anemia slowly progressive and less marked.
Uremic symptoms are generally less severe—amaurosis, vomiting, diarrhea, headache.	Uremic symptoms are, generally severe —coma and convulsions, great dyspnea.
Runs a shorter course—from two to six or seven years.	Has a more chronic course—seven to thirty years.

Prognosis.—This is invariably bad as to cure, though life may be prolonged in certain cases. In severe cases death may take place in from three months to a year, either from uremia, dropsy, cardiac dilatation, or complications. Cases of a year's duration almost never recover, and, *a fortiori*, those in which advanced secondary contraction of the kidney may be inferred are incurable, and may soon terminate fatally. Complete recoveries from the disease, particularly in children that have had scarlet fever, may occur but rarely. The prognosis depends greatly on the quantity of urine passed in the twenty-four hours, the excretion of urea and total solids, and upon the amount and persistency of the albumin, as well as upon the degree of cardio-vascular and retinal changes.

Treatment.—The indications for treatment are similar to those in acute nephritis. The dropsy and uremia must be treated symptomatically, and the *diet* is of importance. Skimmed milk and buttermilk, or "zoolak," with dried bread, crackers, and zwieback, perhaps, should be depended on as much as possible when dropsy is pronounced. When dropsy is slight, more solid food, meats sparingly and vegetables, rice and other light cereals and fruits, and out-of-door life should be recommended.

Residence in a warm, dry climate may aid in prolonging life. Woollens should be worn next to the skin, and prolonged, sudden, and severe exercise should be forbidden.

The infusion of digitalis, strophanthus, spartein, adonidin, or convallaria, may be needed in cardiac weakness, or nitroglycerin for contracted and tense arteries with a tendency to uremic twitchings. Unirritating diuretics, such as Trousseau's diuretic wine and Basham's mixture for the anemia, are useful. Strontium lactate in doses of from 15 to 20 grains (0.972–1.29), three times daily, I have found useful in some cases. Diuretin has also been tried lately with favorable results.

CHRONIC NEPHRITIS (NON-EXUDATIVE).

(*Chronic Interstitial Nephritis; Chronic Bright's Disease; Primary, or Genuine, Contracted Kidney; Cirrhotic Kidney; Red Granular Kidney; Chronic Productive* (*Diffuse*) *Nephritis without Exudation* (*Delafield*); *Gouty Kidney.*)

Definition.—A chronic diffuse inflammation of the kidneys, attended with a growth of connective tissue in the stroma, degeneration and atrophy of the parenchyma, and marked cardio-vascular changes.

Pathology.—In genuine primary contraction of the kidneys there is a reduction in size and weight about equal in both organs. They may be only one-half or one-third the size of normal kidneys, and the two kidneys together may not weigh over two ounces. They are often found imbedded in thick adipose tissue, the capsule being thick, opaque, and very adherent, so that on stripping it off it brings away portions of the renal cortex. The outer surface of the kidney is red, irregularly granular, or finely nodular, and occasional small cysts are sometimes present. The consistence is firm, dense, and resistant to the knife. Examination of the cut-surface shows a thin atrophied cortex, and dark, reddish streaks alternating with pale portions. The pyramids are also diminished, and darker than the cortex. In the gouty contracted kidney the pyramids show fine striations of sodium urate or of uric acid, or crystals representing uric-acid infarctions.

Microscopically, the essential changes are an increased production of connective tissue, especially in the cortical substance, and a more or less proportionate degeneration and atrophy of the renal parenchyma, the destruction of which is due to the circulation of noxious agents, but which is replaced by cicatricial fibrous tissue (Weigert).

The new tissue is not uniformly distributed in the cortex, but occurs in irregular masses around the shrunken glomeruli or between the tubules. The distribution of connective tissue in the pyramids is diffuse. Many of the glomeruli are quite small and fibrous in advanced cases, while in the earlier cases the cells of the tufts and capsules are swollen and multiplied, and a small-cell infiltration is seen around the glomeruli and tubules. Later this infiltration of cells becomes fibrillated and ends in thickening. Glomerular atrophy is due partly to the changes in the capillary and intra-capillary cells, as well as in those around the tufts; partly also to capsular thickening and hyaline or waxy degeneration; and partly to the thickening and occlusion of arterioles.

The tubules show marked changes. Some are included in masses of connective tissue, so that there is compression-atrophy and even total obliteration of the lumen. In other instances the intertubular connective tissue constricts the tubules in certain places, so that the lumen is elsewhere increased. This dilatation is especially prominent in the granules seen on the outer surface of the kidney, and, owing to the damming back of urine in some of the tubules thus obstructed, little cysts are visible to the naked eye here and there. The epithelium lining these tubules shows granular, fatty, or waxy degeneration, and may be either flattened, cuboid, or swollen. The tubes may contain granular or fatty débris and tube-casts.

An important change in most cases is the growth of fibrous tissue in the walls of the arteries, causing sclerosis. This affects the intima (endarteritis), the media, and adventitia, all of which are thickened by the hyperplasia of connective-tissue elements, and by newly formed elastica. The arteries and capillaries are thus mostly occluded by the obliterating endarteritis or by their conversion into masses of connective tissue. Waxy or hyaline degeneration is seen also (*vide* Arterio-sclerosis). These arterio-capillary changes may in some cases be the primary condition, and may represent the renal effects of a general arterio-sclerosis or fibrosis.

An almost constant accompaniment of chronic, non-exudative, productive nephritis is cardiac hypertrophy. The degree of the latter depends upon the extent of the renal, and also of the general arterial, degeneration and sclerosis. The whole heart may become so large that the term *cor bovinum* has been fittingly applied to it. In moderate enlargements the left ventricle only is hypertrophied.

Complicating lesions that may be mentioned are cerebral hemorrhage, cirrhosis of the liver, pulmonary emphysema, chronic endocarditis, chronic endarteritis, pericarditis, bronchitis, and gastric catarrh.

Etiology.—The cause of the very slow primary, diffuse degeneration, atrophy, and fibroid contraction of the kidneys is sometimes quite obscure. (*a*) In some cases it would seem to be "only an anticipation of the gradual changes which take place in the organ in extreme old age" (Osler)—the "senile kidney." (*b*) *Heredity* undoubtedly plays a part in the causation of certain cases, even to the third or fourth generation. (*c*) *Age and Sex.*—The disease is more common in males than in females, and it usually begins near middle life; it is seldom manifested symptomatically until about fifty or sixty years of age, and is therefore an affection of advanced life. (*d*) Individuals having a special tendency to sclerotic degeneration of the arteries, from whatever injurious influence, are more liable to chronic interstitial nephritis, although the prolonged irritation of deleterious (especially chemico-toxic) agents may give rise to the disease in those whose cellular nutrition is usually not defective. Thus, the disease has been attributed to the following causes: alcoholism, uric acid, and lead, giving rise to chronic poisoning. Chronic syphilis and chronic malaria probably are also causative factors. (*e*) Habitual overeating and drinking, owing to the imperfect metabolism of the substances ingested, cause a constant excretion of irritating products by the kidney, and no doubt frequently cause granular atrophy and sclerosis of the organ. The continuous and even moderate use of alcohol for many years, especially of spirituous liquors, is a widespread cause of the disease. It is equally likely that the excessive use of red meats in the diet leads to the production of the uric acid that induces the renal disorder (*uricemia; lithemia*), by deranging the function of the liver (Murchison). (*f*) Allied to the above is gout, which causes chronic Bright's disease—in England perhaps more than in this country, lithemia and nervous dyspepsia being more common there. (*g*) According to Strümpell, severe acute articular rheumatism is sometimes followed by contracted kidney. (*h*) Chronic Bright's disease with renal sclerosis is favored in origin and development by the anxieties, worries, and high nervous tension connected with modern business activity and "social functions," the latter particularly acting their part among elderly ladies. Associated with these are usually over-indulgence in rich foods and wines, and sedentary habits. (*i*) The cold, moist climate of New England and the Middle States would seem, according to Purdy, to predispose to contracted kidney. A chronic productive nephritis without exudation, though not the true "contracted and red granular" kidney, may be caused by hydronephrosis, chronic pyelitis, and chronic congestion of the kidney, as from heart-disease.

Symptoms.—These may be latent for years, while the morbid productive changes in the kidneys are slowly effected. The first symptoms may not appear until late in life, although the kidneys may be in an advanced stage of degeneration. Or some complicating or intercurrent affection may set in, as pneumonia or pericarditis, and cause the development of grave or fatal renal symptoms. More commonly, however, there is an attack of *uremia*, with headache, stupor, or convulsions, dyspnea, nausea and vomiting, and a tense pulse. This attack may be recovered from. Then there is an interval of variable duration, during which the health is more or less impaired, and lassitude, drowsiness, disordered digestion, headache, failing vision, dyspnea, and frequent micturition are complained of. This is followed by another uremic attack, severer than the first, or perhaps fatal; if not fatal, the general health is still more reduced, and confinement to the house or bed is necessary, until the vital forces can no longer compensate for the destruction of the renal parenchyma.

Spasmodic dyspnea (uremic; cardiac) is sometimes the first manifestation of contracted kidney. The gradual onset of periods of uncontrollable drowsiness during the day is often marked. An attack of hemiplegia may also be the first indication of renal disease. Sometimes progressive loss of flesh and strength, with a *dry, harsh, wrinkled skin,* may be from the beginning the only clinical features of the affection until death results from sheer feebleness and emaciation. The complexity and variability of the symptoms make it best to describe them under the heads of the various systems:

Urinary System.—The daily *quantity of urine* is usually increased so much that patients are troubled with a desire to urinate frequently, not only during the day, but two or three times during the night. This complaint may be aggravated by the hyperacidity of the urine and the irritability of the prostate (especially in advanced age) that are so often associated with cases of renal cirrhosis. The urine voided during the twenty-four hours may measure several quarts (2 to 4 liters) in well-marked cases of the disease. Early in the attack, when the incipient degeneration and destruction of the parenchymatous cells is taking place, the quantity of urine may be slightly decreased; but as the "blood-flow to the parts that remain must, *cæteris paribus*, be as great as it would have been to the whole of the organs if they had been intact," excessive pressure is brought to bear within the capillaries by the compensating hypertrophy of the heart, and the secretion of the urine, especially of the watery elements, becomes more active. The *polyuria* may give rise to a suspicion of diabetes. The urine is clear and pale-yellow in color, the *specific gravity* being seldom above 1010 or 1012, and it may be as low as 1002 or 1005. *Albumin* is found only in traces or it may be absent altogether (*glomerular atrophy*), especially in urine voided in the early morning. The urea is diminished, as in all forms of Bright's disease, and there is little or no sediment. A very careful microscopic examination may reveal a *few*, usually *narrow*, *hyaline* or *granular casts*, perhaps some *leukocytes*, and rarely a few *erythrocytes*. In the later stages of the disease or upon the supervention of an uremic exacerbation or of a complicating inflammation the urine may be decreased, the albumin increased, and numerous casts be discovered in a more apparent urinary sediment. Hematuria is rare.

Circulatory System.—The *physical signs* of cardiac hypertrophy are present. Symptoms referable to the heart are absent, unless dilatation and feebleness, sudden arterial contraction, cardiac complications, or endocarditis occur. *Inspection* and *palpation* of the *hypertrophied heart* show an apex-beat displaced downward and to the left, and an increased, heaving, and rather circumscribed apical impulse. These signs may be less evident both in cases of coexisting emphysema and later when dilatation may eclipse the hypertrophy. The left border of the deep cardiac dulness extends outside the nipple-line in the fifth or sixth interspace. The first sound of the heart is loud and may be duplicated. A distinctive auscultatory sign is the *accentuation of the aortic second sound*, indicating increased vascular tension; it may have a metallic quality. In quite a majority of the cases I observe, sooner or later, a mitral systolic murmur; it is due to relative insufficiency.

The *pulse* is *increased in tension*, and is hard, incompressible, and persistent, the duration of each pulse-wave being increased (*pulsus tardus*). The radial artery itself—and this is true of most of the palpable arteries—feels hard, thickened, and often tortuous, on account of the arterio-sclerosis. As soon as compensation of the heart fails, symptoms of breathlessness (especially on exertion), palpitation, and the like, appear, and sometimes in paroxysmal attacks ("cardiac asthma"). The resultant stasis gives rise to transudation into the lungs (bronchorrhea; pulmonary edema), and later to edema of the extremities.

Respiratory System.—Epistaxis may be a serious symptom. Sudden edema of the larynx may also occur, and is always grave. Transudations into the pleural sac (hydrothorax), as well as into the lungs (*vide supra*), may precede death. Dyspnea, which is either cardiac or uremic, is usually worse at night, and a true orthopnea, together with Cheyne-Stokes breathing, may be observed toward the end of the patient's life and in association with uremic stupor and coma.

Nervous System.—Symptoms referable to the nervous system are very important, since they are usually indicative of grave uremia. Cephalalgia is frequent, and neuralgic pains throughout the body, and insomnia, may be complained of. Later great *drowsiness* is often a premonition of uremic coma. Convulsions may be preceded by muscular twitchings, which should attract attention to the imminent danger of the former. Cerebral apoplexy with hemiplegia may be the first symptom of contracted kidney. It is especially apt to occur in cases of marked hardening and weakening of the arteries. There may be an hemorrhagic pachymeningitis, as well as a hemorrhage into the brain-substance. The hemiplegia may persist until death; or it may disappear in a short time, and be followed by subsequent attacks at intervals. *Formication*, *numbness*, and pallor of one or more fingers (the so-called "dead finger") I believe with Dieulafoy to be sometimes the earliest symptoms of chronic Bright's disease.

Of the **special senses**, *nephritic retinitis* is often the earliest evidence of chronic Bright's disease. The patient may or may not have had slight dimness of vision (mistiness) prior to the ophthalmoscopic examination. The loss of vision affects both eyes, and is usually partial (*amblyopia*). Sudden and complete blindness may come on in grave cases —*uremic amaurosis*—the condition being due to neuro-retinitis. The optic papilla is swollen, and surrounded by retinal hemorrhages or by white dots and streaks ("feather-splashes"). Tinnitus aurium, deafness, and *vertigo* are not uncommon.

Digestive System.—Anorexia, nausea, and annoying dyspepsia are often complained of. Severe vomiting may usher in an uremic attack. Catarrhal gastritis may exist for some time, the tongue being coated and the breath heavy and urinous. *Uremic diarrhea* may also occur.

The Skin.—Edema is usually absent in renal sclerosis; when it does occur, however (as in the ankles and limbs), it is due to dilatation and failure of the heart. The skin is dry, and minute lustrous scales of urea may be seen around some of the pores. A certain degree of pallor is noticed, and often the skin has a cyanotic tinge. *Pruritus* and troublesome eczema are frequently present, and *muscular cramps*, occurring especially in the calves of the legs and at night, may also be associated. The general nutrition gradually fails, so that in advanced cases the debility and emaciation are extreme.

It is important to bear in mind the fact that *uremia* may come on at any time during the course of the disease, and that it may be the first symptomatic manifestation; also that it may either be sudden and severe in its onset (acute uremia) or mild, insidious, and gradual (chronic uremia). Moderate fever may attend an uremic attack, or the temperature may be normal; in chronic uremia, with prostration, coma, delirium, and feeble pulse, it may be even subnormal.

Among the **complications** that may occur in the red, granular, and contracted kidney are the following: pneumonia, either lobar or lobular; pleuritis, pericarditis, laryngitis, bronchitis, gastritis, enteritis, peritonitis, meningitis, endocarditis, emphysema, phthisis, acute dermatitis exfoliativa (Duckworth), and hepatic cirrhosis.

Diagnosis.—This depends in great part upon the physical, chemical, and histologic examination of the urine. Both the morning and evening urine should be examined repeatedly for albumin and casts, since one examination—and especially that of the morning urine—may give negative results, owing both to the scarcity of these two pathologic elements and to the fact that albumin may be altogether absent in some instances. The mere discovery of a trace of albumin or of a few casts is not always positive evidence of chronic Bright's disease, as both may exist in other conditions. But the age, habits, and symptoms of the patient must be studied in connection with frequent urinary examinations; and a persistent slight albuminuria, with casts, and the passage daily of large quantities of clear, pale urine of low specific gravity, afford sufficient grounds for making the diagnosis.

Contracted kidney should be suspected in all cases in which, during middle life, either one or more of the following symptoms and signs may be noticed: frequent headache, congestive disorders, repeated epistaxis, vertigo, dimness of vision, intractable conjunctival irritation (Alleman), impaired strength, dyspneic attacks, gastro-intestinal dyspepsia, noises in the ear, itching of the skin, cramps in the calves, muscular twitchings, growing mental dulness, increasing pulse-tension, and rigidity and tortuosity of the temporal and radial arteries. Sudden coma, convulsions, amaurosis, apoplexy, vomiting, or dyspnea in persons in the middle period of life, with or without a history of polyuria, should create the suspicion of chronic Bright's disease. It will be found in such cases that there has been a diminution in the urinary flow before the attack. Persons of lithemic, gouty, rheumatic, or alcoholic habits, or in whom lead-toxemia is discoverable, with evidences of cardiac hypertrophy, an accentuated aortic second sound, and a hard pulse, are often readily diagnosed as subjects of contracted kidney when a further examination of the urine is made.

If the first examination of the patient is made during a sudden uremic or apoplectic attack, catheterization should be done if necessary, and the detection of albuminuria will then clear the diagnosis.

In order to differentiate between primary renal affection with secondary cardiac hypertrophy and *primary heart-disease with a secondary congested kidney occurring late in the case*, the general features, course, symptoms, and signs must be carefully and judiciously balanced. Prominent cardio-vascular changes would indicate an arterio-sclerotic kidney, rather than the primary granular and contracted kidney of toxic origin. The symptoms of ordinary non-inflammatory *senile kidney* may not be unlike those of chronic interstitial nephritis, though not so severe; and yet, from excessive eating and drinking at times, uremic attacks may supervene to cloud the diagnosis.

Prognosis.—The *duration* of chronic interstitial nephritis varies. In uncomplicated cases it may last for five, ten, twenty, or possibly thirty years. Complications or intercurrent affections may, however, shorten the duration very much, or the existence of the condition may be unknown, as frequently happens, when the *postmortem* examination shows the characteristic kidneys in one who during life had no symptoms indicating renal disease, and whose death was caused by some intercurrent disease. The gradual destruction of the renal parenchyma and its replacement by scar-tissue cause irreparable damage to the organs. On the other hand, the fact that the process is usually a slow one and its duration long is compatible with the preservation of life for many years, and with comparative comfort, even, in many instances. The prognosis in a given case depends very much upon the general constitutional condition, the cardio-vascular state, and the presence or absence of uremia and inflammatory complications. Cardiac dilatation and insufficiency indicate a not far distant end. Convulsive and apoplectic seizures are often fatal, and hemorrhages, persistent vomiting, and diarrhea, *retinitis nephritica*, coma, and delirium render the prognosis as to further systemic tolerance of the degenerated kidneys exceedingly grave.

Treatment.—An early recognition of the disease and the steadfast practice of careful hygienic measures will prevent, to a considerable degree, the advance of the cirrhotic changes. Noxious substances entering into the etiology of the affection must be avoided and removed as far as possible. The formation of uric acid must be reduced by dietetic management, alcoholics must be interdicted, and lead—when the cause of the condition—must be kept from further poisoning the system by a change of occupation. By diminishing these irritants the heart and blood-vessels are also conserved—a point of vital importance.

The **hygienic treatment** must embrace a regulation of all the habits of body and modes of life. The patient must be treated, and not his malady, since that is incurable. A dietary that is suitable for each individual case must be made out, and on general principles. Saundby's rule is a good guide: "Eat very sparingly of butcher's meat; avoid malt liquors, spirits, and strong wines." An exclusive milk diet may be necessary for short periods when gastric irritation is present, but in such a chronic disease undue weakness would result from a restriction to milk alone. I would therefore recommend a light nourishing diet, including lean meat once daily in favorable cases. Vegetables, greens, fruits, and light, well-cooked farinaceous articles may also be partaken of, and tea, coffee, and cocoa may be drunk. The use of natural mineral waters aids in the renal circulation and keeps the kidneys flushed. In general a mixed diet will be of advantage; the nitrogenous and carbohydrate elements (sugars and starches) are used in limited amounts, while pure fats and fruits (raw or cooked) are to be recommended. Stout persons and those leading sedentary lives should have less food than those taking exercise, and gastric disorder requires the elimination of all but soft, bland foods, or a liquid diet until digestion is restored. Extremes of bodily, mental, and emotional activity should be avoided, and physical exercise should be moderate, regular, and taken in the open air, provided the latter be warm and dry. Mental labor should never be excessive, nor should the patient be subjected to the vicissitudes of worry, anxiety, or competitive tension. Venereal excitement and indulgence of any kind tending to disturb the equanimity, cheerfulness, and contentment, should be strictly forbidden.

A change of residence to a warm, mild, and dry climate is often of service in prolonging life. The variability and humidity of temperate climates, particularly during the winter months, aggravate this disease, while a sea-voyage or a sojourn at some southern, western, or European resort, where the soil is dry and sandy and the climate equable, may be very beneficial to one who can afford it.

The indications for **medicinal treatment** are principally as follows: The bowels should be kept free by the aid of laxatives or laxative alkaline mineral waters. Papoid, peptenzyme, and other digestants, with bitter tonics, are useful in some cases in which a furred tongue and indigestion are troublesome. Acids or alkalies, according to special indications, may also be used simultaneously. An increased vascular tension (vaso-constriction), such as to place a serious strain upon the heart; the other extreme, of a very low tension that induces dropsy; and complications, usually uremic (convulsions, dyspnea, headache), also call for therapeutic assistance. High tension is to be met by the cautious use of nitroglycerin in gradually ascending doses, beginning with 1 minim (0.066) three or four times daily, until all danger of rupture of the vessels seems to be past. Headache, vertigo, and the so-called renal asthma (dyspnea) are also often relieved by this drug.

Low tension, with signs of cardiac dilatation, scanty albuminous urine, and edema, requires heart-tonics and stimulants, in conjunction with purgatives. Digitalis (preferably in infusion) has good effects, especially when combined with strychnin nitrate or with caffein citrate. Calomel and the salines should be given for the dropsy.

Uremic symptoms should be treated as in acute Bright's disease by causing profuse sweating and free catharsis, and in some cases by phlebotomy. Inhalation of amyl nitrite or chloroform, or, what is often a useful and necessary measure, the hypodermic injection of morphin (gr. $\frac{1}{6}$–0.0108), may be tried in convulsions, severe headache, or dyspnea.

Contracted kidney of a probable malarial or syphilitic origin may be benefited somewhat by the use of arsenic and the iodids respectively; but no drugs can possibly restore the destroyed renal parenchyma or transform connective-tissue cells into secreting kidney-cells.

PYELITIS.

(*Pyelonephritis; Pyonephrosis.*)

Definition.—Inflammation of the pelvis of the kidney. The compound terms above represent inflammation of the kidney-structure as a result of, and combined with, pyelitis.

Pathology.—In the mildest varieties of pyelitis (the catarrhal) the morbid changes consist simply of a reddened, swollen, and turbid mucous membrane, covered with an exudation of viscid muco-pus and desquamated epithelium. Ecchymoses are sometimes seen. The urine in the pelvis of the kidney is also turbid from the admixed pus-corpuscles and pelvic epithelium. In calculous pyelitis, owing to prolonged and severe irritation, purulent inflammation and ulceration prevail, and the kidney-structure is also involved by extension (pyelonephritis). Renal abscesses are thus formed, and small dark calculi are frequently found mingled with the pus in quite a number of small abscess-cavities; or perhaps, as noted before (*vide* Nephrolithiasis), one large abscess-cavity may replace the destroyed renal parenchyma (*pyonephrosis*).

A diphtheritic inflammation, with the formation of a false membrane and sloughing of the pelvis, sometimes follows the severe infections of the specific fevers. Marked hemorrhagic areas may be seen also. In tuberculous pyelitis there is usually an association of nephritis with areas of tuberculous softening and ulceration, and later pyonephrosis. In very chronic and sluggish cases the pyelitis may be followed by an infiltration of the kidney-structure with cheesy or putty-like masses that may become the seat of calcification.

Persistent obstruction leading to pyelitis is associated with dilatation of the pelvis from retention of urine or of pus (pyonephrosis). This in turn, from prolonged pressure, causes the marked atrophy of the secreting structure of the kidney that is seen in such cases. There is also an increase in the interstitial tissue and secondary contraction.

The so-called *surgical kidney* is found when an acute bilateral pyelitis, following a severe cystitis, has excited an acute suppurative inflammation of the kidney. Acute suppurative or interstitial inflammation of the kidney due to metastatic or miliary abscesses is considered under the heading *Pyemia* (*vide* p. 201).

Etiology.—Pyelitis rarely is primary or independent in origin, as after exposure to cold and wet. The secondary causes of pyelitis are as follows: (1) renal calculi (the most frequent); (2) extension upward of urethritis, cystitis, or ureteritis, particularly when gonorrheal in origin; (3) retention of decomposed urine in the pelvis of the kidney; (4) renal affections, as tubercle, carcinoma, and acute nephritis; (5) specific fevers; (6) foreign bodies, other than stone in the pelvis; (7) irritating diuretics. To point out briefly certain additional facts bearing upon the causation of pyelitis in the order named, it should be mentioned that *calculous pyelitis* may result from the irritation of the constant presence and passage of small stones ("gravel"), or even of uric-acid "sand," as well as from the large dendritic concretions that send offshoots into the calyces. Extensions of inflammation to the pelvis from lower portions of the urinary tract may occur in protracted cases of such affections as gonorrheal urethritis and puerperal and calculous cystitis. *Obstructive pyelitis* sometimes follows the impaction of renal calculi or of other foreign bodies in the ureter when there is pre-existing inflammation of the tract, or when, as usually happens, there is chemical irritation from the decomposition of the accumulated urine. There may be obstruction in the bladder and urethra, as from enlarged prostatic tumors, stricture, phimosis, and paralysis of the sphincter vesicæ, or as in paraplegia. Under the consideration of tuberculosis and carcinoma of the kidney is included the involvement of the pelvis by these conditions. *Infectious pyelitis* may also result from small-pox, diphtheria, typhus and typhoid fevers, and scarlatina, and it depends upon the irritating effect of certain substances eliminated by the kidneys. It is usually associated with more or less nephritis (pyelonephritis). Parasites, such as the echinococcus (hydatids), distoma, strongylus, and filaria, may give rise to pyelitis. Cantharides, cubebs, copaiba, turpentine, and diabetic urine even, may in rare instances also excite a pyelitis.

Symptoms.—These are frequently overshadowed by those of the primary condition that causes the pyelitis: they are varied also for the same reason. The clinical manifestations of a simple catarrhal pyelitis are slight pain and tenderness in the region of the affected kidney or kidneys, mild fever, with a *turbid urine of acid reaction*, showing a few pus-cells, a little mucus, rarely some red blood-corpuscles, and a trace of albumin.

In the severer varieties, as in calculous pyelitis, especially when there are attacks of renal colic, the urine frequently shows to the naked eye the presence of *blood* and a marked amount of *pus*, some *mucus*, and the transitional *caudate epithelial cells* from the middle layers of the mucosa. The presence of the latter, however, is not constant, hence its absence does not exclude the existence of a pyelitis, since some of the most destructive forms of the affection, as the acute or chronic suppurative or the pyelonephritic, may be unaccompanied by the presence of the pelvic epithelium in the urine. This holds still more true in the case of true pyonephrosis, in which the kidney usually becomes one large abscess.

In severe pyelitis the *pain* is often acute, coursing *down the ureters*. The fever is moderate, and there are present the common symptoms described under Nephrolithiasis (*vide* p. 974).

The *fever* in purulent pyelitis (pyonephrosis) and pyelonephritis takes on a *hectic* or *typhoid* type. Paroxysms of rigors or chills, followed by a rapid rise in temperature and ending in perspiration, may be observed; or there may be marked prostration and feebleness of circulation, delirium, and stupor. The temperature-curve runs an irregular course, with marked remissions, in cases having a pyemic nature.

In obstructive pyelitis the urine sometimes flows freely and normally for a while, until the developing pain over the inflamed kidney ends in relief by the expulsion of the obstacle and the passage of purulent urine. This *alternation* of normal with pyoid urine is indicative of a unilateral pyelitis.

Ammoniacal urine is met with in *cysto-pyelitis*. *Albuminuria* is decidedly shown according to the degree of pyuria.

In chronic suppurative pyelitis or pyelonephritis the pyuria is variable both in quantity and constancy. *Intermittent pyuria* may be due to the temporary blocking of the ureter by a stone (*vide* Obstructive Pyelitis). The pus is seldom mixed with epithelium in chronic purulent pyelitis. The associated intermittent fever may be like that of tuberculous pyelitis, and marked prostration, anemia, and emaciation are concomitants. Evidences of amyloid change may be revealed in long-standing, chronic cases.

The term *ammoniemia* has been applied to that complexus of nervous symptoms that is supposed to arise from the decomposition and absorption of urinary substances. These symptoms may be similar to the manifestations of diabetic coma.

Distinct *enlargement* and *fluctuation* of the diseased kidney may be determined in some cases of pyonephrosis. This may also be intermittent, being detectable while there is obstruction to the flow of pus, and *vice versâ*. According to A. H. Smith, at the menstrual periods pyelitis may be subject to marked exacerbations, simulating renal colic.

In chronic pyelitis with atrophy of the kidney the onset of uremia may terminate the case. Granular kidney alone may have been simulated by the passage of an increased quantity of urine of proportionately low specific gravity.

Diagnosis.—This embraces the discrimination from other affections, and the possible detection of the variety—etiologically considered—of the pyelitis. It is most important to pay attention to the clinical history of any case with a view to the discovery of the cause; also the urinary condition must be carefully studied. In the very nature of this affection it is often impossible to exclude other affections of the urinary tract, as *nephritis*, *cystitis*, and *urethritis*.

Epithelium from the pelvis of the kidney cannot be distinguished from transitional bladder-cells; but, given the indications of a pyelitis, its calculous cause is at once made clear upon the passage of the characteristic uratic or oxalatic concretions. It may happen that the urine from one kidney is prevented from flowing by the impaction of a stone in the ureter. The urine may now flow clear from the other and vicariously acting kidney until, the stone having given way, it suddenly increases in quantity and changes in character, owing to the return of the morphologic elements of the pyelitis (corpuscles, desquamated epithelium, crystals, and débris).

In women catheterization of the ureters and renal pelves, as described and practised by Pawlik and Kelly, is a most certain method of determining in doubtful cases from which side the purulent urine arises. Palpation of the ureters through the lateral and anterior fornix of the vagina will sometimes reveal thickening and tenderness in cysto-pyelitis, and ureteral distention sometimes may be felt in pyelitis calculosa.

Vierordt mentions having seen in some cases of pyelo-nephritis peculiar hyaline casts "split like a pair of trousers." Casts and albumin are usually present when the kidney-structure is involved by extension of the pyelitis, while marked pain in the region of the kidney indicates predominant pyelitis, though it does not exclude the possibility of coexisting nephritis. Marked vesical irritability points to associated cystitis, but in intense pyelitis with much pus and an acid urine vesical tenesmus may also be troublesome. Tuberculous can be discriminated from calculous pyelitis by finding tubercle bacilli in the pus. The presence of a fluctuating tumor in the lumbar region is significant enough of pus; but it may be difficult to determine whether it is due to pyonephrosis or perinephric abscess, although pyuria and the previous history of pyelitis, as well as the more circumscribed and less edematous character of the swelling of the former, are important distinguishing points.

Differential Diagnosis.—The *hemorrhagic pyelitis* of Senator, Delafield, and others, described as occurring in milder forms, and particularly in girls of neurotic types, may be distinguished by the intermittent hematuria and the occasional lumbar pain, lasting but a few days or a week, and followed uniformly by recovery. Digestive disturbances may be prominent in these cases.

Difficulty is sometimes experienced in diagnosticating pyelitis when coexistent with cystitis—*pyelo-cystitis*. These affections will not be confounded, however, when it is recollected that their histories differ. There is pain in one lumbar region in the former, and in the bladder in the latter.

According to Rosenfeld, (1) an alkaline reaction is not found in uncomplicated pyelitis; (2) the limit of albumin in the urine, even with severest cystitis, is 0.1 per cent. (maximum, 0.15); (3) if the pus-corpuscles are crenated, and, in the absence of vesical tumor, if the red corpuscles of a microscopic hemorrhage are chemically or morphologically decomposed, pyelitis exists, and especially if non-imbricated, small epithelial cells are practically absent. Stress is laid upon the relation of the albumin-content, which is from two to three times greater with pyelitis than with cystitis.

Prognosis.—Renal complications always make the pyelitis a serious affection. Catarrhal cases recover. Calculous pyelitis tends toward chronicity. Pyelo-nephritis and pyonephrosis are apt to end fatally from exhaustion or uremia. Perforation and the discharge of pus into the peritoneal cavity, pleural sac, intestine, and bronchi even, may precede death. The gravity of all cases of pyelitis depends upon the causes and upon the tendency to consecutive suppuration.

Treatment.—This varies according to the cause: the latter needs to be removed, its effects counteracted, and its return avoided. The treatment of calculous pyelitis is essentially the treatment of nephrolithiasis. Primary inflammation of the lower portions of the urinary tract must be combated; causes of retention of decomposed urine, as an urethral stricture or enlarged prostate, must be diminished; infectious fevers must be judiciously handled and irritating diuretics withheld.

Local measures are of value in all forms of pyelitis. Hot-water bags, fomentations, poultices, and dry cupping are often of great service. Internally, the use of diluents is to be recommended, especially the alkaline mineral waters, flaxseed tea, barley-water, skimmed and buttermilk, and lemonade.

Potassium citrate, uva ursi, pichi, buchu, and pareira brava are sometimes selected for their soothing properties. But, practically, none of the remedies named nor any other drug is of any avail when suppuration is once established. Irrigation by means of Kelly's ureteral catheter may be practised with good results in females. Hypodermoclysis of normal salt-solution may be of sustaining value at critical times in cases of infectious pyelonephritis. In chronic pyelitis salol and the oils of turpentine, sandalwood, juniper, copaiba, and erigeron have been used for their stimulating and alterative effects upon the mucous membrane. Surgical intervention is necessary in severe purulent pyelitis, pyelonephritis, and pyonephrosis.

HYDRONEPHROSIS.

Definition.—An obstructive accumulation of urinary fluid in the pelvis and calyces of the kidney; it may cause dilatation, pyelitis, or inflammation and atrophy of the renal structure.

Pathology.—Hydronephrosis is usually unilateral. The pathologic changes consist of a dilation of the pelvis of the kidney, associated with a degree of atrophy of the renal tissue depending upon the degree and persistence of the pressure. The accumulated fluid causes flattening and atrophy of the papillæ, and gradually of the tubules and glomeruli, as the dilatation and distention increase, until in extreme cases remnants only of the renal structure remain in the walls of the hydronephrotic cyst. The mucous membrane lining the pelvis and calyces first becomes thinned, and later thickened, by the growth of connective tissue, thus forming the dense sac-wall. There is also a growth of connective tissue in the renal parenchyma, medullary and cortical, a chronic nephritis with degeneration and atrophy of the renal cells being set up.

A *nephrydrotic cyst* may be very large, containing as much as several gallons of liquid. Sometimes in medium-sized sacs the external appearance of the walls may be lobulated; the interior, however, usually shows only partial septa projecting from the wall into the cavity of the sac. The smaller sacs partially enclosed by the membranous septa probably represent the dilated calyces. According to the seat of obstruction one or both ureters may also be dilated. If one kidney is affected, its fellow is often hypertrophied.

The fluid contained in the sac varies in composition, but usually is a clear, thin, yellowish, watery urine. The specific gravity is low, and the reaction is often slightly alkaline. Traces of albumin, urea, uric acid, and salts are found. Turbidity may be present, owing to admixture with pus, blood, or epithelium, but only in instances in which previous inflammatory conditions, as a calculous pyelitis, or subsequent complications of like nature have existed.

Etiology.—Hydronephrosis—or, better, *nephrydrosis*—is in most instances secondarily produced by diseases—congenital or acquired—that cause occlusion of the ureter. Probably from 20 to 35 per cent. of cases are congenital (Roberts). In these cases the causal condition is one of stricture, due to obstruction caused by a defective development or malformation in the urinary passage of one or both sides, usually the latter. Thus, there may be a valve-like formation or a very acute insertion of the ureter into the kidney. The dilation has occasionally become so great in the fetus as to cause considerable mechanical difficulty during labor.

Among adults, women are more often subject to hydronephrosis than men, and especially women who have borne children. The condition may be bilateral, as from a stricture low down and due to gonorrheal urethritis, but more often it is unilateral. The causes of these acquired cases are as follows: (1) Impacted calculi in the ureter or renal pelvis. (2) Disease of the ureteral walls, as inflammatory thickening and cicatricial stenosis from ulcers. (3) Flexion and twisting of the ureter, as from movable kidney. (4) Pressure upon the ureter from without, as by tumors and constricting bands (pelvic adhesions). The gravid and retrodisplaced uterus, uterine and ovarian neoplasms, enlarged and prolapsed spleen, and similar conditions causing compression or traction and obliteration of the lumen of the ureter, are found in this class. (5) Diseases and tumors of the bladder that involve the ureteral orifices, particularly carcinoma and papilloma, or that cause retention, as prostatic enlargement. (6) Urethral stricture.

Symptoms.—These depend somewhat upon the cause and extent of the hydronephrosis. Marked bilateral disease, when congenital, may render the fetus inviable. The unilateral variety may be overlooked for years, and no symptoms may point to the trouble until a tumor can be made out by inspection and palpation, or until the ureter of the remaining kidney may become obstructed and symptoms of uremia supervene. The latter are more apt to come on, and earlier too, in double hydronephrosis.

Locally, the patient may complain of frequent and severe ***pains*** that shoot about the affected loin and downward toward the thigh. Sensations of weight and a dragging discomfort are common. Anorexia, nausea and vomiting, eructations, and irregularity of bowel-action are associated sometimes. In large hydronephrotic cysts a continuous dull, aching pain only may be felt, or, as is not infrequently the case, the tumor may be absolutely painless. Obstinate constipation may result from compression of the colon, or in moderate enlargements diarrhea may occur from the pressure-irritation.

Usually a swelling is detected in the region of the affected kidney. It gradually increases in size, and in marked enlargements distinct bulging may be visible in the hypochondriac and lumbar regions. *Palpation* reveals a rounded, firm, yet somewhat elastic and sometimes fluctuating tumor. There may be slight tenderness. Dulness on percussion is found over the mass, except where the colon overlies it, when tympany is elicited; this is a characteristic sign of kidney-tumors. Moderate enlargements generally do not descend during inspiration. There may, however, be exceptions to this rule.

The *intermittent* form of hydronephrosis (Landau) is interesting from the variations that occur in the size of the tumors. A *marked diminution is coincident with a more or less sudden increase in the quantity of urine passed;* and, on the other hand, as the tumor gradually enlarges the flow of urine decreases. These cases are in most instances due to movable kidney. *Colicky pains* often usher in the periods of greatest distention preceding the sudden increase in the flow of clear urine. This variety of the affection occurs most frequently in women that have borne children. The *general symptoms* scarcely amount to more than a certain loss of flesh incident to the associated worry and anxiety. The filling of the nephrydrotic cyst, the distention, and the pain and discharge, with subsidence of the tumor, recur with variable frequency. According to Osler: "Among the circumstances liable to cause them are sudden and violent exercise, the jarring and jolting of riding and driving, any fatigue, mental emotions, and errors in diet." The tumor may continue to develop in size for several days after the pain has disappeared. The latter may last from several hours to a day. During the intervals, and after the urine has increased in quantity, gradually or quickly, the patient feels tolerably comfortable, and this sometimes for weeks or months. For obvious reasons the tumor is rather mobile in intermittent hydronephrosis.

The occurrence of chills, fever, and sweats, nausea and vomiting, abdominal distention, and rapid pulse usually indicates suppuration, and pyonephrosis may be the consequence. The urine will then be cloudy and reveal pus, following both discharge and aspiration. A lowered specific gravity and the presence of albumin will be noted when a chronic nephritis has been set up. Increased arterial tension and symptoms of acute febrile or chronic afebrile uremia may be added.

Hydronephrosis paraplegica is a form of the disease in which paraplegia develops as a complication.

The *course* of nephrydrosis is usually chronic, with variations and exacerbations depending upon the cause of the affection.

Diagnosis.—This is obviously very difficult in cases in which the accumulation of liquid is small. Characteristic signs are the gradual development of a tumor in either flank, as described above, with diminution in the urinary flow, followed by a more or less sudden free discharge and the subsidence of the tumor, with recurrences (as in the intermittent variety). When these do not occur and the tumor continuously enlarges, aspiration may be practised to determine whether the mass is solid or liquid; the nature of the latter may also thus be ascertained, whether urinary or not. Ureteral catheterization will determine which is the dry side.

The history of the case and the detection of some causative occlusion will point to the diagnosis.

Differential Diagnosis.—The nephrydrotic sac must be distinguished by exclusion from an *ovarian cyst*, *cystic kidney*, and *tumors of the spleen*, *liver*, and *gall-bladder*. Very large cysts may be mistaken for *ascites*. Assurance of the presence of the colon over the tumor is diagnostic, and a chemical examination of the fluid obtained by the use of the exploring needle will suffice in most cases. It should be remembered, however, that a slight amount of urea is sometimes found in ovarian cystic fluid. The presence of pus-cells in abundance in the aspirated fluid, with symptoms of suppuration, is significant of pyonephrosis.

Prognosis.—This is generally unfavorable, though in unilateral hydronephrosis evidences of compensation on the part of the unaffected kidney should render the case guardedly favorable, particularly if the cause be a movable kidney. The bilateral affection is always grave, owing to the danger of uremia. Infection of the cyst with pus-organisms is usually a fatal complication. Recovery may ensue in rare instances in which a spontaneous discharge of the liquid takes place. Rupture of the sac is unlikely.

Treatment.—The removal of the cause is seldom feasible. Symptomatic treatment only is required in mild cases, though sometimes gentle massage over the sac, properly directed and cautiously applied (to avoid rupture), may cause a reduction in the size of the tumor. Most often surgical measures only are of use. These embrace puncture and aspiration, incision (nephrotomy) and drainage, nephrorrhaphy, nephrectomy, and the formation of a renal fistula. These procedures, however, are undertaken only when successive reaccumulations of the fluid follow those measures first mentioned.

PERINEPHRIC ABSCESS.

(*Perinephritis.*)

Definition.—Suppurative inflammation of the connective tissue surrounding the kidney.

Pathology.—The suppuration attacks the lax adipose tissue or the fatty capsule in which the kidney is imbedded and the adjacent retroperitoneal tissue. The starting-point of suppuration is usually behind the kidney. There may be several small abscesses at first, but more often a single large abscess is found. The walls may be soft and shreddy, or in more chronic cases thickened and fibrous. A bulging externally over the affected lumbar region is not infrequent, particularly in large and extensive accumulations of pus. The latter has a tendency at a given point to burrow into the surrounding tissues, and especially downward toward the iliac fossa, pointing in the groin near Poupart's ligament. It may extend backward and open upon the skin-surface. Sometimes the pus perforates the diaphragm and discharges through the pleural cavity and lungs, or the colon, vagina, bladder, or peritoneum may be perforated. The pus is occasionally quite offensive, and may be ichorous from an admixture of infiltrated urine. Perirenal abscess due to calculous pyonephrosis may contain calculi that have ulcerated through pelvic or renal walls. Thickening of the adjacent peritoneum is often found. In certain cases of perinephritis, which usually gave no symptoms during life, the *postmortem* examination has revealed fibrous adhesions and a firm and thickened and fatty capsule, stripped with difficulty from the true capsule of the kidney.

Etiology.—Perirenal abscesses, when not traumatic in origin, develop most frequently as a result of purulent pyelo-nephritis or pyonephrosis. Hence they are *usually secondary.* Other primary conditions that may cause perirenal suppuration are the following: extension of inflammation from the ureter or pelvis of the kidney; from a pelvic abscess; from appendiceal or hepatic abscesses; and from spinal caries (psoas abscess) and empyema. Sometimes tuberculous processes in the kidney and suppurating new growths, as carcinoma and cysts (including the echinococcus), are complicated by perirenal abscess. More rarely such severe infectious diseases as typhus fever, small-pox, and pyemia lead to purulent perinephritis. Finally, there are cases for which no cause is discoverable.

Symptoms.—Subjectively, there is noted a *dull, throbbing pain* over the affected region that is increased by motion; sometimes, when the abscess is large and presses on the large nerve-trunks, the pains may become shooting in character and be felt in the leg on the same side. *Numbness* may also be felt. Pain and tenderness on palpation are common. The patient is prostrated, weak, and often quite emaciated, and flexure of the thigh on the affected side is frequent. The characteristic fever of suppuration is present in the deeply remitting or intermitting type, with alternating chills and debilitating sweats. Pus is found in the urine only when the kidney is involved. Sooner or later evidences of a *tumor* are seen; the areas can be palpated, and a gradual bulging in the lumbar area, increasing slowly, with smoothness and glistening of the skin and pitting (edema), may be observed. *Fluctuation* is frequently apparent in advanced cases, and in favorable cases signs of "pointing" appear.

Diagnosis.—Should the abscess tend to burrow downward, the condition may be somewhat obscure on account of the absence of distinct local symptoms. Indeed, involvement of the psoas may give rise to symptoms of coxitis, as pain referred to the knee-joint. The diagnosis is usually easy, and when in doubt as to whether the tumor is an abscess or an hydronephrosis or solid mass, the exploring needle should be used.

Differential Diagnosis.—An important point in differentiating perinephric abscess from suppurative pyelitis or pyelo-nephritis alone is the fact that in the latter the quantity of urine is usually diminished, whilst in the former there is less apt to be any interference with the renal secretion. Again, whilst in the latter the urine usually contains blood and pus, in the former the urine is free from blood, though not necessarily from pus, and casts are also absent here.

Prognosis.—This is guardedly favorable if the abscess points externally in the lumbar area. Of course rupture into the peritoneal cavity, bladder, bowel, and groin is always a serious occurrence.

The **treatment** is essentially surgical, and consists in free incision and drainage.

CYSTIC KIDNEY.

(*Renal Cyst.*)

Pathology.—Congenital cystic kidneys are in reality collections of cysts, varying in size from a pea to a marble, and separated from each other by septa of compressed renal or fibrous tissue. Either one, or frequently both, kidneys may be affected with what is sometimes termed *congenital cystic degeneration of the kidneys.* There is considerable enlargement of the organs, and during intra-uterine life they may attain a size so enormous as to render parturition extremely difficult and dangerous. The fetus is usually non-viable, though in mild cases the affection may be tolerated for some years after birth. The cystic fluid may be either clear or turbid, a reddish-yellow or a dark-brown in color, acid in reaction, and holds in solution urinary salts, blood, cholesterin, and sometimes uric acid and urea. A single layer of flattened epithelial cells lines the cyst-walls. The cysts themselves seem to be dilatations of the renal tubules and of Bowman's capsules, due, in some instances, to an obliteration of the tubules of the papillæ or to stenosis of some portion of the urinary tract.

The cystic kidneys usually met with in adult life (acquired) are of several varieties: (1) One or perhaps a few cysts may be present, larger usually than those in the congenital cystic kidney, which seem to cause no interference with the normal renal functions. Sometimes a reddish-brown colloid material is contained in these cysts.

(2) Small and often quite minute cysts frequently accompany the chronic nephritic kidney that is small, contracted, and cirrhotic. These result from dilated tubules and capsules when the former are narrowed by the hyperplasia of fibrous tissue.

(3) Cystic kidneys in adults may have the pathologic characteristics of the congenital variety—a mere conglomeration of cysts containing a clear or colored serum or a cloudy, dark, thick, and colloid liquid. This condition is sometimes associated with similar cystic disease of the liver and spleen. It may be a late manifestation of mild congenital disease. The kidneys have been found converted into cysts in cases in which the presence of calculi (uric acid) in the tubules has probably started the the cystic degeneration.

(4) Solitary cystic adenoma occurs rarely. It is in the form of a globular tumor projecting from the surface (usually the anterior) of the kidney. It may be as large as an orange, and may be enclosed in a distinct capsule. On section the mass is found to be composed of various-sized cysts separated by septa of fibrous tissue lined with cuboid or columnar epithelium. The remainder of the kidney appears to be quite healthy.

Etiology.—Cystic disease of the kidneys is either congenital or acquired. The former is probably commoner than the latter condition, may even be hereditary, and may persist for a while in extra-uterine life, while the acquired variety may be of unknown origin or secondary to chronic interstitial nephritis or to urinary calculi in the renal tubules. The direct cause of intra-uterine renal cysts is not definitely known, but they are probably developmental rather than pathologic, since other defects of embryonic growth are frequently associated with the disease.

Symptoms.—These may be absent in adults until the sudden development of uremia. Ordinarily, the clinical picture is similar to that of chronic interstitial nephritis. There is an increase in the quantity of urine, which is of low specific gravity; the normal solids are diminished in quantity: and aceto-soluble albumin may be present (Clifford Mitchell).[1] Slight albuminuria may be present. On *palpation* a large, rounded, and *sponge-like* mass may be felt in either hypochondrium or on both sides. Cardiac hypertrophy and increased arterial tension, as in chronic cirrhosis, are also frequently met with in cystic degeneration of the kidneys. Parker[2] reports a case which was followed by exfoliative dermatitis. Cystic disease of the liver may be associated.

The **diagnosis** can only be made upon the presence of the above symptoms and the discovery of the clear physical signs of the tumor. It should be pointed out that a possible complication of perinephric abscess, due to rupture of one or more of the cysts (as has occurred—Osler), would of course render a diagnosis wellnigh impossible.

Prognosis.—Bilateral cystic disease of the kidney must eventually prove fatal, owing to the sudden onset of uremia or cardiac failure. Solitary cysts give a tolerably favorable outlook under proper surgical interference.

Treatment.—The unilocular cysts just referred to above may be removed, capsule and all, and the kidney sutured. Bilateral disease cannot be operated upon for obvious reasons; unilateral cystic degeneration may be treated by nephrectomy, with narrow chances of success.

NEW GROWTHS OF THE KIDNEY.

The most common tumors of the kidney are those belonging to the class of adenomata (benign) and those that are either sarcomatous or carcinomatous (malignant).

Adenomata may be congenital or acquired. They grow in the cortex of the kidney in the form of small nodular masses, which in some cases may increase to a considerable size before any symptoms are produced. A cystic growth may be combined with adenoma (*cystic adenoma*), and *lymphadenoma* is also occasionally seen as a secondary growth. Other benign tumors that may affect the kidney are *angioma*, *fibroma*, and *lipoma*. Very large vascular adenomata may become malignant. Grawitz, Lubarsch, Kelly, and others have described a variety of tumor (*hypernephroma*) derived from aberrant adrenal tissue misplaced in the kidney.

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[1] *Philadelphia Medical Journal*, Aug. 19, 1899.

[2] *American Journal of Medical Sciences*, Sept. 1900

Sarcoma and **carcinoma** may be either primary or secondary. *Sarcoma* is frequently congenital in origin, and may have an admixture of striped muscular tissue. The presence of the latter in the kidney points to developmental disturbances during embryonic life as the cause of a variety of tumor known as *rhabdomyoma.* Alveolar sarcoma is also met with.

Renal carcinoma is probably of less frequent occurrence than sarcoma; it may, however, be found in children as well as in aged persons, the two extremes of life. Carcinoma of the kidney is usually of the soft medullary or encephaloid variety. As a primary affection it probably originates in the renal tubules. Both sexes are subject to the disease. Secondary carcinoma of the kidney, although probably more frequent than the primary form, is seldom of clinical importance. Renal carcinoma may occur as a diffuse infiltration or in nodular masses, one kidney usually being affected in primary carcinoma. The tumor sometimes reaches an enormous size, and instances are recorded in which nearly the whole abdomen has been filled, and in which the growth weighed as much as 31 lbs. (14 kgms., Roberts). Rhabdomyomata do not, as a rule, attain a very large size, though sarcomata may grow quite large. Softening and hemorrhage within these malignant growths may occur. The pelvis of the kidney may be invaded, and metastatic areas may form in the liver or the lungs, though this occurs in the case of primary renal carcinoma less readily than from carcinoma in other organs. Metastatic growths arise most likely through involvement of the renal vein. The renal parenchyma is either partially or wholly destroyed, the pyramids being attacked later than the cortex.

Symptoms.—*Lumbar pain* on the affected side is often an early symptom, and may persist throughout the course of the disease. It may be paroxysmal, and be felt extending down the thigh, or it may be dull, dragging, and limited in character. Pain is not, however, a constant symptom in a certain proportion of the cases.

Hematuria may occur early or late, and often appears before any tumor is palpable. The blood may be in a fluid state or in clots, the latter not seldom taking the form of pelvic or urethral casts, the passage of which may give rise to colicky pains. Casts of the ureter sometimes resemble lumbricoid worms. The hemorrhage may be excessive and cause marked weakness and a symptomatic anemia, superadded to the cancerous anemia that is usually present; on the other hand, it may be so slight as to be discoverable only microscopically. It recurs at irregular intervals of days or weeks. Large clots may accumulate in the bladder and cause vesical irritability. The urine from the healthy kidney may be quite normal, and may be secured for observation by ureteral catheterization. Cancer-cells or tissue-fragments of the neoplasm very rarely appear in the urine, at least so as to be distinctly recognizable as such. *Anorexia, nausea and vomiting, progressive loss of flesh* and *strength,* increasing pallor, and the concomitant symptoms of the cancerous cachexia are seen to develop.

Physical Signs.—These may not be sufficient to reveal the presence of the tumor for some time after the above symptoms have been observed. The appearance of a palpable tumor in either flank is a definite aid to diagnosis. It is felt between the ribs and pelvis latero-anteriorly, and at first, when small and on the right side, it may be movable. Both sarcoma and carcinoma of the kidney may assume enormous sizes. The tumor feels dense and hard (except rapidly-growing tumors, as encephaloid), either smooth or lobulated, and, when not too large, may retain the natural position and form of the kidney. The growth extends downward and inward, and in the very large malignant renal tumors of childhood the abdomen shows considerable enlargement, along with an abnormal pulsation and a prominence of the veins. Usually the tumor does not move with respiration. Percussion gives dulness over the mass, although in small and moderately large tumors the overlying colon may cause a tympanitic note to be heard. Neighboring organs, as the liver and spleen, may be found by palpation and percussion to be displaced by the renal tumor.

Diagnosis.—The presence of a tumor, when not too large and distinctly occupying the lumbar and lower lateral abdominal region, together with hematuria, pain of a local nature, and progressive failure of nutrition, may be looked upon as diagnostic of a malignant type of renal tumor. The relation of the colon to the tumor and immovability of the latter during respiration are also diagnostic. When the tumor is very large and adhesions have formed, as in cancerous kidney, it may be mistaken for other conditions.

Differential Diagnosis.—Affections such as hydronephrosis, pyonephrosis, cystic kidney, hydatids, ovarian, splenic, and hepatic tumors, and (particularly in children) retroperitoneal sarcoma must be differentiated from renal growths. Careful bimanual palpation will aid in the diagnosis, but the exclusion of other lumbar enlargements must be made by close attention to the history and to the development and course of the symptoms. Hematuria alone, in aged persons, is suggestive of carcinoma when no tangible cause for the presence of the blood is at hand. Hepatic and splenic tumors are usually movable during deep breathing, whilst renal tumors are not so. In cases of hepatic growths also the area of dulness extends higher, whilst in renal growths on the right side a tympanitic area generally lies between the liver and the tumor. The characteristic notch and edge of the spleen, and the absence of the overlying colon-tympany, are points that distinguish splenic enlargements from those of the left kidney. Pelvic growths (ovarian and uterine) enlarge from below upward, and are readily detected by vaginal examination. In children Löbstein's cancer (retroperitoneal sarcoma), if very large, is easily mistaken for a renal tumor, except that it is usually more centrally situated and more firmly fixed.

Prognosis and Treatment.—The termination in cases of renal carcinoma is inevitably fatal, and children succumb more quickly than adults. The disease may last from a few months to sometimes a year or two.

If the kidney be removed while the growth is still small, the prognosis is fairly good; but if large or if metastatic tumors have formed, the prognosis is always bad. Bloch warmly advocates in some cases the removal of small sections of kidney-substance, to avert the necessity of a nephrectomy by proving the non-malignancy of the growth. The *treatment*, aside from early surgical measures, is entirely symptomatic and supportive, and obviously it is unsuccessful. Renal colic, excessive hematuria, and a gradually lowered vitality may be met by the use of palliatives, tonics, and by nutritious and e[illegible] digestible diet. Nuclein may be tried hypodermically or by

II. DISEASES OF THE BLADDER.

CYSTITIS.

Definition.—Inflammation of the mucous membrane of the bladder. It may be either *acute* or *chronic*, the latter being clinically the much more frequent condition.

ACUTE CYSTITIS.

Pathology.—Cystoscopic examination performed according to Pawlik's or Kelly's method, hereafter to be described, reveals an intensely hyperemic condition of the vesical mucosa, which is puffy, edematous, and of a bright-red color; this may be more intense at points, especially in the vicinity of the trigone. The membrane is bathed in a thick, tenacious muco-pus, and here and there may be noted denuded areas, and the exfoliated epithelium often hanging in shreds from the bladder wall; overlying these denuded patches hemorrhagic effusions may be observed. In the severer grades of the disease the intense general hyperemia causes a disappearance of the blood-vessels that are to be seen in the normal condition. Occasionally small patches of ulceration, due to abscess-formation (*phlegmonous cystitis*), may be observed, and in rare and fatal instances the entire bladder-wall is involved in a necrotic process.

Etiology.—Cases of acute cystitis may be grouped according to their origin into four main classes, as follows:

(1) **Catarrhal.**—Like other mucosæ, the vesical epithelium is very responsive to systemic circulatory disturbances. Thus, sudden exposure to extremes of cold or heat or violent atmospheric changes, thereby abruptly suppressing the action of the skin, may be potent influences in the etiology of the disease. An intense acute catarrhal inflammation may follow retention of the urine in the bladder, with or without its subsequent decomposition; it may also be the result of pressure from an enlarged prostate or other tumor, and may follow cystocele, urethral stricture, or paresis of the bladder-wall. In simple over-distention of the bladder, with the accumulation of a gallon (4 liters) or more of urine, the so-called acute *exfoliative cystitis* may result, in which the entire mucous membrane of the bladder may be shed, and the patient shortly manifest all the symptoms of grave uremic intoxication. The prolonged retention of urine is followed by decomposition of the fluid, and this by its irritant action always excites a cystitis that soon assumes the chronic type.

(2) **Septic.**—This may result either from the direct introduction of pus-producing germs into the bladder or from the systemic transmission of these micro-organisms to the organ. This is known as the *bacterial origin* of cystitis. Under the first class may be mentioned the passage of a dirty catheter or sound; this is a cause of cystitis in puerperal women, and in men who are the subjects of minor grades of urethral stricture, and who have been subjected to gradual dilatation by means of bougies. *Gonorrheal cystitis* is also to be included under this heading. There is a condition known as *febrile cystitis*, which constitutes the second class of septic cases. This comprises the vesical in-

flammation that is present in the various febrile conditions, and which is probably a direct result of the presence in the urine of the causal bacilli or their toxins (Fitz). Thus, in all of the infectious diseases and fevers (typhoid and the other exanthemata, rheumatism, diphtheria, tuberculosis) there is noted a cystitis of varying degrees of severity that can be accounted for only by the local irritant action of the specific germ of the associated disease, or its eliminating toxins. The so-called *gouty cystitis*, which is often present in lithemic individuals, and which is due to the irritating and concentrated urine, may also be here included.

(3) **Toxic.**—Certain drugs when introduced into the system manifest an irritant action upon the vesical mucosa, and promptly excite a severe grade of acute cystitis. Prominent among these may be mentioned cantharides and other irritants of the urinary tract—cubebs, copaiba, and sinapis. Workers in coal-tar dye-stuffs are sometimes affected with acute cystitis.

(4) **Traumatic.**—Traumatic inflammation of the bladder follows the improper and careless use of the catheter, sound, or other instrument; the presence in the bladder of calculi or other foreign bodies; and the pressure of the fetus in parturition, or of large masses of impacted feces.

(5) **From Adjacent Inflammation.**—Irritation with consecutive inflammation may result from the extension of an inflammatory process from surrounding structures either by continuity or contiguity of tissue. Thus, a cystitis may follow a urethritis—gonorrheal or otherwise; it may result from an extension downward of a ureteritis, or it may be consequent upon a vaginitis, a malignant neoplasm of an adjacent viscus, a salpingitis, pelvic peritonitis, or pelvic abscess in the immediate vicinity of the bladder, as in the vesico-uterine pouch.

Symptoms.—The symptoms of acute cystitis are very marked. *Pain*, *vesical irritability*, *vesical* and *rectal tenesmus*, *frequency of micturition*, *fever*, and *urinary changes* are all pronounced. Prominent among these is *pain*, which may be most intense and is the earliest and most persistent manifestation of the disease. Its seat is the suprapubic region, whence it may radiate to the sacral region, the perineum, the end of the penis, or the upper portion of the thighs; it is most constant, but is worst just before micturition, by which it may be alleviated. It is considerably relieved by the recumbent posture, and is aggravated by pressure over the bladder. As the inflammatory process diminishes the pain gradually disappears, and the entire attack may subside in a few days or a week.

With the pain, and probably ranking second in severity, is the *rectal* and *vesical tenesmus*, or *strangury*. There is an almost constant desire to urinate, the patient sitting upon the urinal, it may be, for hours. The *urine* may be opaque or highly-colored. It is often bloody (in very acute cases the vesical contents may consist of a small quantity of pure blood only), is of a specific gravity varying from 1005 to 1030 (in the febrile cases), and contains pus-corpuscles in abundance, mucous flakes

rated and exfoliated epithelium (blad-

ococci, staphylococci, gonococci, proteus vulgaris, bacilli of tuberculosis, and very commonly the bacillus coli communis); fungous mycelial threads and yeast-cells have even been found in certain cases (*mycotic cystitis*). Its reaction may be either acid or alkaline; if alkaline, it contains ammonium urate, amorphous phosphates, and triple phosphates (crystalline) as a rule. More or less albumin will be noted, and on standing a dense sediment forms in the bottom of the flask, composed of all the foregoing substances, as shown by chemical and microscopic examination. The total quantity of urine voided in the twenty-four hours may be normal in amount or even slightly in excess of the normal. On the other hand, if exfoliation of the mucous membrane takes place, there may occur partial or even total suppression of the urine. *Fever*, with or without an initial rigor, persists throughout the attack, but is not of a severe type, save in the septic and malignant (diphtheritic) forms of the disease, when it may reach 103°–105° F. (39.4°–40.5° C.).

Abscesses may form, and betray themselves by localized pain, tenderness, and, in some cases, by a circumscribed induration. These may rupture into the bladder, followed by the free escape of pus from the urethra and by relief (temporary as a rule) from urgent symptoms, or they may spread to the peritoneum and induce peritonitis, which, if not promptly treated by surgical measures, may prove fatal by gradual asthenia.

In the variety associated with extreme exfoliation of the vesical mucosa *grave uremic manifestations* follow. These include all the features of the typhoid state (dry, brown tongue, mild delirium, nervous and muscular twitching; headache; gastric disturbances; and coma). There is also some degree of malaise and anorexia.

It must not be forgotten that acute cystitis may represent an acute exacerbation in the chronic form, and at times may assume a severe type of the disease.

Diagnosis.—Cystitis should be readily recognized from the history of the case and the frequency of the two almost pathognomonic symptoms—suprapubic pain and vesical tenesmus. An examination of the urine will reveal the characteristic clinical features. Cystitis may be confounded with *acute nephritis* or *pyelo-nephritis*, but a careful study of the clinical manifestations and, if need be, the catheterization of the ureters after vesical irrigation, will reveal the true condition. The presence of tube-casts in the urine would indicate renal involvement. The percentage of albumin is usually much larger in nephritis than in irritability of the bladder. The differentiation between cystitis and vesical irritability will be noted under the latter condition.

The **prognosis** of the milder grades of cystitis is good; the septic and malignant (diphtheritic) cases offer a much graver outlook. Extension of the process upward toward the kidneys is always serious.

Treatment.—The treatment of acute cystitis includes prophylactic, hygienic, and medicinal measures.

Prophylactic.—Most important is the prevention of the disease, and this includes, in addition to the usual care of the body, the observance of thorough asepsis whenever it becomes obligatory to introduce an instrument (catheter, sound) into the bladder.

Hygienic.—The cause of the disease, if evident (calculus, external pressure), should be sought and removed. The patient should at once be placed absolutely at rest in the recumbent posture. The value of this injunction will be most clearly understood when it is stated that in the erect position the intra-vesical pressure is three times that in the dorsal position. The simple observance of this law will do much toward relieving the sufferings of the patient. The *diet* must be regulated, and all irritating, highly seasoned articles of food must be interdicted. Alcohol in any form is prohibited. If it can be enforced, during the early stages of the disease an absolute milk diet will be most beneficial. The patient should be instructed to drink freely of water and other diluent drinks, whereby an internal irrigation of the bladder may be secured and much of the irritating substance removed. The free action of the skin may be secured by friction and warm bathing.

Medicinal.—The drugs to be employed are the saline laxatives and the various mild diuretics and urinary alterants. The reaction of the urine will indicate the variety of alterant to be employed. If it be acid, alkaline waters are serviceable, as the soda-preparations, Vichy, or the potassium salts. In alkaline conditions of the urine probably the most valuable drugs are benzoic and boracic acid and salol. Benzoic acid is best administered in the form of ammonium benzoate, which may be given in 10-grain (0.648) doses thrice daily in the compound infusion of buchu, or in uva ursi. Hot applications and hot local bathing (sitz-baths) will do much to relieve the pain and tenesmus ; if these be severe, a rectal suppository of opium and belladonna or an enema of chloral hydrate will generally give prompt relief. Tincture of cannabis indica, administered internally, may answer if opium be contraindicated. Under such a course as the preceding a cure may be expected within eight or ten days. It is prudent to advise the patients to wear flannel or silk binders over the abdomen, to avoid chilling of the surface and subsequent acute attacks.

CHRONIC CYSTITIS.

Pathology.—The vesical mucosa is not so hyperemic as in the acute variety, but is of a peculiar muddy or grayish-blue (slate) color, dotted here and there with patches of erosion or of actual ulceration. The muco-pus that bathes its surface is not so apt to be hemorrhagic as in the acute form of the disease, although slight hemorrhages may and do occur. Owing to the slow course and long duration of the disease there follows an immense thickening of the bladder-wall from hyperplasia of its constituents, conjoined with more or less edema of the tissues. The result is a contraction of the wall with a proportionate diminution in the vesical capacity. The mucosa may become, as it were, polypoid in spots, and there may follow obliteration or partial obstruction of the ureteral orifices, with consequent dilatation of the ureters and renal pelves from a damming back of the secretion. The urinary changes are about as in the acute form, save that the reaction is always alkaline and the amount of mucus and pus is proportionately greater.

Etiology.—Chronic inflammation of the bladder may be the result of a neglected or oft-repeated acute attack. It may occur from the persistent action of an exciting cause, as the presence of some irritati substance (calculus) in the bladder, or of some excitant external to that viscus, as a localized inflammation or a displaced uterus. Again, the inflammation of the bladder may be of a chronic tendency from the beginning; especially is this true of the tuberculous variety and of that due to neoplasmata of the organ.

The **symptoms** and **diagnosis** differ but slightly from those of acute cystitis. It may, however, be pointed out that the pain and tenesmus are less intense. Oppositely, the *amount of albumin* in the urine is comparatively large. The same remark applies to the *quantity of mucus* and *pus* (*vide* Pathology); indeed, the last-named ingredient often forms a thick gelatinous mass in the standing urine that tends to adhere to the receptacle. Chronic cystitis is accompanied by debility and emaciation, which, however, are of slow development.

The **prognosis** is always serious, and the course of the disease is at the best protracted.

Treatment.—Very generally, the treatment set down for the acute disease will not answer in the chronic form. Undoubtedly, there will follow more or less amelioration of the symptoms, but the tendency is toward a prolonged chronicity. In such cases, after the removal of the ascertainable causes so far as practicable, we are compelled to resort to local treatment of the bladder. This includes—(1) Vesical irrigation; (2) Topical applications; (3) Permanent drainage of the bladder.

Vesical irrigation is secured by means of an aseptic soft-rubber catheter which is connected with a graduated glass funnel: a siphonage is produced by the alternate elevation and depression of the funnel, which contains the irrigating fluid. The latter may consist of plain sterilized (boiled) water, sterile normal salt-solution (40–60 gr. to the pint—2.59–4.0 per ½ liter), or a weak solution of mercuric chlorid (1:50,000–100,000). The irrigation should be done slowly, and not more than twice or thrice daily in severe cases, and much less frequently in ordinary cases, according to the exigencies of the condition.

Vesical medication may be secured by means of the funnel after irrigation, the medicating substances being dissolved in a pint of water and allowed to flow slowly in and out of the bladder. The drugs that may be used in this manner are silver nitrate or zinc sulphate (1–5 gr. to the ounce—0.0648–0.324 to 32.0) or a saturated solution of boric acid. If the salts of zinc or silver are used, not more than an ounce of the solution should be allowed to enter the bladder, and much less than this amount will generally suffice. In cases in which there exist patches of ulceration the application must be made directly to these areas through the endoscope or cystoscope (Pawlik, Kelly). In women this may be readily done by placing the patient in the exaggerated lithotomy or knee-chest posture, dilating the urethra, and introducing the cystoscope, through which a reflected light is thrown upon the distended bladder-wall. Stronger solutions may now be employed, as silver nitrate, 20–30 gr. (1.29–1.94) to the ounce. This application should be followed by a slight irrigation of the bladder.

When this local medication fails to effect a cure, permanent drainage of the bladder must be secured—in the male by a suprapubic or perineal incision, and in the female by the establishment of a vesico-vaginal fistula. This places the bladder absolutely at rest, and gives the inflamed mucosa a chance to heal under proper medication.

As to internal remedies, various agents that possess a local stimulating effect upon the genito-urinary tract are advised by most authors, but I think little is to be gained from their employment as compared with the results achievable from topical treatment. Most efficacious among internal remedies are—oil of sandalwood, terebene, urotropin, pichi, buchu (fluid extract), and the oil of copaiba. If disinfection of the bladder *in loco* is not practicable, antiseptics should be given internally, combined with those stated above. Salol and potassium chlorate are excellent for this purpose.

NEOPLASMS OF THE BLADDER.

PRIMARY new-growths of the bladder are exceedingly rare, occurring, however, with greater frequency in males in about the proportion of 3 to 1; they may be either benign or malignant. On the other hand, secondary neoplasmata, particularly carcinomata, are relatively common. The most frequent variety of new-growth encountered is carcinoma, particularly the so-called villous or papillomatous carcinoma, Williams[1] finding in 20 women affected with bladder-tumor, carcinoma in 16. Other growths are sarcomatous, fibromatous, cystic, and papillomatous in nature.

The **symptoms** are the same for all varieties, and include, first and most commonly, *hemorrhage* (which is both persistent and free), together with pain, frequency of micturition, and occasionally the discharge of detached fragments of the growth. In carcinomatous cases of advanced standing cachexia will be marked. Examination by means of the cystoscope will reveal the nature of the complaint. In the case of secondary growths the primary tumor may often be detected.

The **prognosis,** of course, will depend upon the nature of the growth.

The **treatment** is purely surgical, and comprises enucleation of the tumor either by means of the snare, or after a vesical section.

VESICAL HEMORRHAGE.

(*Vesical Hemorrhoids.*)

HEMORRHAGE of the bladder has been mentioned as a symptom of various affections, both general and local, among the former being leukemia and malarial hematuria, and among the latter nephrolithiasis and tuberculosis and carcinoma of the bladder. It is also a prominent manifestation in stone in the bladder, and not infrequently appears in pregnancy (late). Independently of the operation of all of the above-mentioned etiologic factors, hemorrhage has been known to occur from the bladder, and recent precise methods of exploring the viscus (endoscopic examination) have shown it to be due to a hemorrhoidal state of the vessels. The hemorrhage may be profuse, and, rarely, even fatal in its effects.

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[1] *Brit. Med. Journ.*, 1889.

The **diagnosis** is based in part upon the absence of the more obvious causes of hematuria and the presence of free bleedings, but chiefly upon the result of a careful cystoscopic exploration of the bladder.

The **prognosis,** so far as my experience extends, is eminently favorable, though a few fatal cases have been reported.

Treatment.—This is mainly local. The bladder may be irrigated with an astringent solution (1 per cent. tannic acid, ½ per cent. alum), and this may be alternated with an antiseptic solution (3 per cent. boric acid, 1 per cent. salicylic acid). I have recently observed a case in which recovery followed the internal admission of the extract. hamamelis fluid. (ʒj–4.0), *t. i. d.*

NEUROSES OF THE BLADDER.

IRRITABILITY OF THE BLADDER.

Definition.—By this term is meant a condition of the bladder in which there exists an hyperesthesia of the organ, especially of the neck—that portion surrounding the urethral and ureteral orifices (*vesical trigone*)—without the presence of any tangible cause therefor. This must be distinguished from the irritability that is associated with true organic disease of the bladder itself, as in the presence of calculi, tumors, or fissure of the neck, or with disease of the surrounding structures.

Pathology.—There are no pathologic features to be noted. A cystoscopic examination of the bladder may reveal a slight increase in the vascularity of the mucous membrane, but the condition, in most instances at least, must be regarded as a true neurosis. The condition of irritable bladder in women, which has previously been held to be a purely functional derangement, is now regarded by Dacheux and Zuckerkandl as a localized hyperemia, especially at the *bas fond*, and less often at the beginning of the urethra.[1]

Etiology.—While in many instances no well-defined causal relations can be determined, it is very generally true that the patients who are the subjects of vesical irritability are individuals of a neurotic temperament, very often manifesting strong hysteric tendencies. They present the characteristic features of this unfortunate group. They are generally illy-nourished, fretful, irritable, peevish, suffering almost constantly from vague neuralgic attacks in different portions of the body (cephalalgia, tic douloureux, lumbo-sacral pain), and in a chronic condition of physical prostration. Frequently they eventually develop a true hypochondriasis or melancholia. In others there may be found a history of extreme mental and physical tire, overwork, business anxiety, over-indulgence in venery, menstrual irregularity, dysmenorrhea, ovarian or uterine disorders, long-continued gastro-intestinal disturbance (dyspepsia), improper hygienic surroundings, improper regimen, indulgence in late hours, and a general lack of will-power. It must, however, be remembered that subjects of chronic malarial intoxication very often manifest all the symptoms of vesical irritability, marked, it may be, by a feature of more or less periodicity. This has been termed by some *malarial fever of the urethra and bladder*. Lithemic individuals also are very prone to develop a pronounced vesical irritability, the affection in them probably resulting from the local action of the highly concentrated and irritating urine. The condition must commonly, however, be regarded as belonging essentially to the large group of neuroses.

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[1] *The American Year-Book of Medicine and Surgery,* 1897, p. 576.

In a certain percentage of cases the bladder-trouble is a reflex manifestation of some disease of an adjacent organ, as the urethra, ureter, vagina, rectum, anus, or the internal organs of generation. These are not, however, to be looked upon as cases of true neurotic vesical irritability.

Symptoms.—The symptoms of irritable bladder are mainly extreme painfulness and *frequency of micturition*, associated with marked *vesical* and *rectal tenesmus*. The dysuria is not always or altogether relieved by micturition; indeed, the pain may be just as severe, or even worse after, than before, the voiding of the urine. Especially is this true when there coexists a more or less spasmodic muscular action of the bladder-walls, the hypersensitive mucosa then being squeezed, and the patient suffering at times to such an extent as to be thrown almost into a state of collapse. There is usually a sense of weight or pressure in the pubic region, which is largely relieved when the patient assumes the recumbent posture. Urination is often performed spasmodically, or there may be a *spasm* of the urethra and neck of the bladder resulting in an utter inability to perform the act. The *urine* may be normal in appearance and amount. Very often it is increased in quantity (*hysteric polyuria*), and at times the opposite may be true and more or less suppression be noted. In lithemic cases the urinary characteristics already mentioned under that condition will be present (*vide* p. 405).

Diagnosis.—Very frequently will simple vesical irritability be confounded with true cystitis. The points of differentiation, however, are as follows:

IRRITABLE BLADDER.	CYSTITIS.
The patient is of a neurotic temperament, and generally gives no history of organic bladder-disease nor of operations upon the bladder.	May occur in any individual, irrespective of temperament. It frequently follows catherization, sounding, or other traumatism.
Pain is severe, and often worse after micturition.	The pain is usually much relieved by micturition.
The constitutional symptoms are those of nervous depression.	The constitutional symptoms are not marked, save in grave cases.
Never results fatally.	May result fatally.
The urine does not present any marked alteration in its physical or chemical qualities. It may show hyperacidity, or extreme concentration, or dilution.	There are always present marked and characteristic alterations in the physical and chemical qualities of the urine.
The appearance of the mucosa is negative in true neurosis.	Cystoscopic exploration reveals the angry and diseased mucosa, and may show the cause (calculus, tumor).
The duration is always protracted.	The duration of acute attacks may be short.

Prognosis.—Good as regards life; doubtful as regards the ultimate cure of the patient.

Treatment.—Since the condition is largely one of neurotic origin, the attention of the physician must be directed mainly toward a betterment of the state of the nervous system. Absolute rest, physical and mental, must be insisted upon, and the patient must be subjected to a course of strict moral suasion whenever this may be deemed necessary. Any cause of reflex irritation must be removed, and a careful search should be instituted for some such condition as cervical stenosis, uterine displacements, anal fissure, hemorrhoids, stricture of the rectum, vaginitis, urethritis, tuberculous infection of Skene's glands of the urethra, chronic gastro-intestinal catarrh, and the like. The habits of the patient must be inquired into, and late hours, the eating of improper and unwholesome articles of food, masturbation, or the reading of sensational and trashy literature corrected. In many instances the pronounced neurasthenic condition demands a course, more or less protracted, of the Weir Mitchell rest-treatment (*vide* Neurasthenia, p. 1181). The urine should be carefully examined for lithemic and other pathologic features, and by an appropriate course of treatment it should be rendered as bland and unirritating as possible. Large draughts of diluent drinks may be of benefit, and if these be combined with the prolonged administration of nerve-sedatives and antispasmodics, a marked amelioration of the patient's condition may be secured. In cases associated with spasmodic muscular contraction it may become necessary to employ an occasional suppository of opium and belladonna, or an enema of chloral hydrate. Change of air and scene, regulation of the diet, the institution of a proper course of gymnastics, mental and physical, and the observance of a happy and cheerful atmosphere will generally do much to improve the patient's condition. The administration of tonics (strychnin, iron) and the prevention of constipation are very essential. Especially must it be remembered that in all these cases of simple vesical irritability physical exploration of the bladder is absolutely contraindicated. The patient's mind must be directed away from the bladder in order to secure good results.

NEUROSES OF MICTURITION.

1. **Incontinence of Urine** (*Enuresis*).—An inability to retain the urine. This may arise from a number of causes. Frequently it is the result of some lesion of the spinal cord involving the sphincteric center of the bladder; this is known as *paralytic incontinence*, and is to be recognized by a constant dribbling, alternating with spurts of urine when voluntary or involuntary muscular action is brought into play, as in the act of coughing, sneezing, or bending forward of the body. It may be the result of a general bodily weakness or after prostrating diseases (typhoid, late stages of pulmonary tuberculosis). Again, it may result from some local condition in the bladder or urethra. Here may be mentioned paralysis of the urethra from over-dilatation or from traumatism, or that due to pressure of the fetal head in a prolonged labor; imperfect vesical innervation; over-distention of the bladder, producing a paresis of its walls; or from some temporary obstruction at the urethra or base of the bladder, such as a tumor or a sharply retroflexed uterus.

It may be a result of over-distention of the bladder, with partial paralysis of the sphincter, the bladder remaining overfilled, while there is a constant escape of a few drops of urine (*incontinence of retention*). It may follow some local causes of irritation, as the presence of vesical calculi, pressure from an anteflexed uterus upon the fundus of the bladder, cystitis, and parasites. The condition known as *spasmodic incontinence* is that due to an over-action of the compressor muscle of the bladder, as a consequence of which there is a diminution of the vesical capacity, the urine being forcibly and involuntarily ejected at irregular intervals. Finally, *nocturnal enuresis* is that variety which is so common in young, delicate, and often neurotic children: this is usually noticed in the early hours of sleep, and is often the result of some local irritation acting upon a hypersensitive organism, such as the presence of ascarides, an elongated prepuce, contraction of the urethral meatus, or masturbation. Bierhoff[1] is of the opinion that the essential or ultimate condition is hyperesthesia of the deep urethra or sphincter from hyperemia or inflammation. Nocturnal incontinence may be a manifestation of nocturnal epilepsy or of incipient cerebral or spinal disease (Fitz). The constant escape of urine in the paretic cases is apt to result in extensive excoriation of the parts.

The *treatment* varies according to the cause. The enuresis of children, if left alone, will eventually cure itself as the age and strength of the patient increases, though obvious exciting causes, if present, should be removed if not impracticable. Good hygiene, systematic evacuation of the bladder, elevation of the hips on a pillow in bed, plenty of out-of-door exercise, a change to the seashore or mountains, an abundance of suitable and strengthening food with a minimum of water, and the administration of tonics (iron, cod-liver or olive oil, and strychnin), will generally effect a cure. The fluid extract of rhus aromatica in 5- to 15-drop doses, thrice daily, has been very beneficial in children. Excellent results often follow the administration of minute doses of atropin or tincture of belladonna. A favorite formula of my own in cases possessing a hypersensitive nervous organization has long been as follows:

℞. Tr. belladonnæ,	ʒss–j	(2.0–4.0);
Sodii brom.,	ʒij	(8.0);
Ac. hydrobrom. dil.,	ʒijss	(10.0);
Ext. ergotæ fl.,	ʒij	(8.0);
Glycerini,	ʒj	(4.0);
Elix. simplicis, q. s. ad	℥iv	(128.0).

M. et Sig. ʒj (4.0) three or four times a day for a child of five years.

In very delicate or feeble children suffering from enuresis I substitute a motor tonic and stimulant (tr. nucis vom.) for the bromids or nerve-sedatives.

Spasmodic action of the vesical compressor may be relieved by the cautious use of the motor depressants, while its converse, paresis, demands the exhibition of full doses of strychnin or tincture of nux vomica. The judicious and careful use of the catheter, followed by the administration of strychnin, will promptly effect a cure in [illegible]nce of retention. Any local cause of vesical irritation must be removed. Galvanism in the paretic cases, applied both to the bladder and to the urethra, may be of service, and in the female Sänger suggests massage of the urethra. Should excoriation occur, bland ointments, as of zinc oxid and lanolin, should be used.

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[1] *Philada. Med. Journ.*, May

2. **Retention.**—Nervous retention of the urine is occasionally encountered in hysteric and highly neurotic individuals. Its most common manifestation is an inability to urinate in the presence of others. It is also occasionally noted after childbirth, when it may be due to nervous reaction, to edema and tortuosity of the urethra, or to a temporary inability of the bladder-walls to contract upon their contents, thereby permitting a longer retention of the vesical contents, and even favoring over-distention of the organ. If the urine be allowed to remain for too long a period in the bladder, fermentative changes follow and a secondary cystitis will result. Under these circumstances an exfoliation of a portion or even of the entire bladder-epithelium may be noted.

The *treatment* consists in the administration of strychnin and other nerve-tonics, in building up the general constitution, and in affording a change of air and recreation. In that variety following childbirth the patient should be urged to make voluntary efforts at micturition, and these may be seconded by the firm application of an abdominal binder and compress, or of hot, moist flannel cloths, kept up for twenty minutes or a half hour. The sound of running water, as when pouring water from a pitcher into the basin, often causes a contraction of the bladder and excites the flow of urine. It may become necessary, the foregoing methods failing, to resort to catheterization, the usual antiseptic precautions being observed.

PART VIII.

DISEASES OF THE NERVOUS SYSTEM.

THE central nervous system is generally divided into two parts—the brain and the cord. The *brain* consists of the cerebral hemispheres, the basal ganglia, the pons, the cerebellum, and the medulla. The cerebral hemispheres are joined together by the corpus callosum and the anterior and posterior commissures. They are united to the pons by the crura cerebri, and the pons is continuous with the medulla, which in turn is continuous with the spinal cord. The surface of the cerebral hemispheres is divided by sulci or fissures into various regions, known as the frontal, parietal, temporo-sphenoidal, and occipital lobes. The superior longitudinal fissure separates the two convolutions; the fissure of Sylvius is between the frontal and parietal lobes above and the temporo-sphenoidal lobe below. The fissure of Rolando divides the frontal from the parietal lobe, and the parieto-occipital fissure the latter from the occipital lobe. The continuation of the last-named fissure upon the median surface forms the upper boundary of the cuneus, the lower boundary of which is the calcarine fissure. The hippocampal fissure separates the fascia dentata from the hippocampal gyrus, and by its extension inward produces an elevation in the lateral ventricle known as the hippocampus major. Each lobe is subdivided by secondary fissures into a number of lobules. The topography of the hemispheres is important because it is now possible to map out with considerable accuracy the regions in which various motor impulses originate, and with less accuracy the regions in which various sensory phenomena are perceived. The accompanying diagrams illustrate, more satisfactorily than could any description, the regions that have been hitherto determined. There is some discussion in regard to the degree of individuality of these centers, but the weight of evidence inclines to the belief that they are not sharply delimited. Ordinarily speaking, one side of the brain innervates the opposite side of the body; but certain parts, as the muscles of the trunk, appear to receive impulses simultaneously from both hemispheres, and other functions seem to be accomplished exclusively upon one side; thus motor speech is ordinarily disturbed only when the lesion is in the left hemisphere.

The central nervous system is composed practically of two elements—the neuroglia, or supporting substance, and the neurons. The neuroglia consists of round cells with [illegible] lying in the midst of a tangled network of fibers. Its function appears to be exactly similar to that of connective tissue. The neuron, or nerve-unit, consists of a ganglion-cell, the protoplasmic processes springing from it, and the neuraxon, or axis-cylinder. The cell-body consists of protoplasm and nucleus. The latter contains a nucleolus and a small amount of chromatin; the former is composed of a reticulum of fibrillar ground-mass, in which are found, in certain cells, peculiar bodies, that take the basic stain, are irregularly spindle-shaped, and are often arranged concentrically to the nucleus; they also extend a short distance into the protoplasmic processes. The protoplasmic processes branch irregularly, and along the sides of the finer ramifications are placed short lateral offshoots, the buds or gemmules. The axis-cylinder is a single process, of uniform thickness, usually single, but sometimes branched, and giving off at regular intervals fine, long branches, the collaterals; it terminates either as a tuft of fine fibers surrounding a ganglion-cell, or in a motor plate in the muscles, or in a special sense-corpuscle in the skin. It cannot be too frequently reiterated that each neuron constitutes an individual unit that is entirely independent of all other neurons and has no anatomical connection with them whatever.[1] A physiological communication must, of course, exist, that perhaps is analogous to electric induction; and it has been suggested, by Dercum among others, that during life the protoplasmic processes move about and make contact with the surrounding nervous structures. The functions of the various elements of the neuron are as yet imperfectly understood. The cell-body appears to exercise a trophic action over the

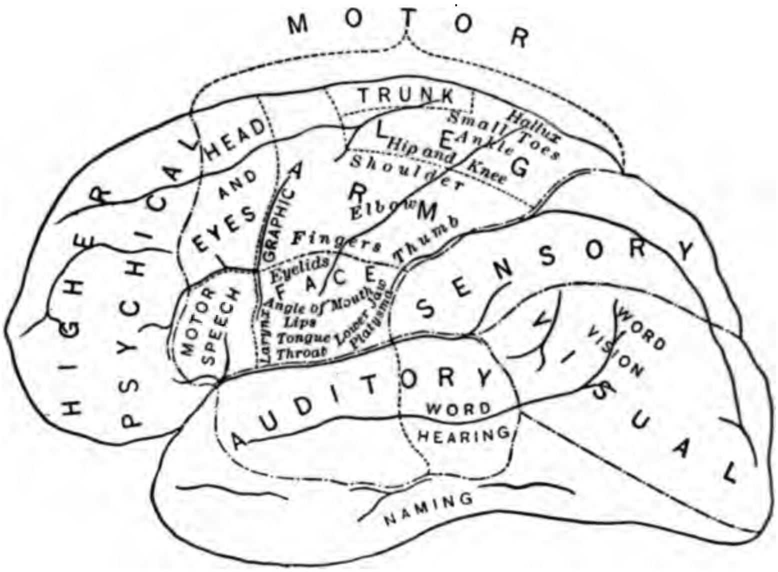

FIG. 65.—Diagram of the cortical centers and areas of representation on the lateral aspect of the hemicerebrum (Mills).

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[1] Apathy, and more recently Bethe, have claimed that delicate neuro-fibrils pass from one neuron to another. This has not yet been confirmed.

other parts, especially the axis-cylinder. It probably also generates the nervous impulses. The protoplasmic processes may have nutritive functions, or serve to conduct impulses to the cells (cellipetal). The axis-cylinder conducts impulses from the cell (cellifugal), except in the case of the peripheral process of the cells of the spinal ganglion.[1] A short distance from the cell the axis-cylinder is enveloped by the myelin-sheath, giving rise to the nerve-fiber, and when aggregated together these fibers form the white matter of the nervous system.

It has been possible to trace more or less accurately the course of many of the groups or systems of fibers. These exist because cells having the same functions are usually grouped together, forming centers or ganglia, and the fibers from these, taking the same course, form a bundle. Three classes are recognized: (1) fibers wholly within one hemisphere, fibriæ propriæ, uniting adjacent convolutions, and long association-fibers, uniting different lobes; (2) fibers passing from one hemisphere to the other, commissural fibers; (3) fibers passing from the cerebrum to the other parts of the central nervous system, the projection-fibers, forming the corona radiata.

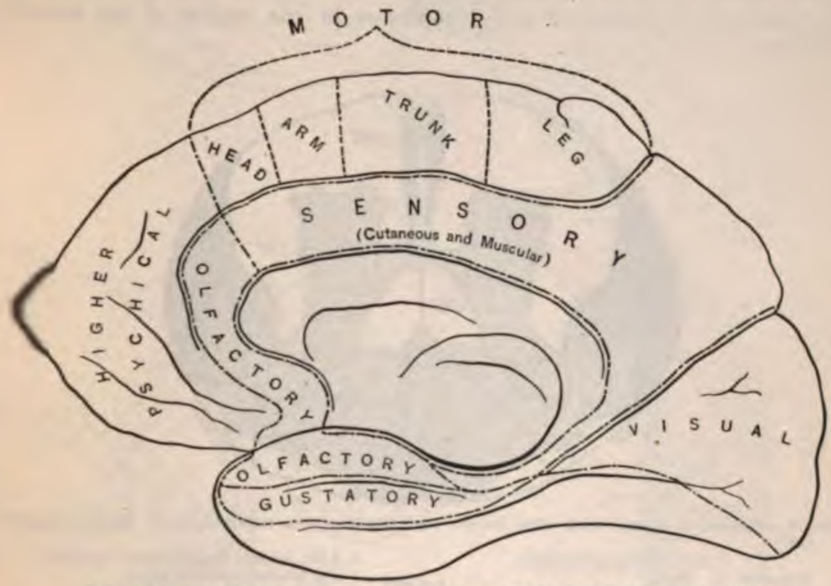

FIG. 66.—Zones and centers of the mesial aspect of the human cerebrum (Mills).

The columns or tracts that have been mapped out in the cord may be seen in the accompanying diagram (Fig. 67). In the antero-lateral portion are found the anterior or uncrossed pyramidal column, the antero-lateral column of Gowers, the cerebellar column, and the crossed pyramidal column. In the posterior region are the columns of Goll and Burdach. The rest of the white matter forms the so-called ground-bundles.

In the area comprising the anterior and lateral columns both ascending and descending fibers are found.

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[1] Lenhossek has suggested that this is a modified protoplasmic process.

The columns that transmit *ascending impulses* are—1. The direct lateral cerebellar column. 2. The antero-lateral ascending column of Gowers. 3. The antero-lateral ground-bundle or fundamental column. 4. The columns of Goll and Burdach. *Descending impulses* are transmitted chiefly by the direct and crossed pyramidal tracts and the antero-lateral descending tract. The direct lateral cerebellar tract of Flechsig takes origin in the cells of the column of Clarke, and first appears in the lower dorsal region, and passes through the restiform body to the cerebellum. Gowers' tract, or the antero-lateral ascending column, is first seen in the lumbar cord, and arises from some of the cells of the posterior horn. It then crosses to the other side of the cord through the posterior commissure and terminates in the region of the lateral nucleus.

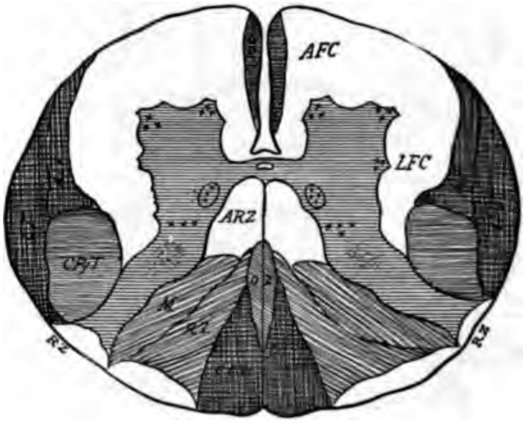

FIG. 67.—Section of spinal cord (after Dana), showing complete subdivision of white columns into—

Anterior columns.
- *DPy*, direct pyramidal tract.
- *AFC*, anterior fundamental column.

Lateral columns.
- *LFC*, lateral fundamental column.
- *LL*, lateral limiting layer.
- *CPyT*, crossed pyramidal tract.
- *CT*, direct cerebellar tract.
- *ALT*, antero-lateral ascending tract.

Posterior columns.
- Column of Goll.
- Column of Burdach.
- *RZ*, rim-zone, or Lissauer's column.
- *ARZ*, anterior root-zone.
- *MRZ*, middle root-zone.
- *OZ*, oval zone.
- *PRZ*, posterior root-zone.

The direct and crossed pyramidal columns constitute the great motor path by which fibers descend from the cortex and end in the motor nuclei of the cranial and spinal nerves—in the latter case in the multipolar ganglion-cells of the anterior horns. Their origin is in the motor region of the cerebral cortex—*i. e.* the ascending frontal and parietal regions, the paracentral lobule, and the posterior part of the inferior frontal convolution; they then approach one another, as do fibers from all parts of the cerebral cortex (known collectively as the *corona radiata*), to enter the internal capsule. This may be described as a wedge, bounded in front and to the inner side by the caudate nucleus and the optic thalamus, and on the outer side by the lenticular nucleus.

All of the fibers of the corona radiata do not pass through the internal capsule, some being lost in the gray matter of the basal ganglia, while

FIG. 69.

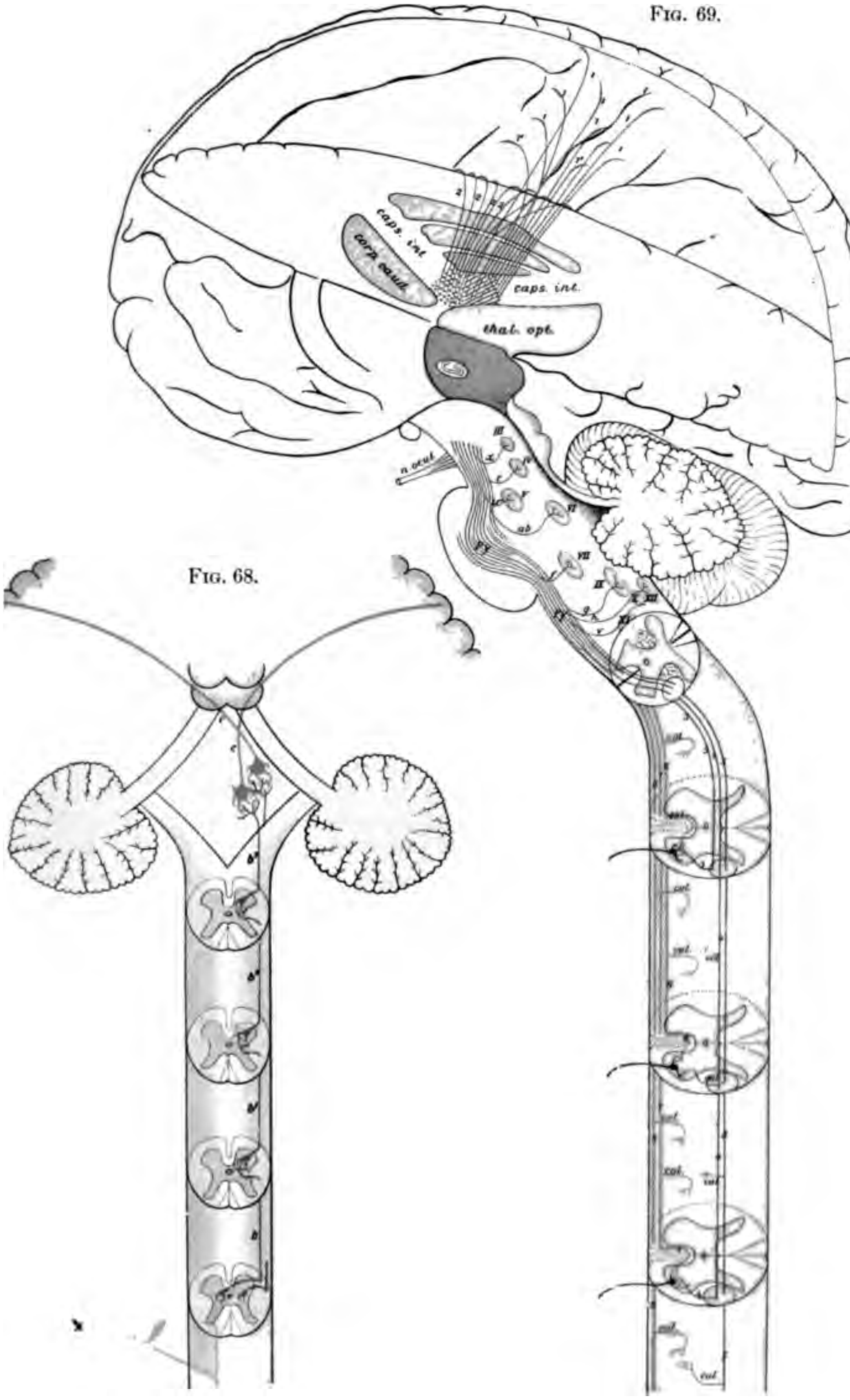

FIG. 68.

FIG. 68.—×, **Peripheral sensory tract**; *b*, *b*¹, *b*², *b*³, **cells in the short fibers, through the intercalation of which sensory impulses are conducted to the brain**; *c*, **continuation of the paths for sensory impulses leading to the cortex.**

FIG. 69.—1, Motor centers for the lower extremities; 1¹, **motor centers for the upper extremities**; **2, motor centers for the nerves of the face**; 3, 4, 5, **lateral pyramidal tract** (red); 6, 7, 8, **anterior pyramidal tract** (green); *py*, **pyramids** (red); *col* (red and green), **collateral fibers leading to gray substance.** *The Roman numerals* (III, IV, etc.) **indicate the nuclei, and correspond with the** *numbers of the cerebral nerves*; **the letters** (*g*, *h*, etc.) **represent the points of decussation and the** *names of the individual nerves.*

others take origin in the ganglia. The angle of the internal capsule is known as the genu or knee, the part anterior to it as the anterior limb, and the part posterior as the posterior limb. Through the anterior limb pass the fibers from the frontal region; in the region of the genu are the fibers for the muscles of the face and tongue; and in the posterior limb, the motor fibers to the extremities, also the sensory or tegmental fibers, and at its posterior end the fibers of the optic radiation.

The crusta consists of fibers that pass through the pons and enter the medulla, constituting its pyramidal tracts.

The tegmental fibers are continuous through the longitudinal fibers of the pons with those derived from the *formatio reticularis* of the medulla. This is formed by fibers from the superior cerebellar peduncles, the olivary body, and the posterior and lateral columns of the cord, which are reinforced in their upward course by fibers derived from the quadrigeminal and geniculate bodies.

Tracing the pyramidal fibers through the medulla, they will be found to divide into two unequal portions at its lower part. The larger decussates at this point (the region of the first and second cervical nerves), constituting the decussation of the pyramids; it then crosses to the posterior part of the lateral column of the opposite side, in which it runs as the crossed pyramidal tract.

In their course these fibers give off collaterals at right angles to themselves. These pass into the gray matter, and terminate in arborizations about the root-cells of the anterior horn of the same side. The main axes end in the same manner. As these main fibers with their collaterals pass into the gray matter at various levels of the cord, the tract becomes more and more attenuated, and terminates finally in the lumbar enlargement of the cord in the neighborhood of the third or fourth sacral nerve. The smaller division of the medullary pyramids passes directly into the anterior region of the cord without decussating, and is known as the direct pyramidal tract, or the column of Türck. In its course it gives off collaterals at right angles. These pass through the anterior commissure at different levels of the cord, and end in relation with cells of the anterior horn of the opposite side. The main fibers terminate precisely in the same manner.

Thus it will be observed that the fibers of the column of Türck decussate in the anterior commissure of the cord; like the tract previously described, it becomes gradually smaller from above downward, and ends in the lower part of the dorsal cord. The axis-cylinders of the multipolar ganglion-cells of the anterior horns pass out through the anterior roots of the same side and terminate in end-plates of muscles. Dejerine, Oppenheim, Monakow, and other neurologists believe that each motor cortex sends fibers to both sides of the body, and that the decussation of the pyramids is not a complete one, a small number of the fibers running in the lateral pyramidal tract on the same side as the lesion. This is borne out clinically by the slight paresis and the plus knee-jerk on the same side, neither of which, however, approaches in degree the palsy and increased knee-jerk on the side opposite to the lesion.

Pathologic confirmation of this view has been obtained by several observers, who have found degeneration in both latero-pyramidal columns in cases of a unilateral lesion in the motor cortex.

Motor-fibers from the nuclei of cranial nerves after decussating join with motor fibers of the internal capsule. The exact course of these fibers, however, has not been demonstrated anatomically. Since many of the muscles supplied by the cranial nerves functionate bilaterally—*e. g.* the eye-muscles and the muscles of mastication—the supposition is that in addition to fibers from its own nucleus each motor cranial nerve receives fibers from the corresponding nucleus of the opposite side. It was Broadbent who first pointed out that parts that functionate bilaterally are supplied from both sides of the brain.

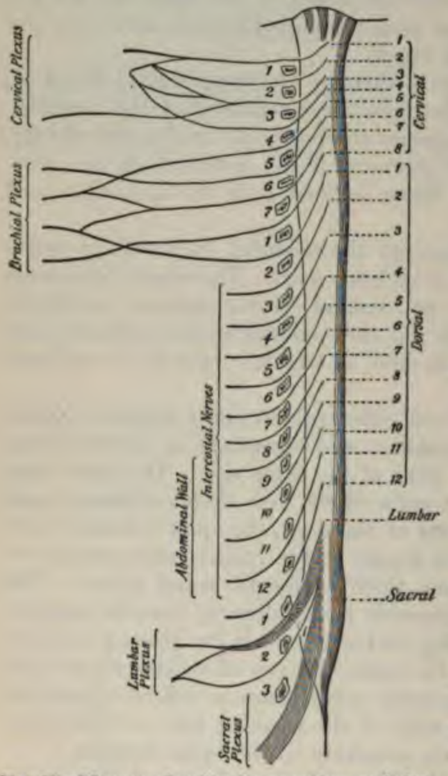

FIG. 70.—Diagram showing the groupings and plexuses of the spinal nerves (redrawn after Baker).

The course of the fibers of the posterior column is as follows:

The ganglion-cells on the posterior roots give rise to two fibers, fused for a short distance from the cell, but soon bifurcating. The longer of the two, the centrifugal fiber, extends to the surface and terminates in pointed or bulbous endings in the epidermis, or in special sensory nerve-endings in tactile cells, tactile corpuscles, or end-bulbs. The centripetal fibers or axons penetrate the cord, and divide in the white matter into ascending and descending fibers. The former may be either long or short. The short fibers are vertical at first, but finally bend into the gray matter, and end in relation with certain cells of the anterior cornua, forming perhaps a part of the reflex arc. Their collaterals end in a similar manner. The long fibers extend up the cord to the medulla, ending in the usual manner in the gray nuclei of the columns of Goll and Burdach; these are known as the *nucleus gracilis* and *nucleus cuneatus*, respectively. They also give off collaterals in their course. The descending fibers, on the other hand, are all short, and probably constitute the so-called *comma tract* of Schultze.

Since fibers continue to enter the cord at different levels, those that have entered below are pushed more and more toward the median line. It will thus be seen that the column of Goll is made up almost entirely of long fibers, and that the column of Burdach also contains long fibers, although it is probable that the short ones predominate. The long fibers are concerned in muscular coördination and equilibrium. It is likely that the fibers of pain and temperature sense, although entering by the posterior roots, do not pass up through the posterior columns, but rather through the gray substance of the spinal cord.

Since the post-natal growth of the vertebræ is more rapid than that of the cord, it follows that the spinal nerves assume a more and more oblique position, until finally the spinal segments, each of which consists of an anterior and posterior nerve-bundle with a transverse plane of white substance, lie considerably above the vertebræ after which they are named (see Fig. 65). The following table (Starr, modified by Mills and Dana from the experimental and clinical studies of Thorburn and others) shows the localization of function (not organs) in the different segments of the cord:

Localization of the Functions of the Segments of the Spinal Cord.

SEGMENT.	MUSCLES.	REFLEX AND CENTERS.	SENSATION.
First cervical.	Rectus laterales. Rectus capitis. Anticus and posticus. Sterno-hyoid. Sterno-thyroid.		
Second and third cervical.	Sterno-mastoid. Trapezius. Scaleni and neck. Omo-hyoid. Diaphragm.	*Hypochondrium* (?). Sudden inspiration produced by sudden pressure beneath the lower border of the ribs.	Back of head to vertex and neck. (Occipitalis major, occipitalis minor, auricularis magnus, superficialis colli, and supraclavicular.)
Fourth cervical.	Diaphragm. Deltoid. Biceps. Coraco-brachialis. Supinator longus. Rhomboid. Supra- and infra-spinatus.	*Pupillary* (fourth cervical to second dorsal). Dilatation of the pupil produced by irritation of the neck.	Neck. Shoulder, anterior surface. Outer arm. (Supraclavicular, circumflex, external musculo-cutaneous, cutaneous.)
Fifth cervical.	Deltoid. Biceps. Coraco-brachialis. Brachialis anticus. Supinator longus. Supinator brevis. Deep muscles of shoulder-blade. Rhomboid. Teres minor. Pectoralis (clavicular part). Serratus magnus.	*Scapular* (fifth cervical to first dorsal). Irritation of skin over the scapula produces contraction of the scapular muscles. *Supinator longus.* Tapping the tendon of the supinator longus produces flexion of forearm.	Back of shoulder and arm. Outer side of arm and forearm to the wrist. (Supraclavicular, circumflex, external cutaneous, internal cutaneous, posterior spinal branches.)
Sixth cervical.	Deltoid. Biceps. Brachialis anticus. Subscapular. Pectoralis (clavicular part). Serratus magnus. Triceps. Pronators. Rhomboid. Latissimus dorsi.	*Triceps* (fifth to sixth cervical). Tapping elbow tendon produces extension of forearm. *Posterior wrist* (sixth to eighth cervical). Tapping tendons causes extension of the hand.	Outer side and front of forearm. Back of hand, radial distribution. (Chiefly external cutaneous, internal cutaneous, radial.)
Seventh cervical.	Triceps (long head). Extensors of wrist and fingers. Pronators of wrist. Flexors of wrist. Subscapular. Pectoralis (costal part). Serratus magnus. Latissimus dorsi. Teres major.	*Anterior wrist* (seventh to eighth cervical). Tapping anterior tendons causes flexion of wrist. *Palmar* (seventh cervical to first dorsal). Stroking the palm causes closure of the fingers.	Radial distribution in the hand. Median distribution in the palm, thumb, index, and one half of the middle finger. (External cutaneous, internal cutaneous, radial, median, posterior spinal branches.)
Eighth cervical.	Triceps (long head). Flexors of wrist and fingers. Intrinsic hand-muscles.	. . . .	Ulnar area of hand, back, and palm, inner border of forearm. (Internal cutaneous, ulnar.)
First dorsal.	Extensors of thumb. Intrinsic hand-muscles. Thenar and hypothenar muscles.	. . . .	Chiefly inner side of forearm and arm to near the axilla. (Chiefly internal cutaneous and nerve of Wrisberg or lesser internal cutaneous.)
Second dorsal.	. . . .	. . . .	Inner side of arm near to and in the axilla. (Intercosto-humeral.)

SEGMENT.	MUSCLES.	REFLEX AND CENTERS.	SENSATION.
Second to twelfth dorsal.	Muscles of back and abdomen. Erectores spinæ.	*Epigastric* (fourth to seventh dorsal). Tickling mammary region causes retraction of the epigastrium.	Skin of the chest and abdomen, in bands running around and downward, corresponding to spinal nerves.
		Abdominal (seventh to eleventh dorsal). Stroking side of abdomen causes retraction of belly.	Upper gluteal region. (Intercostals and dorsal posterior nerves.)
		Vaso-motor centers. Second dorsal to second lumbar.	
First lumbar.	None.	*Cremasteric* (first to third lumbar). Stroking inner thigh causes retraction of scrotum.	Skin over groin and front of scrotum. (Ilio-hypogastric, ilio-inguinal.)
Second lumbar.	Vastus internus.	*Patellar*. Striking patellar tendon causes extension of the leg.	Outer side and upper front of thigh. Lumbar region. (Genito-crural, external cutaneous.)
Third lumbar.	Sartorius; adductors of thigh. Flexors of thigh.	. . . .	Front and outer side of thigh. Inner side of leg and foot.
Fourth lumbar.	Extensors of knee. Abductors of thigh.	*Gluteal* (fourth to fifth lumbar). Stroking buttock causes dimpling in fold of buttock.	Inner side of thigh, leg, and foot. (Internal cutaneous, long saphenous, obturator.)
Fifth lumbar.	Outward rotators. Flexors of knee. Flexors of ankle. Peronei. Extensors of toes.	*Achilles tendon*. Over-extension causes rapid flexion of ankle, called *ankle-clonus*.	Back of thigh and outer side of leg and ankle; sole; dorsum of foot. (External popliteal, external saphenous, musculo-cutaneous, plantar.)
First and second sacral.	Calf-muscles. Glutei. Peronei. Extensors of ankle. Small muscles of foot.	*Plantar* (fifth lumbar to second sacral). Tickling sole of foot causes flexion of toes and retraction of leg.	Back of buttock and thigh, side of leg and ankle: sole; dorsum of foot.
Third, fourth, and fifth sacral.	Perineal. Muscles of bladder, rectum, and external genitals.	Genital center. Vesical center. Anal center.	Circumanal region, anus, rectum, penis, urethra, vagina, perineum. (Small sciatic, pudic, inferior hemorrhoidal, inferior pudendal.)

To the foregoing table, which illustrates spinal localization, should be added another, showing what functions reside in the pons and medulla, as follows:

NUCLEI.	
III.	Sphincter iris. Ciliary muscles. Levator palpebræ superioris. Rectus internus (in convergence). Rectus superior. Rectus inferior.
IV.	Obliquus inferior. Obliquus superior. (Upper facial group.)
V.	(Associated movement of levator palpebræ.) Muscles of lower jaw.
VI.	Rectus externus. Rectus inter. of opposite side in lateral movements.
VII.	—Facial muscles.
XII.	(Lower facial group.) Muscles of tongue.
IX.	Muscles of pharynx.
X.	Muscles of esophagus.
XI.	Muscles of larynx.

Sensory Cortical Area.—Owing to the extensive compensation of sensory fibers, by means of which each side of the brain sends fibers to both sides of the body, it is impossible to map out the center with precision.

It is generally believed, for reasons already stated, that the central convolutions (motor area) contain muscular and tactile sensory functions. These are also spread out over the parietal lobe, and it is possible, indeed probable, that the sensory zone extends to the mesial surface of the hemisphere, as does the motor area. That this is the chief sensory center, as claimed by some observers, is, however, very questionable.

From the cuneus, fibers pass to the pulvinar, forming the optic radiation of Gratiolet. From the pulvinar they apparently pass to the external geniculate bodies, and thence to the anterior corpus quadrigeminum. The optic tracts arise by two roots that curve round the crusta on either side and unite immediately in front of the tuber cinereum. Fibers from the two tracts pass to the homologous sides of both retinæ; therefore the lesions posterior to the chiasm give rise to blindness of half of the retina on the same side.

Visual Centers.—The exact center for ordinary vision is in the cortex of the occipital lobe of the inner surface in the region of the calcarine fissure. A higher center exists, probably located in the angular gyrus, a lesion of which produces mind-blindness; this is a condition in which vision is not lost, but the objects seen are not recognized by the individual. Ferrier says that a lesion in this region sometimes gives rise to crossed amblyopia. The eye opposite to the lesions is chiefly affected, though vision is also restricted in the eye on the same side of the lesion (visual tract).

Olfactory Center.—This is located in the anterior part of the uncinate convolution, on the inner surface of the temporal lobe. It is possible, too, that fibers pass from this region through the anterior commissure to the cortex of the opposite hemisphere.

Auditory Center.—A lesion in the posterior part of the first temporal convolution produces a deafness in the opposite ear that is transient in character, owing to compensation. Bilateral lesions produce complete deafness. Mind-deafness, or an inability to understand spoken words, has resulted from a lesion in the first temporal convolution of the left side.

Speech Center.—The articulate speech center is located in the posterior part of the left third or inferior frontal convolution, and in the adjacent part of the ascending frontal in right-handed people (but on the right side in left-handed persons).

It is not known exactly what part the island of Reil plays in articulate speech. Word-blindness results from a lesion in the angular gyrus. Word-deafness results from a lesion in the posterior part of the first left temporal convolution.

Taste Center.—The area of cortical representation is unknown. By some it is located in the gyrus hippocampus.

Psychic Centers.—It is possible that the frontal lobes, anterior to the precentral fissure, contain the psychic centers. Such extensive compensation probably exists that no ordinary lesion produces mental aberration, but these centers are probably represented by the whole cortex.

The function of the cerebellum is that of coördination. Fibers pass from its cortex to that of the cerebrum, and *vice versa.* The impressions derived from the cerebrum are believed to be inhibitory.

Peripheral impressions reach the cerebellum through the direct cerebellar tracts of the lateral columns of the cord, and also from fibers derived from cells in the nuclei of the columns of Goll and Burdach.

Motor impulses run from the cerebellar cortex to the motor region of the cerebral cortex by way of the superior or middle peduncle, and by way of the inferior peduncle (restiform body) to the multipolar ganglion-cells of the anterior horns.

GENERAL AND TOPICAL DIAGNOSIS.

Nervous diseases are usually spoken of either as being *functional or organic;* but, as our methods of research become more refined and our technic more perfect, the breach between these two groups is being gradually but steadily lessened.

Organic nervous diseases may be produced by two types of lesions:

1. *Irritative,* causing an increase of function, continuous or intermittent. 2. *Destructive,* resulting in paralysis of motion or sensation, or both.

Irritative lesions are prone to become destructive in course of time. They may be operative in the upper segment, which includes the brain and fibers leading to or from it as far as the ganglion-cells of the cord; or in the lower segment, including the multipolar ganglion-cells of the anterior horn, together with the peripheral motor and sensory nerves.

When a complete pathway is involved a systemic disease is said to be produced. When two or more paths or neuron complexes are simultaneously involved combined systemic disease results.

Brain-lesions may be (*a*) focal or (*b*) diffuse. Cord-lesions are either (*a*) transverse, (*b*) focal, or (*c*) insular (a series of foci).

Cord-lesions result in ascending or descending degeneration, the destructive process travelling, as a rule, in the direction in which impulses are normally transmitted. In the fillet degeneration may extend up or down.

The theory has been advanced that the vulnerability of the tracts of the spinal axis is in direct proportion to the degree of their functional activity; hence the reflex (sensory and pyramidal) tracts are more likely to degenerate under nutritional disturbances or toxic processes than other parts.

It has been supposed that the tardy myelination of the pyramidal tracts predisposes to various nervous maladies, and particularly to those of a convulsive type. The following may be accepted as a general rule: the motor-nervous system is the last to develop, the first to lose, and the last to regain, its function; while the sensory nervous system is the first to develop, the last to lose, and the first to regain, its function. In making a diagnosis it is, therefore, of the utmost importance to try to determine the locality and extent of the morbid process, and to ascertain whether the lesion is a focal or systemic one. The symptomatology of systemic diseases is pretty constant, and, except in their very incipiency, they are usually not difficult of diagnosis. The symptoms of focal diseases, on the other hand, vary, of necessity, according to the location of the focus. They are often difficult and at times impossible to diagnose. Especially is this true of lesions occurring in the frontal lobes of the cerebrum, in the basal ganglia, and in the cerebellum.

Since the study of the motor centers and tracts has been pursued with so much more success than that of the sensory system, positive or negative motor phenomena occurring in the course of nervous diseases furnish us with much more valuable information than do sensory manifestations.

Further, motor symptoms are objective, and consequently appeal to us in a much greater degree than the sensory symptoms, which are purely subjective, and the elicitation of which depends so much upon the mental capability of the patient.

Irritative motor-lesions produce, according to the degree of irritation, either fibrillary muscular twitchings or mild or severe convulsions, tonic or clonic in character.

Destructive motor-lesions, according to their extent, produce mere muscular weakness, paresis, or actual paralysis of a single muscle, groups of muscles, or of the entire musculature of one or more limbs.

Irritative sensory lesions give rise to neuralgia, hyperesthesia, or hyperalgesia.

Destructive sensory lesions cause a more or less complete absence of sensation, as analgesia, anesthesia, or loss of temperature-sense.

Upper-segment or Upper-system Diseases.—A lesion occurring in the motor pathway anywhere between the cortex and the multipolar cells of the anterior horns (but not including the latter) gives rise to the following symptom-complex: Loss of motion, both automatic and volitional, and chiefly on the side of the body opposite to the lesion. The paralysis is usually spastic in type. The muscles resist passive movements, showing that their tone is increased. This is relative, and is due to the removal of cerebral inhibition, which allows the lower centers free play. They also tend to undergo shortening, and contractures result. Reflexes are increased chiefly on the side opposite the lesion, but also on the same side, the increase being the result of the removal of cerebral influences.

Owing to inactivity, the muscles of the paralyzed members undergo a more or less marked atrophy, though there are no degenerative changes, since the neuron bodies are intact. For the same reason the response to electric stimulation is not interfered with.

An irritative lesion of this upper system, particularly when operative in or upon the cortical region, gives rise to tonic or clonic convulsive movements. When the lesion is localized to a single center, focal or so-called Jacksonian epilepsy results. The cortex is wonderfully tolerant, when the lesion is of gradual onset and the parts accommodate themselves to the slowly increasing pressure. However, a local irritative lesion may at first cause widespread symptoms, due, as Nothnagel pointed out, to pressure, vascular disturbances, or irritative inhibition.

Lower-segment or Lower-system Diseases.—This includes the peripheral neuron system. Since there is no crossing of the fibers, the lesion and resulting paralysis are on the same side of the body. The paralysis, however, is of the flaccid, flail-like variety, hypotonus being present. The muscles offer no resistance whatever to passive movement, contractures do not occur, and reflexes are lost. Extreme degrees of wasting occur in this type of paralysis, owing partly to disuse, but chiefly to the fact that the neuron body, the nutritional or trophic center for the fiber, is injured. Pathologic changes therefore take place in the muscles themselves, and form a true degenerative atrophy. The protoplasm first becomes granular, and then fatty; it then breaks down and is absorbed. Its place is taken by the connective tissue, which is both relatively and absolutely increased, so that in the course of time fibrous masses alone remain. Electric changes also occur. The muscles first cease to respond to the faradic current, and soon respond in an abnormal manner to the galvanic. Instead of short, sharp contractions, they react in a slow, wavy manner, ACC being stronger than KCC. Irritative lesions of this system cause fibrillary muscular contractions and peripheral convulsions, of which laryngismus stridulus is a type.

I. DISEASES OF THE PERIPHERAL NERVES.

NEURITIS.

Definition.—An inflammation of a nerve or of its fibrous envelope.

Pathology.—A true neuritis is almost always an inflammation of the nerve-sheath or of the septa between the fasciculi, and usually begins as a perineuritis. The so-called parenchymatous neuritis is really a degenerative process; it is prone to follow neuritis, and is the result of excessive or prolonged irritation or of pressure by the products of inflammation. The affected nerve becomes red and swollen. The sheath becomes hyperemic and the seat of a round-cell infiltration.

We may have a *perineuritis* or an *interstitial neuritis*. Again, these may be *focal* or *diffuse* (*disseminated*), involving limited patches or continuous areas of a nerve. Finally, many nerves may be simultaneously affected, constituting a *multiple neuritis*. In the parenchymatous form the ordinary signs of inflammation are absent. The nuclei of the sheath increase in size and number, and the protoplasm about them increases in amount. The white substance of Schwann becomes segmented, breaks up into droplets, then becomes granular and fatty, and is finally absorbed. The axis-cylinder becomes discontinuous at the site of disorganization of the myelin. Ultimately, there may be seen scattered promiscuously among the more or less healthy fibers the withered nerve-sheaths, containing many nuclei, some granular débris, and pigment. Occasionally fatty aggregations occur along the nerve. Leyden has termed this condition *lipomatous neuritis*, but it is not worthy of a special name, as it is only a stage in the ordinary degenerative process.

Parenchymatous degeneration is similar to the secondary or Wallerian degeneration previously mentioned. It is the chief lesion in multiple neuritis, though in this disease changes have also been found in the multipolar ganglion-cells of the anterior horns.

Etiology.—(*a*) *Focal neuritis* may be due to—(1) Exposure or cold (the so-called *rheumatic neuritis*). (2) Extension of inflammation from neighboring parts. (3) Traumatism—wounds, compression, excessive stretching resulting from fractures or dislocation. (4) Microbic and autogenetic poisons.

(*b*) *Multiple neuritis* may be due to—(1) Poisons of extrinsic origin

—alcohol, carbon bisulfid, lead, arsenic, mercury, ether. (2) Poisons resulting from the infectious fevers (typhoid, diphtheria, variola, typhus, leprosy, beri-beri, measles, syphilis, tuberculosis, septicemia, malaria, influenza). (3) Cachexias, anemia, carcinoma. (4) Auto-intoxication. (5) Cases arise in which no definite cause can be ascertained; these are the so-called *idiopathic* or spontaneous cases.

Symptoms.—(*a*) **Focal Neuritis.**—In localized neuritis the symptoms vary according to the function of the nerve involved. In the case of a sensory nerve there is pain, usually of a boring or shooting character, along its course and distribution. There is also tenderness on pressure along the nerve, and especially at its point of emergence from bony canals. Weir Mitchell believes this to be due to irritation of the *nervi nervorum.* The skin is generally hyperalgesic (though tactile sensation is often lowered), reddened, sometimes edematous, and local sweatings may occur. In the more chronic cases trophic symptoms eventually arise, as glossiness of the skin and an impaired growth of the nails. When a motor nerve bears the brunt of the attack fibrillary twitchings will be observed in the muscles it supplies, and are soon followed by more or less impairment of motion, even amounting to paralysis; sometimes contractures occur, and ultimately wasting of the muscles, and even reactions of degeneration, take place. When both motor and sensory nerves are simultaneously involved the symptoms will necessarily partake of a mixed character. The constitutional symptoms are, as a rule, of little moment.

(*b*) **Multiple neuritis** is an involvement of the peripheral nerves in various parts of the body, affected simultaneously or in quick succession, and due to endogenous or exogenous poisons. Lettsom's paper, published in 1789, embodied the first description of the disease.

Among cases due to poisons of extrinsic origin is *alcoholic neuritis.* In 1822, James Jackson, of Boston, clearly gave its clinical history, and Dumesnil in 1864 published the result of an autopsy upon a case. Other pioneers were Leyden, Buzzard, and Ross. This is the most common type of multiple neuritis. It results from spirit-drinking in moderate amounts and continued over a long time. The onset is generally slow, being preceded by gastric catarrh, insomnia, tingling of the extremities, a rapid, weak heart, and a tendency to sweating on exertion. Some muscular twitching and paresis may exist contemporaneously, but the loss of power soon becomes more marked—first in the lower and then in the upper extremities, the extensors being chiefly affected. Wrist-drop and foot-drop follow. Occasionally paraplegia and, more rarely still, a loss of control of the bladder and rectum take place. Fever is rarely present. Sensory symptoms may vary from the tingling or numbness already noted to burning or boring pains of great severity. The skin is hyperesthetic at first, at all events. Later, paresthesiæ develop, with anesthesia and a more or less decided loss of muscular sense. The muscles are tender when touched.

The cutaneous reflexes are preserved unless the anesthesia is marked. The knee-jerks are generally lost, though exceptionally they may be increased. In the less severe cases a certain amount of incoördination may be present. When this is the case the absence of the knee-jerk, the loss of muscular sense, ataxia, and the pains in the extremities simulate locomotor ataxia, and the term *pseudo-tabes* has been applied to the condition. Vaso-motor and trophic symptoms appear, and in some cases the special senses are involved (impairment of vision, amblyopia, limitation of the color-field). The cerebral symptoms are important. They may be so slight as to consist merely of loss of memory, irritability, perhaps an hallucination or illusion (particularly after nightfall, and especially if the patient has had insomnia), or they may be of the type and degree seen in general paralysis. The duration of an attack varies from a few weeks to a year or so.

Arsenic neuritis differs from the above in that the head-symptoms are generally absent. The onset may be much more abrupt and the course is usually shorter.

Carbon bisulfid neuritis occurs chiefly in workers in rubber-factories. There are noted intense frontal headache, giddiness, marked excitability, muscular cramps, and possibly convulsions. *Saturnine neuritis* is confined to motor nerves, and especially to those of the upper extremities. Very rarely some disturbance of sensibility may result. Lesions of the anterior cornua are more likely to occur in saturnine multiple neuritis than in any of the other varieties. Head-symptoms are not common, but optic neuritis and convulsions may occur.

Cases due to an attack of some infectious disease may be local or multiple, and generally present the same symptoms as neuritis due to any other cause. (1) *Malarial Neuritis.*—According to Romberg, malaria gives rise at times to "intermittent paraplegia." The legs of the patient suddenly become paralyzed, with or without alteration of sensation or loss of control of the sphincter. That the cause is probably malarial is shown by the fact that the condition is periodic, each attack subsiding with a critical sweat, and finally yielding to quinin. (2) *Recurring Multiple Neuritis.*—A few cases have been reported in which attacks of more or less widespread paralysis, due to neuritis, have recurred.

Spontaneous or the so-called *idiopathic neuritis* does not differ from the general type of the disease, except that no cause can be discovered to account for it.

Diagnosis.—This does not present any difficulty, as a rule. The spontaneous cases, in the early stages, may simulate *acute spinal paralysis* or *acute ascending paralysis.* The fever, palsy, electric change, and the loss of knee-jerks are common to both, but in *acute spinal paralysis* there are never any sensory symptoms. The palsy in idiopathic cases rapidly spreads, but soon subsides again.

In other forms of peripheral neuritis the onset is not only apt to be less abrupt, but some sensory symptoms are almost invariably present: the distribution of the palsy is more symmetric bilaterally, and after it has reached its acme no improvement takes place for a few weeks or months. In *ascending paralysis* there are no sensory symptoms, the knee-jerks are preserved, there is neither muscular atrophy nor electric change, and the order in which the paralyses supervene differs from that of peripheral neuritis.

Cases of pseudo-tabes are sometimes confounded with *locomotor ataxia.* The main points of differentiation are included in the following table:

PSEUDO-TABES.	LOCOMOTOR ATAXIA.
The course is shorter, and often results in recovery.	The course is progressive from bad to worse, and chronic in nature.
Pain is never of the fulgurant type.	Fulgurant pains often are present. Pain-crises are almost diagnostic.
There is tenderness over the nerve-trunks.	There is no tenderness over the nerves.
Sensory disturbances are more marked (tingling and numbness).	Sensory disturbances are less marked.
Argyll-Robertson pupil is absent.	Argyll-Robertson pupil is present.
There is a "foot-drop," with the typical "steppage" gait.	No "foot-drop." The toes are raised, and the foot is brought down flatly, with the heel first.
Paralysis is often present.	There is no actual loss of power.

Prognosis.—Peripheral neuritis may terminate in one of the following ways, according to Drs. Gibson and Fleming[1]: 1. In complete recovery; 2. With damaged peripheral nerves; 3. With injury to the central nervous system, such as to cause symptoms of ataxia, spastic paraplegia, or disseminated sclerosis; 4. In death, from failure of the organic centers, especially that of respiration. The prognosis is generally good, though in the acute variety (from any cause) it should be guarded, and occasionally is grave. Exposure and chill, alcohol, diphtheria, and beri-beri give rise to the most serious types, and often cause death by failure of the heart or respiration or by coagula in the vessels. Mild cases may entirely recover in a few weeks, while severe ones often require a year or two.

Treatment.—First ascertain the cause, and, if possible, remove it. It may be unwise in alcoholic cases to stop the alcohol suddenly, but each case must be judged on its merits. Rest is very important, and all sources of worry should be stopped. Locally, anodynes may be employed and the part wrapped in cotton wool. In febrile cases, especially in the earlier stages, the salicylates are valuable. The general health should be toned up by strychnin and tonics, and by nourishing but easily digestible food. Further medication will depend upon the etiology, quinin being demanded in malarial, and mercury or the iodids in syphilitic cases. As soon as the acute symptoms have subsided massage and passive movements should be begun, galvanism applied to the muscles, and warm-water or sulphur baths administered.

BERI-BERI.

THIS is a tropical disease, characterized by weakness, wasting of the muscles, paralysis, anasarca, anemia, numbness, pain, areas of anesthesia, and diminution or loss of tendon-reflexes. There are two forms, the *acute* and the *chronic*. Its etiology is obscure, though it is apt to supervene upon any condition that impoverishes the physical or nervous vitality. Intestinal parasites have been said to cause it. Ogata of Tokio has described a specific bacillus; Pikelharing and Winkler, however, claim that it is due to a micrococcus. Whatever its cause, the researches of these latter observers, together with those of Baelz and Sheube in Japan, prove it to be a peripheral multiple neuritis. The symptoms of the *acute form* are fever, anemia, general edema, effusion into the serous cavities, dyspnea, precordial pain, vomiting, and peripheral paralysis. Death often results, even in a few days, from emboli or thrombi in the pulmonary or systemic circulation. In the *chronic form* the symptoms are less pronounced. The face is apt to be puffy, and palpitation and serious cardiac dilatation may occur. The gait is tottering, the muscles are somewhat wasted, the tendon-reflexes are lost, and paresthesiæ develop.

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[1] *Edinburgh Hospital Reports*, vol. iii.

The cases associated with the cachectic states may be general; though usually they are local and of the interstitial variety of neuritis.

The cases due to auto-intoxication are usually associated with fever, and at first simulate rheumatism or some infectious disease. Soon, however, the tingling, pain, palsy, loss of the knee-jerks, and anesthesia reveal the neuritis. Death may result from cardiac or repiratory paralysis. When life is spared the convalescence is exceedingly slow.

NEUROMATA.

NEUROMATA, or tumors of nerves, have been described as (*a*) true and (*b*) false.

(*a*) *True neuromata* consist of medullated or non-medullated nerve-fibers (the myelinic and amyelinic varieties—Virchow), and rarely of ganglion-cells also.

(*b*) *False neuromata* contain no nerve-elements. The growth is situated on the nerve-trunk itself, and consists of either fibrous, myxomatous, gliomatous, or sarcomatous tissue.

Neuromata have also been classified according to their situation as (1) Stump neuromata, or bulbous nerves; (2) Subcutaneous neuromata, or *tubercula dolorosa;* (3) Nerve-trunk neuromata; (4) Plexiform neuromata.

(1) *Stump neuromata* develop on stumps or on the ends of divided nerves as the result of traumatism. They may consist of fibrous tissue, but are usually myelinic.

(2) *Subcutaneous tumors*, or *tubercula dolorosa*, are painful, as the latter word implies, and are apt to be multiple. In individuals so afflicted nerve-trunk neuromata may coexist.

(3) *Nerve-trunk neuromata* are usually multiple. In one case quoted by Gowers as many as 3020 were found. They may be true or false. In the former case the nerve-fibers are less apt to be interfered with than in the heterologous growth.

(4) *Plexiform neuromata* consist of beaded and tortuous, interlacing neural cords. They are usually congenital.

Etiology.—Neuromata may be due to traumatism. When multiple, however, they are usually hereditary, occurring in families of a neurotic or strumous diathesis. They are most commonly found in men.

Symptoms.—There may be none. When present their character necessarily depends on the nature of the nerve involved and whether the lesion is an irritative or destructive one. More or less pain, numbness or tingling, paraesthesia, and palsy are among the most common symptoms. Various reflex manifestations have been described, and epileptiform convulsions have been attributed to their presence.

Treatment.—Apart from anodynes, operative measures are alone of value, except when the tumors are the result of syphilis, as occasionally happens; in such cases specific treatment must be employed.

It must not be forgotten, however, that stump neuromata may occur in those hereditarily predisposed, in which case, as Bowlby has pointed out, their removal will almost surely be followed by a return.

NEURALGIA.

Definition.—Neuralgia (*nerve-pain*) is the result of some irritation directly or indirectly applied to a nerve. While this is true of all pain, yet the special nerve-pain under consideration presents the following characteristics: 1st. In its distribution it follows the course of a nerve-trunk or its branches. 2d. It shows a tendency to shift from place to place. 3d. The presence of painful points (*points douloureux*). 4th. Intermission and remission of pain.

The pain of neuralgia varies both as to character and intensity. It may be merely a mild ache, or, on the other hand, it may give rise to the most excruciating agony; it may be of a throbbing, boring, tearing, shooting, or burning character, or it may come on in shock-like paroxysms. Any nerve in the body may be affected. Quite often one can find no definite cause of the neuralgia, and as we are not certain as to its ultimate pathology, it may be due to some slight inflammation of the nerve, or to hyperemia, ischemia, exudation, and the like.

Among the *predisposing causes* are—(1) Age, the condition being most common in those between thirty-five and fifty years, and less so above that age. It is least common in children.

(2) Sex. On the whole, neuralgia is most common in women, though the severer grades are found quite as frequently in men. Sciatica is more common in men, while trigeminal neuralgia occurs more frequently in women.

(3) Heredity. Neuralgia is very prone to occur in a family in which hysteria, epilepsy, or other neurosis or psychosis is present.

(4) The general physical condition. In persons reduced by illness or by mental or physical exertion, and in anemia, neuralgia is common.

(5) Occupation. Painters and workers among metallic dust are specially predisposed.

The *exciting causes* are—(1) Exposure to cold and wet. (2) Mechanical, chemical, or thermal irritation, including compression. (3) Traumatism. (4) Neuromata. (5) Infectious diseases. (6) Rheumatism and gout. (7) Endogenous or exogenous poisons. (8) Local disease in the course of the nerve, or of the mouth, nose, or orbit.

We are hardly justified to-day in speaking of idiopathic neuralgia. Such cases probably result from some endogenous or exogenous poison, an auto-intoxication, or malaria, rheumatism, syphilis, some metallic poison, or alcohol or tobacco. Finally, a form exists which we speak of in no certain manner as "reflex neuralgia," and which is said to result from disease of the sexual or other organs often remote from the painful nerves.

General Symptomatology.—The neuralgic attack may be of sudden or slow onset, with or without prodromata. When the latter exist they consist of a sense of uneasiness, perverted sensations, chilliness, and stinging or slight burning pains. The pain may be of the character previously described, either strictly localized or radiating to neighboring nerves, and may be aggravated by drafts, movements, or mental perturbation. On pressure certain tender or painful spots will be found, especially where the nerves emerge from deeper parts and become superficial. The affected part is usually hyperesthetic; occasionally, however, it is anesthetic, and may continue so for some time after an attack.

Reflex muscular contraction may be present in proportion to the intensity of sensory irritation. Vaso-motor symptoms manifest themselves in the flushing or blanching of the affected part and in increased secretions, as sweating.

Trophic disturbances may result in temporary or permanent changes. To the former belong the herpetic and urticarial eruptions, while the latter groups include change of color in, loss of, or overgrowth of the hair, various changes in the skin (as pigmentation and morphea, and even ulceration, though in the latter instance there is probably a more profound pathologic change than that which we regard as the cause of neuralgia). Unless the attacks are severe or prolonged, however, the general system seldom suffers.

Neuralgia may be divided into the following groups: neuralgia of the head, neck, trunk, upper and lower extremities; neuralgia of the genitals and rectal region; and visceral neuralgias.

TIC DOULOUREUX.

This is a neuralgia involving one or more of the branches of the fifth nerve. It varies greatly in character and intensity in different cases, and in its severest forms is one of the most terrible of all the diseases of the nervous system.

The *pathology* is doubtful. In those cases that have been subjected to surgical operation, excised portions of the nerves sometimes were normal and sometimes contained a moderate number of degenerated fibers. In other cases in which the Gasserian ganglion has been removed and examined, considerable sclerosis of the blood-vessels has been detected, alterations in the axis-cylinders of the nerves, and occasionally moderate changes in the ganglion cells. It is not known exactly how these lesions produce the symptoms, but it is probable that vascular alterations are exceedingly important.

The *etiology* is various. Neuropathic heredity appears to play an important part. It is more frequently a disease of late than of early life. Peripheral irritation is frequently found, and when removed often results in complete cure. Among the structures disease of which is a frequent cause of tic douloureux are the nose and the cavities entering into it, and the mouth. Lesions of the former structures comprise chronic irritations, spurs, occlusion of the nasal openings, and suppuration. In the latter, abscesses at the roots of the teeth, irritated pulp, and occasionally malpositions of the teeth, are among the exciting factors. It is possible that eye-strain may also be an exciting cause.

The *symptoms* may be variable in extent, duration, and intensity. In the mild form there is only an occasional paroxysm, limited to one of the branches of the nerve, such as the type that occurs in acute coryza. In the more severe form there may be repeated paroxysms, the intervals varying from a few minutes to several days, involving the whole side of the face, and causing, for the time being, complete prostration on the part of the patient. The pain is often radiating, or of a rending or boring character, and sometimes so severe as to cause nausea. It is often accompanied by certain vasomotor or secretory phenomena, such as flushing, perspiration, or excessive tear-production, and even in some cases more or less persistent edema of the skin. In some instances there may be more or less twitching of the facial muscles. The duration of the attacks varies greatly. The paroxysms may succeed each other frequently for long periods of time, or until the patient becomes insane or commits suicide; in other cases, after a few paroxysms, the attack passes off and may not return for months. In some instances the pain is persistent, and although in these cases it is rarely severe, the discomfort of the patient is sometimes greater. The painful points are: for the first branch, the supraorbital foramen; for the second, the infraorbital foramen; for the third, the mental foramen. Often the surrounding portions of the skin, particularly those where the periosteum is near the surface, are tender. If the disease continues for some time there may be trophic changes, such as the formation of ulcers, drying of the skin, and the appearance of gray hairs.

The *prognosis* depends largely upon the cause. In the so-called idiopathic cases it is exceedingly unfavorable.

The *treatment* consists first in the removal of the cause, if it can be found. The eyes, nose, and mouth should be carefully examined, and any source of irritation thoroughly removed. For the treatment of the paroxysms, if they are mild, the coal-tar analgesics may be employed. If severe, the only drug that is at all effective is morphia, which is preferably given hypodermically. Of course, in nearly all instances, if the disease is chronic, the patient becomes addicted to the use of this drug. Occasionally, the external application of a mixture of the ointments of opium and belladonna has proved of service. The general health of the patient should be improved if possible, and the disease treated expectantly for some time. If these measures prove unavailing recourse must be had to operation, which may consist of section of the involved branch, excision of a portion of the nerve, or, in the most severe cases, excision of the Gasserian ganglion. The latter is a serious operation and is followed by dangerous trophic lesions, and should be performed only as a last resort. Unfortunately, the results even of this do not appear to be permanent.

NEURALGIA OF THE NECK AND TRUNK.

The cervical branches of the dorsal and lumbar nerves are involved in this group.

1. *Cervico-occipital neuralgia*, occurring in the occipital and posterior parietal region, is apt to be quite severe, but when not due to spondylitis (the result of caries) or neoplasms the prognosis is fair. It is sometimes the result of direct pressure, as in carrying heavy loads on the neck and shoulders. The painful spot is found between the mastoid process and upper cervical vertebræ. Falling of the hair may also occur. This is much more apt to take place, however, when the occipitalis minor is involved, as it is said that the latter is generally a syphilitic neuralgia.

2. *Phrenic neuralgia* has been described, but is a rare condition.

The pain is in the lower anterior thoracic region, at the points of insertion of the diaphragm.

3. *Intercostal Neuralgia.*—The middle intercostal nerves are most liable to be affected, and generally on the left side. The posterior dorsal branches are seldom involved. When specially severe and persistent, intercostal neuralgia may be a symptom of disease of the cord or its membranes, aneurysm of the aorta, neoplasms, or disease of the vertebræ or ribs. Traumatism and cold also give rise to it. This form of neuralgia is most common in women, the painful spots being at the extremity and at the middle of the ribs. The pain is of a sharp, lancinating character and radiates along the nerve. It is intensified by all movements of the chest; hence the affected side is more or less fixed. Herpes may develop, but in such cases it is probable that a true neuritis exists.

4. *Mastodynia* is really a variety of intercostal neuralgia, and occurs almost solely among women. It is very painful, and gives rise to the development of tender "lumps" in the breast, simulating malignant disease. The paroxysms are often accompanied by vomiting.

5. *Lumbo-abdominal neuralgia* is not a common form. The pain is chiefly in the lumbar region, though the hypogastrium, genitals, and buttocks may also be involved.

NEURALGIA OF THE EXTREMITIES.

Cervico-brachial neuralgia occurs in the distribution of the four lower cervical nerves. When the condition is bilateral we should look for disease of the cord or membranes, for new growths, or for disease of the vertebræ. When unilateral, any of the causes already enumerated may be operative. The radial and ulnar nerves are more frequently affected than the median. The pain is most apt to be distributed along the whole course of the nerve, but painful points are found in the following situations—in the axilla; over the brachial plexus; on the shoulder, where the cutaneous branches of the circumflex nerve emerge through the deltoid muscle; about the middle of the outer surface of the upper arm; over the ulnar nerve; in the sulcus between the olecranon and epitrochlea; also near the wrist and at the bend of the elbow over the musculo-spiral nerve.

Femoral or *crural neuralgia* is a somewhat rare type that attacks the anterior surface of the thigh, the knee-joint, and the inner surface of the leg and foot.

Obturator neuralgia is distributed along the inner side of the thigh down to, and including, the knee-joint. This form is common in women subject to ovarian diseases.

Sciatica stands next to trigeminal neuralgia in the order of frequency, and is by far more common in men than women. In addition to the ordinary causes of neuralgia—exposure, compression, traumatism—the condition may be an early symptom of tabes or it may be due to constipation or hemorrhoids. The painful points are in the gluteal region and the popliteal space or malleolar region, though tenderness may be elicited along the whole course of the nerve. The pain is sharp and shooting, or more often of a tearing variety. Fine or coarse tremors or spasms may be present, together with some disturbance of sensation and loss of power. Herpes occasionally develops along the course of the nerve. This form of neuralgia is quite common, and is generally easy to recognize, but it may be simulated by hip-joint disease, psoas abscess, or lumbago.

Sciatica may also be caused by neuritis, the result of exposure or compression by pelvic growths, or by the fetal head during labor. In such cases there is, as a rule, slight fever, and the tenderness on pressure and the degree of pain are infinitely more severe than neuralgia. In severe cases the patient is bed-ridden, but in milder attacks he can be about; walking greatly increases the pain, however. It is slightly mitigated by relieving the tension on the nerve, by bending the knee, and walking on the toes. It is an obstinate condition, and relapses are common.

The *diagnosis* is generally not difficult. The distribution of the pain, the location of tender points, and the character of the gait suffice to prevent error, as a rule. The fulgurant pains of tabes, in the early stage of the disease, are frequently mistaken for sciatica. A rectal or vaginal examination should be made to determine whether it is a primary disease or secondary to some pelvic condition.

Neuralgia of the Genitalia and Rectum.—These varieties are not met with frequently. The former is sometimes a symptom of stone, prostatic disease, or stricture, and in women ovarian and uterine neuralgias are generally hysteric manifestations. Coccydynia, unless of traumatic origin, is almost solely found in women. The pain in the region of the coccyx is excruciating at times, and may even call for operation.

Visceral Neuralgia.—As implied by the name, these forms are neuralgias resident in the various viscera. They most frequently attack the stomach or bowel, and are recognized as colic. Other viscera may also be involved (liver, kidney).

Treatment of Neuralgia.—The first requisite in the treatment of neuralgia is to ascertain whether it is due to local or general causes. That of the former class may be caused by a cicatrix, neuroma, aneurysm, neoplasm, or by caries or traumatism; and the treatment must necessarily be directed toward the removal of the cause when possible. When the fault is a general one, the neuralgia may occur either as the immediate result of the systemic disease or remotely, as the result of the altered blood-state (anemia). This is particularly well illustrated by an attack of malaria, in which it is obvious that success can only be obtained by attention to the underlying cause. It is sometimes necessary to use an analgesic, of which morphin is certainly the best. Its therapeutic value is most decided when the drug is given hypodermically, and if injected directly over the track of the painful nerve (*e. g.* supraorbital branch of the fifth), it not only affords immediate relief, but also obviates recurrences of the painful paroxysms in many instances. It is, however, scarcely necessary to urge the exercise of caution, for the morphin-habit is readily formed in these cases. The following may also be used; antipyrin, phenacetin, codein, veratrum viride, aconite, also counter-irritants and vesicants, including the galvanic current. The general tone of the system must be attended to, bad habits prohibited, the statė of the bowels regulated, and the eyes examined and corrected for errors of refraction. Rest is a valuable adjunct to any form of treatment. In neuralgia of the upper extremities and in sciatica I have often obtained good results from putting the limb in splints.

Sciatica is often very intractable. If it fails to yield to the salicylates, counter-irritation, leeches, and the rest-treatment of Weir Mitchell may be used, or nerve-stretching, either by flexing the thigh upon the abdomen or, as a last resort, by cutting down upon the nerve itself. Other nerves are sometimes subjected to this method of treatment also, but less frequently than the sciatic. Absolute rest in bed, with the limb kept perfectly still by means of sand-bags or a long splint, always gives relief, and in some cases seems to cure. Alternating hot and cold douches also give great relief in some instances. Deep injections of thein, ether, or chloroform are sometimes used, and even distilled water may give relief when injected into the nerve. The use of guaiacol (♏j–ij—0.066–0.1332) in association with chloroform (♏x—0.666) by this method has yielded very encouraging results in my hands.[1]

DISEASES OF THE CRANIAL NERVES.

OLFACTORY NERVE.

THE following morbid conditions have been described in connection with the sense of smell:

(*a*) *Hyperosmia* or *Olfactory Hyperesthesia.*—The sense of smell is abnormally acute, so that objects, and even persons, can be recognized by this means. It occurs in hysteria and insanity.

(*b*) *Parosmia* (perverted sense of smell) may occur for one or for many odors, and is often associated with an obtunding of the normal sense.

(*c*) *Subjective sensations* of smell are due to the same causes as the above. An olfactory aura may precede an attack of epilepsy. Olfactory hallucinations occur occasionally in the insane.

(*d*) *Anosmia* or *olfactory anesthesia* (loss of the sense of smell) may be caused by—(1) injury to the peripheral filament by local disease of the nasal mucous membrane. (2) Injury to the nerve-trunk or bulb, bone-disease, and meningitis. Anosmia may occur during locomotor ataxia. Pungent and powerful odors have been said to cause loss of the sense of smell, due to excessive stimulation. There may be a congenital absence of the olfactory nerves. (3) Centric lesions, as tumors in the anterior part of the temporo-sphenoid lobe. Hughlings Jackson has reported cases of unilateral anosmia associated with aphasia, believed to be due to simultaneous involvement of the outer limb of the olfactory nerve as it passes the island of Reil to reach the center and Broca's region. Opposite unilateral anosmia has been described, due to a lesion in the posterior part of the internal capsule.

In testing the sense of smell it is advisable to use aromatic oils, as they only stimulate the olfactory nerve, while ammonia and such strong substances also stimulate the fifth nerve. It is obviously necessary to make a rhinoscopic examination.

Treatment is generally unsatisfactory, though the cause must be removed when possible. When the disturbance is due to some general condition, as hysteria, it may of course be disregarded, as it will improve with the disease.

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[1] "The External and Internal Use of Guaiacol," *Therapeutic Gazette*, Mar. 15, 1895.

DISEASES OF THE RETINA, OPTIC NERVE AND TRACT.

The Retina.—Hemorrhage into the retina may be venous or arterial, single or multiple, monocular or binocular. It may be part of a general vascular change; occasionally it occurs during parturition, but more often at the menopause; it may be an indication of renal trouble or of some primary or symptomatic anemia, as in leukocythemia, pernicious anemia, or malaria. Hemorrhage is prone to occur also in depraved nutritional conditions, in purpura, and in scurvy.

More or less complete loss of vision develops in these cases, either suddenly or gradually. If the hemorrhage is superficial, the eye-ground is red and swollen; if deeper, the blood escapes between the fibers of the retina, spreads them out, and assumes a flame-shaped appearance.

Retinitis.—Three forms of this condition are commonly described—(1) albuminuric, (2) syphilitic, and (3) pigmentary.

(1) *Albuminuric retinitis* is probably not a distinct affection, but part of a general fibro-vascular change associated with nephritis. The failure of vision may precede the advent of albuminuria, but more often the two conditions are coincident. It occurs in chronic nephritis, especially in the interstitial variety.

The retinal changes, according to Gowers, are either *hemorrhagic* or *degenerative*. In the former the arterial blood occupying the interstices between the fibers assumes a striated or feathery aspect, while in the degenerative form white patches of fatty degeneration or deposits of cholesterin are dotted over the fundus; they may also be grouped about the macula lutea, or around the disk. Occasionally the latter appears swollen, owing to the effusion of serum into the fiber-layer.

(2) *Syphilitic retinitis* generally occurs in the later stages of acquired syphilis, and particularly in neglected cases. Failure of vision directs attention to the eye-ground, which is found to have either scattered or uniformly distributed whitish or slightly opalescent filmy patches upon it. The vitreous may be turbid also. Retinitis is far less common than choroiditis or chorio-retinitis.

(3) *Pigmentary retinitis* is essentially a chronic process, usually attacking young adults, and, as a rule, more than one member of a family. It may also occur in inherited syphilis and in low grades of vitality. The affected parts receive a deposit of pigment which specially follows the course of the main arteries. At the same time a circumferential annulus of pigment forms. This gradually encroaches more and more upon the disk, until finally atrophy ensues.

Among retinal affections occur also—

(*a*) **Toxic Amblyopia.**—This is due, as a rule, to tobacco or alcohol, and more rarely to certain drugs or lead-poisoning. Failure of vision is gradual and progressive, though it rarely reaches absolute blindness. The center of the field is chiefly affected, and a central scotoma for red and green exists; this is said to be caused by a chronic neuritis beginning in the fibers that are distributed to the macula lutea.

(*b*) **Hemeralopia,** or *day-blindness,* may either be functional or a symptom of some retinal affection—*e. g.* hyperesthesia or albinism, or the result of central cataract. Objects can either not be seen at all or only indistinctly during the day or in a strong artificial light; but at night vision is excellent.

(*c*) **Nyctalopia,** or *night-blindness.* In this condition vision may be normal during the day or in a strong artificial light, but after nightfall or in a darkened room objects can be seen only with difficulty or not at all. It is usually associated with syphilitic retinitis.

Optic Nerve.—Three distinct pathologic conditions of the nerve exist —viz. (1) Perineuritis, (2) Choked disk, and (3) Neuritis. They may merge into one another, and after lasting some time may lead to (4) Atrophy and complete blindness.

(1) *Perineuritis* is met with in meningitis, and was suggested by Bouchut as a valuable diagnostic sign in obscure cases. It is also found in certain cases of insolation, and may occur in any fever in which impairment of vision is a sequel. The sheath of the nerve is supplied by the blood-vessels of the pia, while the nerve itself derives its blood-supply from the anterior cerebral artery; therefore in perineuritis, in the early stages at least, the disk remains normal, but is surrounded by a zone of congestion and inflammation. If the action has been severe or prolonged, either the direct pressure or that due to the contraction of the inflammatory material causes partial atrophy of the nerve and consequent interference with the vision.

(2) *Choked disk* is almost always bilateral, and occurs in cases of intracranial granulomata or tumors, also in Bright's disease and syphilis. The disk is at first merely congested; soon, however, both the arterial and venous circulations are interfered with, and especially the return venous flow; then exudation of serum takes place. Sometimes secondary inflammatory changes follow. In the early stages vision is not impaired, but as the exudative elements contract, the interference with the circulation becomes more exaggerated, and in time atrophy of the disk supervenes. Should the process be arrested, the retinal dropsy subsides, and it will then be seen that the vessels are thickened and tortuous, and stand out in relief near the margins of the disk. White patches of atrophy may be scattered over the fundus. When the inflammation and a dropsical effusion into the disk exist simultaneously, it is difficult or impossible to differentiate the condition from primary or descending optic neuritis.

(3) *Neuritis.*—The optic nerve derives its blood-supply from the anterior cerebral artery. Evidently, then, in cerebral hyperemia (arterial) from any cause we have an increased injection of the disk, but no venous engorgement; hence there is no dropsy and no tortuosity of the veins. Inflammation may begin in the disk or descend from above, giving rise to plastic deposits on the retina. Sight is early interfered with, owing to involvement of the conducting fibers, which atrophy in time unless the condition ceases. Then the disk appears white, and the vessels show upon it as thin filaments. This condition is met with in syphilis, Bright's disease, intracranial tumors, and rarely in anemia and lead-poisoning; it may be an advanced stage of perineuritis or choked disk.

(4) *Optic Atrophy.*—This may occur as an hereditary affection known

as *Leber's disease*, which chiefly attacks young males, or it may occur during the course of locomotor ataxia, certain toxemias, and diabetes. It may also be brought about either by conditions that produce brain-disease or as a result of the cerebral disease itself.

In any case there is alteration of the field of vision, color-perception is abnormal, and there is more or less dimness of sight. In the hereditary form the disk is less white than in the other, and the vessels are almost normal in appearance (Fig. 71).

The Optic Tract.—The lesions of the optic tract are important rather on account of their situation than their nature. They may exist without corresponding changes in the retina, although when they have lasted for a long time there is usually some consecutive atrophy resulting from a descending degeneration of the optic nerves. Lesions of the chiasm usually affect the decussating fibers, causing blindness of the nasal halves of the retina, and, in consequence, temporal hemianopsia. This condition occurs in basal tumors especially of the hypophysis, and has therefore been observed in acromegaly, in tuberculous basal meningitis, and in hydrocephalus. Lesions of either optic tract, if complete, causes homonymous bilateral hemianopsia; if incomplete, there is irregular disturbance of the visual field, sometimes bilateral, sometimes unilateral. It may be involved in hemorrhage, tumors, softening or basilar meningitis; ordinarily other structures are also involved, giving rise to symptoms of focal disease. Lesions anterior to the anterior corpora quadrigemina usually cause more or less destruction of some of the other cranial nerves, with the production of ocular palsies, or disturbances of the other special senses, or anesthesiæ or neuralgias of the face. A very valuable sign, that, however, cannot always be elicited, is the failure of the pupil to contract when light is thrown upon the blind half of the retina. This is explained by supposing that the pupillary reflex center is situated in the anterior corpus quadrigeminus. If the lesions affect the optic thalamus or the internal capsule, hemiplegia and hemianesthesia are also often present or may form the most important symptoms. Lesions posterior to the anterior corpora quadrigemina produce hemianopsia without disturbance of the pupillary reflex. These lesions are divided into two groups, the cortical and the subcortical and they may be of two varieties, either irritative or paralytic. The irritative lesions give rise to hallucinations of sight, which may vary from the scotomata of migraine to most complex visions. Paralytic lesions ordinarily lead to hemianopsia. Occasionally curious symptoms are produced, the visual field being sometimes irregular, whilst at others only certain elements of sight are affected, cases having been reported in which the hemianopsia only involved the recognition of colors, not of form. In all these cases the pupillary reflexes are not affected. Bilateral lesions do not always lead to total blindness: sometimes the macula lutea escapes and the patient is able to see only by direct fixation. Occasionally a single lesion will produce total blindness in one eye, but this is rare, and no satisfactory explanation has been found to account for it. Cortical lesions are those involving the occipital lobe. The center of visual perception appears to be in the cuneus and calcarine fissure; if this is destroyed, blindness occurs. The center for the recognition of the object seen is apparently upon the convex surface of the occipital lobe, probably in the second

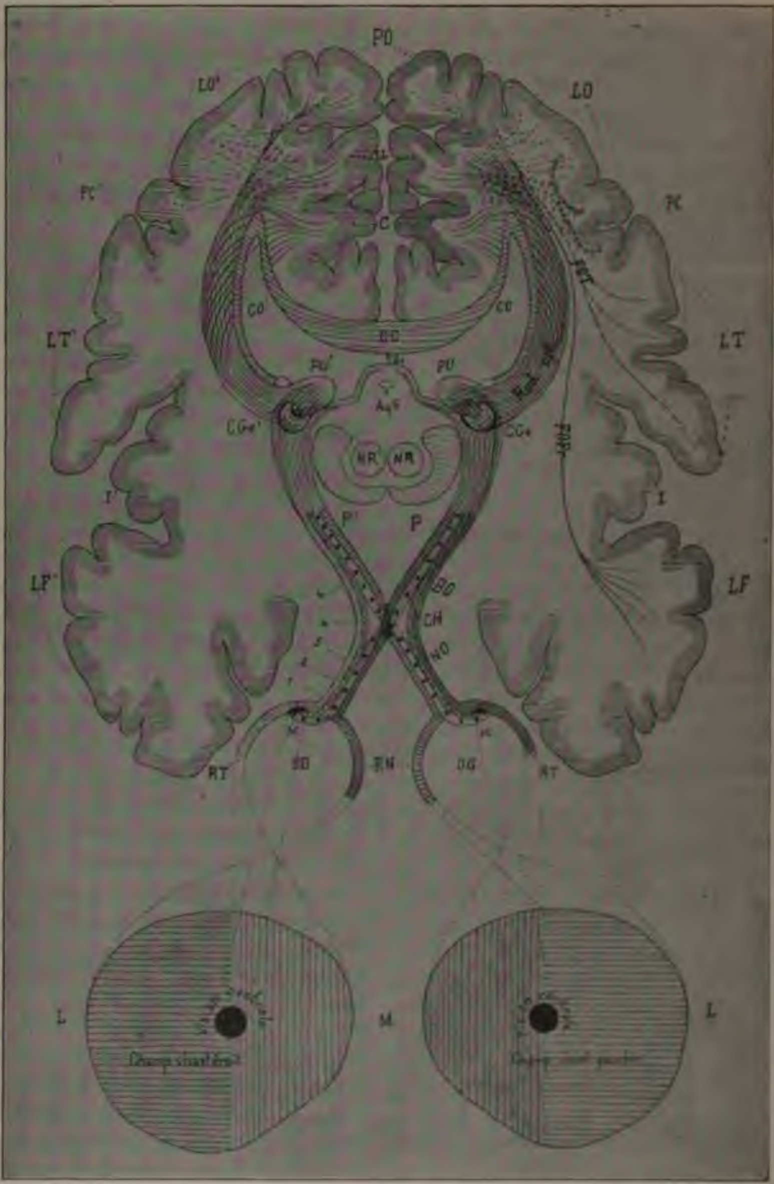

Fig. 71.—Diagram of the visual apparatus (after Vialet): *LO*, *LO'*, occipital lobes; *C*, cuneus; *Rad. opt.*, optic radiation; *TQa*, anterior corpus quadrigeminus; *PU*, *PU'*, pulvinar; *CGe*, external geniculate ganglion; *BO*, optic tract; *CH*, optic chiasm; *NO*, optic nerve; *OD*, right eye; *OG*, left eye; *RN*, nasal half of retina (supplied by the opposite hemisphere); *RT*, temporal half of the retina (supplied by the homolateral hemisphere); *M*, macula lutea. A total transverse lesion at 1, 2, or 3 would cause total blindness of the right eye. A lesion at 4, destroying the central part of the chiasm, would cause blindness of the nasal halves of the retinæ, and therefore bitemporal hemianopsia. A lesion at 5 would cause blindness of the right halves of the retinæ, and therefore left homonymous hemianopsia. The pupillary reflex would be lost in the affected half of the eye in all these cases. A lesion in the optic radiation would cause symptoms similar to those of the corresponding optic tract, excepting that the pupillary reflex would be preserved. Lesions of the cortex cause various disturbances of vision according to the part affected.

and third convolutions, but it may extend also into the temporal lobe. When this is destroyed the patient can see objects, but fails to recognize them; this is called *mind-blindness;* if total, this is the result of a bilateral lesion. Hemianopsia is very frequently merely a temporary symptom, and as such it may occur in uremia, apoplexy, migraine, and certain intoxications, especially that of lead. It may also occur in brain-tumor, and disappear if the pressure is relieved, as by trephining. It is a permanent symptom only when the visual tract has been involved by some destructive lesion. If the patient is perfectly conscious and intelligent, it is not difficult to recognize it; nevertheless, its presence can often be detected in young children and in those who are only partially conscious or unable to speak. This can be accomplished by taking a bright object, placing it behind the head, and then bringing it forward slowly, first on one side and then on the other. It will then be noted that the patient perceives it on the hemianopsic side only when it has been brought to the middle line, whilst when moved on the other side the eyes will turn toward it when it is still a considerable distance from this point. Another method is to bring a blunt object (a wisp of cotton) very nearly in contact with the cornea, first on the one and then on the other side of the median line. The palpebral reflex will occur upon the normal side whilst the object is still some distance away; on the blind side only when it has come in contact with the conjunctiva (see Fig. 71).

DISEASES OF THE MOTOR NERVES OF THE EYEBALL (THIRD, FOURTH, AND SIXTH).

The extrinsic ocular muscles are supplied by these three nerves, while the intrinsic are supplied by the third and the sympathetic.

I. **The motor oculi,** or **third nerve,** is purely motor, and supplies all the muscles of the eye except the superior oblique and external rectus, and controls in part also the ciliary muscle and the sphincter of the iris. Its apparent origin is from the inner side of the crus cerebri just anterior to the pons. It can be traced through the crus, however, to its deep origin in a nucleus beneath the corpora quadrigemina, situated in the floor of the aqueduct of Sylvius. Above the crus it pierces the dura, passes between the two clinoid processes of the sphenoid bone, along the outer wall of the cavernous sinus, where it receives some filaments from the cavernous plexus of the sympathetic; it then divides into two branches that enter the orbit through the sphenoid fissure. The superior and smaller division supplies the superior rectus and levator palpebræ superioris, while the inferior and larger branch subdivides into three portions, one going to the internal rectus, another to the inferior rectus, and the third to the inferior oblique.

Lesions of the third nerve result in (1) spasm or (2) paralysis.

Spasm rarely if ever occurs in all the muscles simultaneously. Any muscle may be affected, but the internal rectus and levator palpebræ are specially liable. The condition is met with in meningitis, hypermetropia, and hysteria; also in nystagmus, in which the spasm is clonic and bilateral; it also occurs in albinism, occasionally in coal-miners, or it may be congenital.

When the levator palpebræ is affected (*lagophthalmus*) inability to close the eye results. Stimulation of the center or nerve may cause contraction of the pupil (*myosis*), as occurs in locomotor ataxia. The same result is brought about by paralysis of the sympathetic.

Paralysis.—The nerve may be involved in any part of its course by inflammatory deposits or tumors, or the nucleus may be diseased. In the latter case there is usually ophthalmoplegia.

Relapsing and recurring palsy are two varieties. The first occurs chiefly in syphilitic subjects. One nerve becomes affected and partially recovers; the other one then becomes paralyzed, and partially recovers, relapses, and so on. The internal muscles may be involved.

Recurring or periodic palsy, the *migraine ophthalmique* of Charcot, is a rare form. It occurs in both sexes, but women are especially susceptible. It may begin in infancy and recur at intervals for years, the attacks being periodic, lasting a few days to six or eight weeks, and ending in complete recovery. They may be precipitated by some emotional disturbance, by menstruation, or by exhaustion. Their exact nature is not understood, but they resemble migraine in that there is severe headache or pain, usually over one eye, and in their association with vomiting.

Generally paralysis of the extra-ocular muscles is partial, and the symptoms will vary according to the muscles affected. When they are all involved there are ptosis, divergent strabismus, diplopia, and contracted pupil, with loss of the light-reflex and accommodation.

Intra-ocular Paralysis.—(*a*) **Cycloplegia,** or ciliary muscle-paralysis, gives rise to a loss of the power of accommodation, so that "far-sight" is good, while "near-sight" is blurred and indistinct. This can be corrected by a convex glass. Bilateral cycloplegia is usually due to a nuclear lesion. It occurs sometimes in diphtheria and in tabes dorsalis.

(*b*) **Iridoplegia.**—The pupil may be dilated (*mydriasis*) from palsy of the sphincter or spasm of the dilator, or it may be contracted (*myosis*) from the antithesis of the above.

The iris has three actions—two reflex and one associated: First, a reflex contraction of the sphincter on exposure of the eye to light; second, a reflex dilatation of the radiating fibers on stimulation of some cutaneous nerve; and, third, a contraction on accommodation, usually, but not necessarily, associated with convergence (Gowers).

First, light-reflex iridoplegia. The iris reflex is lost in locomotor ataxia, in general paresis, and occasionally in disease of the peripheral portion of the third nerve, and sometimes also in syphilis. Accommodation and convergence are, however, usually preserved (*Argyll-Robertson pupil*). When these also are lost the condition occurs to which Jonathan Hutchinson gave the name *ophthalmoplegia interna*.

In testing this reflex care must be taken to avoid the contraction of accommodation. The patient should look at a remote part of the room; then a light is brought suddenly in front of, and three or four feet distant from, the eye. One eye should be examined at a time, the other being covered, but not closed.

Second, skin-reflex iridoplegia. Normally, painful stimulation of the skin of the neck causes reflex dilatation of the pupil, the afferent impulse being carried along the sympathetic. In locomotor ataxia myosis often exists. In such cases Erb showed that the skin-reflex was lost (*spinal myosis*).

Third, accommodation iridoplegia, in which the power of accommodation is lost. The pupil does not become smaller when looking at near objects. Westphal and Piltz have recently discovered independently, that in certain pathological conditions the pupil contracts strongly upon closure, or attempted closure against resistance, of the eyelids. This reflex occurs most constantly in general paresis. Its exact significance is not known.

II. **The fourth nerve,** or **patheticus,** the smallest cranial nerve, supplies the superior oblique muscle. Its superficial origin is to the outer side of the crus cerebri, just in front of the pons. The fibers can be traced backward to the valve of Vieussens, in the substance of which it decussates with its fellow. Its deep origin is in a nucleus in the floor of the aqueduct of Sylvius, immediately behind and in close connection with the third-nerve nucleus. After piercing the dura mater the nerve runs along the outer wall of the cavernous sinus and enters the orbit through the sphenoid fissure. Since the superior oblique muscle directs the eyeball downward and rotates it, paralysis causes defective downward and inward movements, and consequent diplopia with inclination of the head forward and to the sound side. When occurring alone it is probably due to a nuclear lesion.

III. **The sixth nerve,** or **abducens,** has its deep origin in the floor of the fourth ventricle in close proximity to the seventh-nerve nucleus. Its superficial origin is from the lower part of the pons, in the groove between it and the medulla. Emerging, it pierces the dura, runs along the cavernous sinus, and enters the orbit through the sphenoid fissure to supply the external rectus. Owing to its long course, this nerve is specially liable to injury, usually from pressure due to tumors or from syphilitic or other forms of meningitis. Paralysis of the muscle causes convergent strabismus, owing to an inability to rotate the eye outward and consequent diplopia. In nuclear lesions the external rectus of the same side and the internal rectus of the opposite side are paralyzed, conjugate deviation resulting, the eyes being directed away from the side of the lesion. This is due to the fact that the sixth nerve gives off a twig that runs to that region of the opposite third-nerve nucleus governing the internal rectus.

This muscle is not wholly controlled by the sixth nerve, however, for in nuclear lesions of the latter no degenerated fibers are found in the third nerve; and, further, when the eye with the paralyzed external rectus is covered the opposite internal rectus will act, though less readily than normally.

General Symptomatology of Paralysis of the Eye-muscles.—Loss of power in the ocular muscles is indicated by five kinds of symptoms (Gowers): (1) *Limitation of Movement.*—The amount of limitation in the movement of the eyeball is in direct ratio to the degree of palsy. In complete palsy the globe is ultimately fixed, owing to contraction of the unopposed muscle. In partial paralysis, as the limit of movement is approached the motion is often jerky (*paralytic nystagmus*).

(2) *Strabismus.*—Owing to defective movement the axes of the eyes

do not correspond. "The deviation of the axis of the paralyzed eye from parallelism with that of the sound eye is termed the primary deviation."

(3) *Secondary Deviation.*—"If the sound eye is prevented from seeing the object, and the patient looks at this (is made to 'fix' it) only with the affected eye, the sound eye is moved still farther in that direction, and hence the deviation of the visual axes is increased. This is called the 'secondary deviation,' and depends on the fact that two muscles normally acting in unison are equally stimulated (innervated) for any given movement. When one is weak, the amount of nerve-force employed to move the sound eye acts equally on the impaired eye, and hence the over-action. In paralytic strabismus fixation with the sound eye shows the primary deviation, while fixation with the affected eye reveals secondary deviation. In ordinary strabismus due to spasm this does not hold good; it matters not which eye is used, deviation remains the same."

(4) *Erroneous Projection.*—We judge of our relation to surrounding objects by the position of the eyeball as indicated to us by the degree of stimulation necessarily brought to bear on the ocular muscles. When one of these muscles is weak, the additional stimulation (innervation) necessary to move it in fixing an object impresses us with the idea that it is really farther away than is actually the case, and in attempting to touch it the finger goes beyond. This erroneous projection, or interference of visual sense-impressions, causes a disturbance of equilibrium and gives rise to vertigo, which has been named "ocular vertigo."

(5) *Double Vision.*—This is not due alone to a difference in the axis of vision, causing images on non-corresponding portions of the retina, but also to the erroneous projection. "If the patient looks with both eyes, the field of the unaffected eye, being normally projected, does not correspond with the field of the affected eye; the images formed in the two eyes are mentally referred to different positions; objects are seen double" (Gowers). The "true image" is that one formed in the sound eye, while the retina of the affected eye receives the "false image."

Homonymous or *simple diplopia* is that in which the false image appears on the "same side of the other as the eye by which it is seen." This is due to paralysis of an abductor muscle—*convergent strabismus*. *Crossed diplopia* occurs in divergent strabismus, the result of paralysis of an adductor. The false image appears to be on the other side of the real object—*i. e.* toward the sound eye.

Gowers' mnemonic is, "When the visual lines (prolonged ocular axes) cross, the diplopia is not crossed."

Ophthalmoplegia, a paralytic condition of the eye-muscles, may be partial or complete. Either the internal or the external muscles may be involved, constituting *ophthalmoplegia interna* or *externa*, and, when both are affected, total ophthalmoplegia. The lesions may be nuclear or peripheral. Pressure due to neoplasms, gummata, aneurysms, or basilar meningitis may produce it, or it may follow diphtheria. It also occurs in general paralysis, progressive muscular atrophy, and locomotor ataxia. It may be (*a*) of sudden onset, due to some vascular disturbance; (*b*) acute—the *polio-encephalitis superior* of Wernicke—developing in a few days or weeks; or (*c*) chronic. In the latter case symptoms of bulbar palsy are apt to be present also. Von Graefe has described a form of bulbar palsy limited to the ocular nuclei under the name of *progressive ophthalmoplegia.*

The **symptoms** vary necessarily according to the muscles involved.

The **treatment** consists in the removal of the cause when possible. In inflammatory cases counter-irritation is employed by blisters placed on the temples, behind the ears, or at the occiput, or by leeches. Internally, the salicylates, mercury, iodids, and general tonics are useful. Rarely a case will recover spontaneously. Electricity is probably of little value. The diplopia, unless it can be obviated by a suitable lens, should be met by means of an opaque glass.

DISEASES OF THE FIFTH NERVE.

The trigeminus nerve has an extensive origin from the floor of the fourth ventricle. It supplies with sensation the whole region innervated by all the other cranial nerves except the first and second. It resembles a spinal nerve in that it has two roots, a motor and sensory, and on the latter a ganglion (*Gasserian*). From the latter arise three sensory branches—viz. the ophthalmic, superior maxillary, and inferior maxillary. A motor root joins the last named, the largest branch of the fifth nerve.

Morbid conditions of the fifth nerve cause sensory, motor, or gustatory symptoms. The lesion may be—(1) Pontine hemorrhage, softening, sclerosis, or tumor. (2) Disease or injury at the base of the brain—*e. g.* meningitis, gumma or other tumor, caries of bone. (3) Disease or injury of the branches, as neuritis, pressure due to aneurysm of the internal carotid or to a tumor in the pituitary or spheno-maxillary region, orbital cellulitis, and punctured wounds of the mouth and nose. (4) Rarely fracture of the skull.

Symptoms.—**Sensory Portion.**—In the irritative stage the chief feature is pain; this may be shooting, boring, or burning in character. Tenderness along the course of the nerve and hyperesthesia may also exist. Later anesthesia develops in the mucous membrane of the nose, mouth, lips, tongue, and, in some cases, of the hard and soft palate also. Muscular movements are slower than normally, due to sensory interference.

The secretions are often increased, though at first they are lessened; hence the anosmia, due to dryness of the nasal mucosa. Loss of sense of taste may also occur. Other trophic changes are—inflammation and ulceration of the gums, looseness of the teeth, and inflammation of the eye. Corneal opacities, ulceration, sometimes perforation, and finally complete destruction of the eye—neuro-paralytic ophthalmia—are noted. This is especially apt to occur when the Gasserian ganglion is involved. Painful and intractable herpes may develop. Hemifacial atrophy may result from disease of the fifth nerve (Mendel).

Motor Portion.—*Paralysis.*—Partial or complete inhibition of the movement of the muscles in the region supplied—*i. e.*, those of the jaw, the masseter, temporal, pterygoid, mylo-hyoid, and the posterior belly of the digastric. The degree of palsy can be ascertained by placing a finger on each masseter or temporal muscle while the patient alternately opens and forcibly closes the mouth. In external pterygoid paralysis movement toward the sound side is impossible, and on depression of the lower jaw it deviates toward the affected side. Ultimately wasting of the muscles, with deformity, takes place.

Spasm (the so-called "masticatory spasm" of Romberg) may be tonic or clonic. In tonic spasm—trismus or lockjaw—the jaw is firmly set and the muscles are hard, rigid, and sometimes painful. This occurs in tetanus, in certain cases of tetany and hysteria, in caries of the teeth, occasionally after exposure, and in irritative centric or peripheral lesions. Clonic spasm is more or less continuous or intermittent. The former consists of short, quick, vertical or rarely lateral movements (*e. g.*, gnashing of the teeth), usually associated with some other condition, as paralysis agitans, general convulsions, and the like, or it may exist alone, especially in women late in life. The intermittent form is rare and occasionally occurs in chorea. Contractions are single, forcible, and are separated by some little time. The tongue and cheeks may be bitten in the attack.

Gustatory Portion.—*Symptoms* referable to this portion are not always present in disease of the fifth nerve. There may be a loss of taste without sensory disturbance, or *vice versâ*, or both may exist contemporaneously. Lesions of the nerve-root or middle-ear disease may cause it, but pontine lesions, as a rule, do not. A perverted sense of taste—*parageusia*—may be present in hysteria and insanity. Increased sensitiveness—*hypergeusia*—and subjective sensations of taste may result from irritative lesions, and the latter may precede an attack of epilepsy (as an aura).

The **diagnosis** is not difficult as a rule. Anesthesia in the area supplied by the nerve, with loss of taste, is fairly conclusive. Spasm may be simulated in cases of rheumatism or rheumatoid arthritis involving the temporo-maxillary articulation.

Treatment.—The underlying cause should be attacked when possible, and mercury, the iodids, and the salicylates should be administered in specific cases and in those due to exposure. Analgesics, and even opiates, may be necessary. Sometimes vigorous counter-irritation is of value. Attention must be paid to the condition of the general system. The battery may be tried, preferably with the faradic current, or by means of electricity short and extremely rapid blows may be made over the nerve.

DISEASES OF THE SEVENTH OR FACIAL NERVE.

The nucleus of this nerve in the floor of the fourth ventricle is in relation with those of the sixth, eighth, and twelfth nerves. Like the spinal nerves, it has an upper and lower neuron or motor segment, the former extending from the cortical center in the lower Rolandic region to the nucleus, while the latter runs from the nucleus to the periphery. Lesions may involve any part of the tract, producing either spasm or paralysis.

Spasm.—This may be idiopathic or organic, and either general or partial, affecting only the orbicularis palpebrarum (*blepharospasm*). It is sometimes called *tic convulsif* or mimic spasm.

Pathology.—The commonest cause appears to be some psychic disturbance. Next in frequency are peripheral irritations, and particularly those that involve the trigeminus, as carious teeth, conjunctivitis, or some nasal irritation. Less frequently irritation in some other part of the body, as intestinal parasites or uterine disease, may be the exciting cause. Finally, there may be lesions in any part of the motor tract supplying the face, either in the cortex (meningeal tumor, exostoses, or focal softening), in the facial nucleus in the medulla, or along the course of the facial nerve (aneurysm or atheroma of the vertebral artery). Morbid changes in the nerve itself or in the muscles have not been observed.

Etiology.—As in other functional diseases, neuropathic heredity plays an important part. Sometimes, indeed, tic convulsif has existed in a parent or has occurred in several children of the same family. Emotional disturbances have frequently been the exciting cause; besides these, there are the various reflex irritations. The organic causes are irritative lesions, situated in some portion of the facial motor tract. According to Gowers, the functional form occurs only in adult life. It occasionally follows facial palsy.

The **symptoms** of the disease include, first, the *spasm :* this is usually a sudden clonic convulsion of the muscles of one side of the face, with closure of the eyelids and retraction of the angle of the mouth. Rarely there are associated movements of the palate and eyeballs. The spasms may be single or they may occur in groups frequently repeated, or recur constantly at more or less irregular intervals. Less frequently the contraction may be tonic in character, lasting several seconds or even minutes. These forms are frequently associated with clonic spasms. Ordinarily the spasm is painless, though there are certain sensitive points, as where the branches of the trigeminus issue from the skull, and particularly over the supraorbital foramen. Sometimes there is also *tinnitus aurium. Taste* and the muscles of the palate are rarely affected. Occasionally edema of the face, especially in the orbital region, occurs. The immediate exciting cause of an attack may be fatigue or excitement, or it may occur as an associated movement, as in a case that I observed, in which spasm always accompanied the beginning of speaking.

The **diagnosis** is usually easy. It may be occasionally confounded with *chorea*, especially when the latter is chiefly localized in the face, or with *athetosis* due to infantile brain-lesions. Recognition of the cause is often very difficult, and a careful examination of the whole body should be made for any possible source of irritation.

The **prognosis** is extremely unfavorable for cure, since only in cases of recent occurrence, and with a distinct source of peripheral irritation, is permanent recovery likely. Ordinarily, the disease, even if commencing in a mild form, gradually progresses to the most severe type, the intervals between the attacks become shorter, and the attacks themselves more severe.

The **treatment** consists in the removal of any source of irritation and the application of electricity, particularly the mild galvanic currents, with the anode over the sensitive points. Operative interference, as stretching the facial nerve (which rarely produces any result unless paralysis ensues) or cutting the tendons of the facial muscles, may be tried;

and finally, use of antispasmodics, as conium, gelsemium, morphin, and the bromids, may give temporary, but rarely permanent, relief.

Paralysis (Bell's Palsy).—Depending on the seat of the lesion, we have—(*a*) supra-nuclear, (*b*) nuclear, and (*c*) infra-nuclear palsy. The following table presents the general differences between upper and lower neuron palsy:

Supra-nuclear Paralysis.	Nuclear and Infra-nuclear Paralysis.
The upper part of the face is not affected, the muscles of the angle of the mouth being chiefly concerned.	All parts of the face involved, including the orbicularis and frontalis.
Voluntary movements are more impaired than the emotional.	Voluntary and emotional movements equally affected.
All reflex movements are normal.	All reflex movements are lost.
Electric reaction is normal, or only slightly impaired to both galvanic and faradic currents.	Reactions of degeneration are present.
There is no wasting.	Wasting is present.

(*a*) *Supra-nuclear paralysis* is generally associated with hemiplegia, the palsy of face and limbs being on the same side—*i. e.* opposite the lesion, which may consist of a hemorrhage, tumor, abscess, softening, or which may be the result of injury, and may be situated in the cortex, corona radiata, or the internal capsule. When the cortical face-center is alone involved, the limbs escape (*monoplegia facialis*). This form is rare.

(*b*) *Nuclear paralysis* is due to hemorrhage, tumor, or softening at the site of the nucleus in the medulla. It may also result from an attack of diphtheria, and very rarely occurs in cases of antero-poliomyelitis. As already noted, the symptoms are similar to those of infra-nuclear paralysis.

(*c*) *Infra-nuclear paralysis* is caused by pressure on the nerve at the base of the brain by tumors, meningitis, aneurysm, or hemorrhage. In the Fallopian canal the nerve may be damaged by bone-disease or some form of otitis. This is the seat, too, of the so-called "rheumatic neuritis," the result of exposure.

Fracture of the base of the skull or injury to the nerve as it emerges from the stylo-mastoid foramen may result in facial palsy. *Diplegia facialis* is rare, but may be caused by a single lesion in the pons, where the facial paths cross, or by two lesions, one on either side. The causes enumerated above, when bilateral, beget double facial paralysis.

Lesions in the lower part of the pons may result in crossed hemiplegia, the fibers being involved in their course between the nucleus and the point of emergence of the nerve, the side of the pons. The face will be paralyzed on the same side as the lesion, since this latter is below the decussation of the facial tracts, and involves the outgoing nerve, together with opposite hemiplegia. In alternate or crossed hemiplegia the facial palsy is of the infra-nuclear type, while in ordinary hemiplegia the supra-nuclear type is met with. Certain symptoms of nerve-irritation may precede the actual palsy or may be concomitant, such as slight pain and tenderness, some swelling in front of the ear, muscular twitching, and occasionally vertigo.

Symptoms.—The affected side is immobile and expressionless, and the normal lines are diminished or abolished. This is seen most markedly in those above middle life. The eye cannot be closed, owing to drooping of the lower lid, and, as the tears are not directed into their proper channel, the eye waters. Voluntary and emotional movements are lost. Whistling and smoking are performed with difficulty, if at all; if the cheeks are puffed out, air escapes upon the paralyzed side; food collects between the teeth and cheek, owing to paralysis of the buccinator; in drinking the patient inclines the head to the sound side to prevent escape of the liquid from the corner of the mouth. The dilator naris is paralyzed; hence sniffing is interfered with, and the sense of smell is diminished on that side.

When the tongue is protruded it seems to be drawn toward the palsied side. This is not the case, however, the effect being due to contraction of the unopposed muscles on the sound side. All reflex movements are lost. The palate is not affected, and sensation is not impaired. When the nerve is involved between the *intumescentia gangliformis* and the origin of the chorda tympani, taste is lost in the anterior part of the tongue, and there is some diminution in the secretion of saliva. When other parts of the nerve are diseased, taste is not interfered with, unless a secondary ascending—or, even more rarely, a descending—neuritis develops. Hearing may be increased, owing to paralysis of the stapedius, with consequent unopposed action of the tensor tympani. In ear-disease and in disease of the base of the brain, involving both facial and auditory nerves, hearing is lessened. Some degree of wasting takes place in the affected muscles, and both quantitative and qualitative electric changes quickly follow the palsy.

The duration of an attack varies from a few days to several months or a year, and in rare cases it is permanent. The onset is usually prompt, and the acme of the attack may be reached in from a few hours to a couple of days.

Diagnosis.—From the table previously given it will be easy to differentiate supra-nuclear from infra-nuclear palsy. In cases of long standing, when contractures have taken place, owing to the furrows thus produced the affected side may be taken for the sound side, but on getting the patient to whistle the true state of affairs will manifest itself.

Treatment.—Search for the cause. If ear-disease is present, make provision for free drainage; if syphilis, give iodid of potash, mercury, or both. In cases due to cold, the so-called rheumatic palsies, counter-irritation is especially called for, and cantharidal collodion, fly-blisters, or the actual cautery behind the ear or over the occiput are very useful. The bowels should be freely opened, and diaphoretics or hot baths and alkaline diuretics administered; in the inflammatory stage small doses of mercury are of value, and later mercuric iodid or general tonics. Galvanism should be employed to stimulate the nerves and to help in maintaining the tone of the muscles. When contractures threaten in late cases the use of electricity should be dispensed with.

DISEASES OF THE AUDITORY NERVE.

The eighth nerve has its deep origin in the medulla. The center is connected by fibers with the cerebellum, probably by means of an equilibrial mechanism. The auditory fibers decussate in the region of the nuclei, passing in the posterior extremity of the internal capsule to the opposite hemisphere. The cortical center is in the middle of the first temporo-sphenoid convolution. Destruction of that of the left side results in *word-deafness;* thus, spoken words may be heard, but are not recognized as such. This is not a common condition. Rarely the auditory tract may be involved between the cortex and the nucleus. The nerve may be implicated at the base of the brain by tumors, aneurysms, hemorrhage, meningitis, and traumatism. Erb has described a primary nerve-degeneration in tabes dorsalis. Disease may attack the labyrinth, either primarily or secondarily to ear-disease. Drugs—quinin, apiol, salicylates—may cause deafness similar to the labyrinthine variety. In anemia and in other conditions in which the general health is below par, also in hysteria, hearing may be affected. The lesions give rise either to an increased or diminished sense of hearing:

(*a*) *Hyperacusis*, in which certain or all sounds are intensified. Paralysis of the stapedius muscle causes low notes to be heard with great intensity. Auditory hyperesthesia may also occur in hysteria or during the course of cerebral or general disease.

(*b*) *Dysacusis*—difficult hearing—may be due to middle-ear disease, or it may exist as a "nervous deafness," the result of labyrinthine or nerve-disease. These may be differentiated by means of the tuning-fork. Normally, air-conduction is better than bone-conduction, and if in a deaf person a tuning-fork can be heard vibrating longer when held against the skull-vault or temporal bone than in front of the ear, there is some impairment of conduction in the meatus or middle ear. When the patient is deaf, and yet the normal relation is maintained between air- and bone-conduction, the labyrinth or the nerve is at fault.

(*c*) *Tinnitus aurium*—irritation of the auditory nerve—a condition in which subjective sounds occur, such as whirring, buzzing, ticking, or ringing in character. In certain subjects they are worse at night than during the day, and at times they are paroxysmal; as a rule, in any case they are intensified when the general system is below par.

Tinnitus may be caused by anemic or depraved nutritional states, intra-cranial aneurysm, pressure on the cervical sympathetic by enlarged glands, tumor, or aneurysm, impacted cerumen, otitis media, labyrinthine disturbance, blows upon the head, excessive auditory stimulation, loud noises, or it may occur during an attack of migraine or as an epileptic aura. In a neurasthenic individual the subjective noise, no matter what the cause, will be accentuated. The more complex and elaborate the sound, the greater the probability of its being of central origin.

Treatment.—Careful search must be made for the cause of any of these morbid conditions just described, and when practicable they should be removed. The system should be brought into as good a condition as possible. In hyperesthesia bromids occasionally avail. In dysacusis little can be done when the cause is labyrinthine. The same is true when the nerve or its centers are involved. Counter-irritation and electricity may be tried externally, and iodids internally. These measures should be employed in tinnitus, but with more hope of relief; in addition, sedatives are generally called for, and even morphin may be necessary in paroxysmal attacks.

MÉNIÈRE'S DISEASE.

Definition.—An aural or labyrinthine vertigo—originally described by Ménière in 1861; the cardinal symptoms are vertigo, deafness, noises in the ear, and sometimes vomiting.

Pathology.—There may be an inflammation or atrophy of the nerve-endings. There are also changes in the labyrinthine membrane from any cause or from hemorrhage.

Etiology.—Ménière's disease is most common after thirty, and is rarely met with before that age. It is twice as common in men as in women. The precise lesion is labyrinthine, and is the result of exposure, gout, syphilis, senile change, congestion, and, more rarely, hemorrhage. Any cerebral disturbance or gastric or other irritation is apt to induce an attack.

Symptoms.—Vertigo is present, and varies from an extremely slight transient attack, and one that is entirely subjective, to one of almost explosive violence. The patient may have a sensation of having been struck, and then of falling heavily to the ground. The slight form may be continuous with more or less frequent severe attacks, or a complete intermission of days, weeks, or months may transpire. The attacks may arise without apparent cause, or as a result of a blow or even a sudden movement, and occur during both working and sleeping hours. The giddiness, when severe, causes nausea and vomiting, and, if prolonged, bile is vomited as in ordinary bilious attacks. When the attack is very acute momentary unconsciousness supervenes. Nystagmus and diplopia may occur during an attack. Tinnitus and deafness usually exist together, the former being constant, but of slight degree, and possibly worse during an attack; it may be entirely absent between the attacks. The latter (nervous deafness) is constant and of varying severity in different individuals.

Diagnosis.—The occurrence of vertigo and tinnitus in a person with more or less nervous deafness, with or without gastric symptoms, establishes the diagnosis. The tinnitus and the character of the deafness usually suffice to distinguish this from other forms of vertigo. In epilepsy with auditory auræ the period of unconsciousness is generally much longer, and on regaining consciousness the patient is dull and drowsy for some time. It is possible also, as a rule, to elicit a history of convulsions.

Prognosis.—In some cases the condition grows progressively worse until deafness supervenes, when it ceases. Often, however, arrest or improvement, or even complete recovery, may be secured. In heart-disease the shock may prove fatal, and in the very acute but, fortunately, rare cases the prognosis is always bad.

Treatment.—Counter-irritation over the mastoid process and the internal use of bromids to lessen the morbid sensibility will prove valuable. The emunctories must be gotten in good condition, and any underlying disease, as syphilis or gout, must be treated. Charcot suggested the use of drugs that produce tinnitus—quinin, for instance. The cases were worse at the time, but some of them seemed to improve subsequently. Gowers employs sodium salicylate in 5-grain (0.324) doses, thrice daily, believing that more good arises when such drugs are given in moderation. Apiol might be tried in this connection. Nitroglycerin and the nitrites are sometimes of value in cases associated with arteriosclerosis.

DISEASES OF THE GLOSSO-PHARYNGEAL NERVE.

The ninth cranial nerve has its origin in the posterior part of the floor of the fourth ventricle, in close relation with the pneumogastric nerve. Our knowledge as to its function is not exact, both because it is seldom if ever involved alone, and also, on account of its many connections (with the trigeminus, the facial, the pneumogastric, and the sympathetic nerves), it is difficult to say whether the terminal fibers involved represent the functions of its roots or of one of its connections (Gowers).

Its fibers are distributed to the tonsils, the back of the tongue, the soft palate, the pharynx, the Eustachian tubes, and the tympanic cavity. It supplies both motor and sensory fibers, but not those of taste. This nerve is involved in the nuclear degenerations that are spoken of as bulbar palsies. It may be also affected by meningitis or new growths.

DISEASES OF THE PNEUMOGASTRIC NERVE.

As already stated, the origin of the tenth cranial nerve is in intimate relation with that of the ninth. It is also continuous below with that of the eleventh, and all three are associated with the center for the hypoglossal nerve. The nerve proper arises from the side of the medulla, and runs on either side of the neck in the sheath of the carotid artery, lying behind that vessel. It enters the thorax in front of the subclavian artery on the right side, and between the subclavian and the carotid on the left; then it courses beside the esophagus, and is distributed to the pharynx, larynx, lungs, heart, esophagus, and stomach, and sends fibers to the intestines and spleen.

The esophageal fibers are both motor and sensory, gastric fibers being chiefly sensory. The vagus is in part the motor nerve of the intestines. It also contains both accelerator and inhibitory fibers for the respiratory center, is the cardiac inhibitory nerve and a vaso-dilator, and is said to contain trophic fibers for the heart and lungs.

Etiology.—The nerve may be involved at its nucleus either by hemorrhage or softening. The nuclei of the ninth, eleventh, and twelfth nerves are simultaneously attacked, either wholly or in part, giving rise to a group of symptoms known as *bulbar palsy*. The tenth nerve at its superficial origin may be compressed by neoplasms, aneurysms, and the products of *meningitis*; in its course down the neck it may suffer pressure, or may either be tied in ligating the carotid artery or cut in the removal of a tumor or enlarged glands. Very rarely it may be injured by incised or punctured wounds, or be the seat of neuritis due to exposure or to some toxemia. The morbid conditions of the pneumogastric are best studied by considering the branches of distribution separately.

(*a*) **Pharyngeal Branches.**—The muscles and mucous membrane of the pharynx are supplied by branches of the pneumogastric and glosso-pharyngeal nerves, constituting the pharyngeal plexus. The pharynx may be the seat of spasm or paralysis: this is purely a "functional" condition, and usually occurs in hysteric (*globus hystericus*) or in nervous individuals. One of my own patients (a woman) after some domestic trouble became extremely nervous. She complained of increasing difficulty in swallowing, until finally she could scarcely take liquids, this symptom becoming aggravated when any one was watching her. She was cured by the daily passage of graduated esophageal bougies.

Paralysis of the pharynx causes difficulty in swallowing, so that food remains in the mouth instead of being passed into the esophagus. Particles often enter the larynx and give rise to paroxysms of coughing, and at times cause choking. When the soft palate is also paralyzed, the food is regurgitated into the nose. The lesion is generally nuclear, causing bulbar paralysis. The root of the nerve may be involved as it leaves the side of the medulla by meningitis or by pressure from a neoplasm or an aneurysm. Rarely it may be caused by a toxic neuritis.

(*b*) **Laryngeal Branches.**—The superior laryngeal nerve furnishes sensory fibers to the mucous membrane of the larynx above the vocal cords, and supplies also the crico-thyroid and epiglottidean muscles. The inferior or recurrent laryngeal nerve, which takes its origin in the superior thoracic region, winds around the arch of the aorta on the left side and around the subclavian artery on the right, reaching the larynx by running up between the trachea and esophagus. It is the sensory nerve of the larynx below the vocal cords, also of the entire trachea, and supplies all the muscles of the larynx except those named above. It has been shown that the motor fibers of the larynx come from the glosso-pharyngeal nucleus, the pneumogastric fibers being sensory.

Spasm of the larynx is due to over-action of the glottis-closers (the adductors), though some cases described in this category are probably instances of abductor paralysis. The condition is rather rare in adults, but quite common in children (laryngismus stridulus), and particularly in rachitic subjects. An attack may also be induced in those predisposed by any form of nerve-irritation or catarrhal condition of the respiratory tract. It may be part of a general neurosis; it is sometimes seen in tabes dorsalis (*laryngeal crisis*); and Liveing reports that he has seen it take the place of an attack of migraine. *Spastic aphoria* consists of a spasm induced whenever an attempt to speak is made. Laryngeal spasms occur most frequently at night. Dyspnea is the most striking symptom, and is so intense in some cases that suffocation seems imminent. The patient may be cyanotic. Soon the retained carbonic acid gas causes relaxation, but, as the cords open slowly, the inspiration is accompanied by a crowing sound, and the expiratory sound is harsher than normal.

Paralysis of the larynx may be the result of a nuclear degeneration (glosso-pharyngeal), producing chronic bulbar paralysis, as already mentioned; this form may occur in disseminated sclerosis, tabes dorsalis, general paralysis of the insane, and in certain toxemias. The paralysis is generally bilateral; rarely it is unilateral.

Very rarely a cerebral cortical lesion in the laryngeal center may cause pseudo-bulbar paralysis. Since the two centers are compensatory, the lesion must be bilateral.

The nerve may be involved at its root or in any part of the trunk, and such lesions are usually unilateral. The recurrent laryngeal nerve, especially the left, is more apt to be diseased than the superior, on account of its position. Thus, the arch of the aorta is more frequently the seat of an aneurysm than the subclavian; enlarged thoracic glands, neoplasms, and an enlarged thyroid can also damage these nerves. The peripheral filaments may be attacked as part of a multiple neuritis.

In certain cases the muscles become weakened without being paralyzed, this possibly being due to a local neuritis, or to a congestion and inflammation of the mucous membrane from over-use (*clergymen's sore throat*), or as the result of exposure.

The following are the chief forms of paralysis:

(1) *Complete Paralysis.*—By this is generally understood paralysis of all except the crico-thyroid and epiglottidean muscles, though occasionally these may also be involved. Since the cords are paralyzed, phonation is impossible. As a rule, there is no interference with respiration, though the pressure of the in-going air may bring the cords nearer together, and thus produce a certain amount of inspiratory harshness.

As the cords cannot be closed, coughing is impossible, as the air escapes through the glottis, and no expulsive force can be given to it. When the paralysis is unilateral these symptoms will of necessity be modified, and some degree of phonation may be possible. The most common cause of this condition is an involvement of the recurrent laryngeal nerve; the lesion may, however, be nuclear or in the course of the nerve-trunk.

(2) *Paralysis of the Abductors.*—The only special abductor muscles are the posterior crico-arytenoids. When they are involved the glottis fails to open in inspiration, and the unopposed adductors bring the vocal cords together. They are still more closely approximated during inspiration by the column of air, and hence the prolonged, stridulous inspiratory sound. Phonation and expiration are practically unchanged. It is quite likely that many cases supposed to be instances of hysteric spasm of the glottis are really cases of abductor paralysis.

In unilateral paralysis the normal movements of the unaffected vocal cord prevent any marked degree of dyspnea and stridor: phonation is usually hoarse and of a low pitch. In cases of long duration the symptoms become more marked as the unopposed adductors undergo secondary contracture and still further narrow the glottis.

This condition may be due either to central disease or to some local change. The abductor muscles may be degenerated, while all the other laryngeal muscles are healthy, or one or both recurrent nerves may be affected. These nerves innervate both the abductors and adductors, and it is not clearly understood why the abductors alone should suffer when the parent nerve-trunk is involved. At any time it might be a very grave condition, for should any swelling of the cords supervene nothing but a prompt laryngotomy could prevent suffocation.

(3) *Adductor Paralysis.*—The cords move normally during respiration, and hence there is no stridor; as they cannot be approximated, however, phonation is impossible. This condition is met with in hysteria, producing hysteric aphonia, in public speakers who overtax their voices, and also in laryngitis.

The following table, from Gowers' text-book on *Diseases of the Nervous System*, enables one to get a comprehensive idea of the subject:

SYMPTOMS.	SIGNS.	LESIONS.
No voice; no cough; stridor only on deep inspiration.	Both cords moderately abducted and motionless.	Total bilateral palsy.
Voice low-pitched and hoarse; no cough; stridor absent or slight on deep breathing.	One cord moderately abducted and motionless, the other moving freely, and even beyond the middle line in phonation.	Total unilateral palsy.
Voice little changed; cough normal: inspiration difficult and long, with loud stridor.	Both cords near together, and, during inspiration, not separated, but even drawn nearer together.	Total abductor palsy.
Symptoms inconclusive; little affection of voice or cough.	One cord near the middle line, not moving during inspiration; the other normal.	Unilateral abductor palsy.
No voice; perfect cough; no stridor or dyspnea.	Cords normal in position, and moving normally in respiration, but not brought together on an attempt at phonation.	Adductor palsy.

Sensory disturbances of the larynx are rare, and especially hyperesthesia. Anesthesia may be due to hysteria, or to bulbar paralysis, or to disease of the superior laryngeal nerve. It is dangerous, as food may enter the windpipe.

(*c*) **Cardiac Branches.**—These with branches from the sympathetic form the cardiac plexus. The vagus contains both accelerator and inhibitory fibers, but the latter predominate; therefore irritation of the nerve, either centric or peripheral, will slow the heart's action. Czermak was able to slow the action of his heart by pressing a small tumor in his neck against the vagus nerve. When the function of the nerve is lowered, inhibition is removed and the heart's action becomes rapid. This may be brought about by a toxemic neuritis, by pressure, accidental ligature, or by incised or punctured wounds. Various emotions and nervous states may bring about the same result.

(*d*) **Pulmonary Branches.**—Both accelerator and inhibitory fibers exist, but in this case the accelerator influence predominates, so that irritation results in increased respiratory movements or even in bronchial spasm, since the bronchial muscles are also supplied by this nerve. It is this nerve that is supposed to be concerned in the production of asthmatic paroxysms. Therefore, when the nerve-function is lowered the respirations become much slower. The nerve is supposed to contain trophic fibers for the lungs.

(*e*) **Esophageal,** (*f*) **Gastric, and** (*g*) **Intestinal Branches.**—The esophageal branches are rarely damaged, and irritation (spasm) occurs more frequently than paralysis. The pneumogastric gives the sensory, and in part the motor, nerve-supply to the stomach, and irritation gives rise to increased contractions with some pain.

The sensation of hunger is supposed to be associated with the vagus nerve, and vomiting may result from direct or reflex irritation. Paralysis causes some diminution of the gastric contractions. Normally, the vagi accelerate intestinal peristalsis.

Treatment.—It is almost always impossible to remove the cause of the above conditions. Syphilitic lesions are probably the most amenable, and in the various laryngeal palsies electricity may be employed, though it is of somewhat doubtful utility, and in abductor palsy may possibly exert a harmful influence by stimulating the adductors. Strychnin and general tonics should be administered. Massage of the larynx may be tried, and in spasmodic conditions attention should be directed to the general physical state. All sources of nerve-irritation should be removed if possible, and bromids, or even chloral, should be given.

DISEASES OF THE SPINAL ACCESSORY NERVE.

This nerve consists of two parts—an external or spinal, and an internal or accessory, portion. The latter has already been described in connection with the pneumogastric nerve. It forms the motor portion of that nerve, and is distributed to the laryngeal and pharyngeal muscles. The spinal element arises from the multipolar ganglion-cells in the anterior gray horns of the cervical cord, ascends and enters the cranium through the foramen magnum, and leaves it, after joining with the accessory part, through the jugular foramen. It supplies the sterno-mastoid muscles and in part the trapezius.

Injury or disease of the nerve may result in spasm or paralysis. Only the spinal part is considered in this section.

TORTICOLLIS.

(*Wry-neck.*)

This may be a congenital or an acquired condition.

Congenital torticollis, or "fixed wry-neck," is the result of an atrophy and shortening of the sterno-mastoid muscle, brought about by some intra-uterine condition or, possibly, by an injury at birth. The right muscle is most commonly affected. The head turns slightly toward the sound side; the eye may deviate, and curvature of the cervical spine may develop.

Facial asymmetry is a usual concomitant of this condition. The face on the same side as the lesion develops less rapidly than the other side, and in time secondary contracture of the unopposed muscles takes place. The torticollis can be cured by tenotomy, but the facial asymmetry persists. Fixation is necessary for a while when contracture exists.

Spasmodic wry-neck may be tonic or clonic. These forms may coexist, alternate, or occur independently in different individuals. The condition is met with almost exclusively in adults, and occurs most frequently in middle-aged men.

Pathology.—No macroscopic or microscopic evidence of any lesion has been discovered, and the condition is probably dependent upon an over-activity of the neurons in the various centers that control the muscles of the affected part.

Etiology.—The influence of sex and age has been mentioned; a neurotic heredity may also predispose. Torticollis may follow habit-spasm, or some injury to the head or neck, or exposure to cold, the latter constituting the "rheumatic" type. In a case of my own, a man of 23, it followed an attack of acute articular rheumatism and was associated with high arterial tension. Rarely, robust, healthy-looking individuals are attacked without any apparent cause. Cervical caries may cause rigidity of the neck, simulating torticollis. The spasm is usually tonic in such cases, as it is in those of the "inflammatory" type, where, in children particularly, enlarged and painful glands are found under the sterno-mastoid.

Symptoms.—The occiput is drawn toward the shoulder of the affected side, the chin is elevated, and the face rotated more or less toward the sound side. The sterno-mastoid may alone be affected, or the trapezius may also be involved. In the latter case greater depression of the head takes place. Spinal curvature may ensue, the convexity being toward the sound side. This only takes place in cases that have existed for some time. Clonic spasm is infinitely more distressing and more apt to be permanent.

Some pain and muscular twitching may precede the onset of the attack, though, as a rule, muscular contractions are the first indication. These are mild at first, and rarely abruptly, more commonly slowly, they increase in severity. As the case progresses other muscles, and even those of the arm, become involved. Cases have been described in which certain muscles or groups of muscles in the hand or arm have been primarily affected, the condition gradually spreading from them. The spasm usually ceases during sleep. An attack may cause pain, but, as a rule, it induces merely a feeling of fatigue in the muscles; it is worse if the patient is excited or emotional. Bilateral spasm may occur, the muscles of both sides being equally affected (*retro-collic spasm*). Gowers speaks of a case in which the backward displacement of the head was so great that the face was horizontal and looked directly upward.

Diagnosis.—As a rule this is not difficult. When spasm is induced by enlarged and painful glands beneath the sterno-mastoid the age of the patient will be of value in determining the true condition. This usually occurs in children; true wry-neck, on the other hand, very rarely commences before the thirtieth year. Hysteric spasm may also simulate spasmodic torticollis, but it generally occurs in young women, and usually other evidences of hysteria are also present. The *rheumatic type* and the rigidity induced by *caries of the spine* must be differentiated from one another and from spasmodic wry-neck. If the rigidity comes on suddenly, following exposure to cold or wet, and the pain is not increased at night or by depressing the head upon the spine, and is relieved by hot applications, the condition is probably rheumatic. When the rigidity and pain are of slow onset, without history of exposure, and the pain is both worse at night and is increased by depressing the head upon the spine, but is relieved by elevating the head, the condition is very probably one of caries of the spine.

Prognosis.—Very rarely the torticollis may diminish or even cease after an existence of months or years. Usually, however, it is persistent, either being stationary or slowly increasing in severity and widening in range. The prognosis must always be guarded, and in severe cases grave as to recovery, though the disease does not shorten life.

Treatment.—Generally very little can be expected from medication. Bromids, morphin, chloral, hyoscyamus, or cannabis indica may be tried, as may the various forms of counter-irritation. Morphin administered hypodermically, has been most effectual in some cases, but the danger of establishing the habit should not be forgotten. Galvanism should be tried, the negative pole being placed over the occipital region and the positive over the affected muscles. Nerve-stretching and tenotomy of the affected muscles is of very little value. The only surgical procedure that has proved of any distinct value is neurectomy, with excision of a part of the nerve to prevent reunion. This necessarily causes paralysis and atrophy of the muscles supplied; but, since it often abolishes the spasm, the slight loss of power and the interference with the movement of the head are comparatively infinitesimal. The results, however, are not uniform even so far as the spasm is concerned.

PARALYSIS OF THE SPINAL ACCESSORY NERVE.

The accessory portion has been previously considered in describing the laryngeal branches of the pneumogastric.

In the spinal portion the nuclei may be involved in degenerative lesions of the motor region of the spinal gray matter. The nerve-trunk may be damaged by pressure from exudative products (meningitis), tumors, or caries, with resulting paralysis and wasting of the sterno-mastoid and, in part, of the trapezius. This latter muscle is also supplied by the cervical nerves. The patient has difficulty in rotating the head to the side opposite that on which the paralysis exists, and the affected muscle does not stand out in movements of the head. Unless secondary contraction of the unopposed muscle sets in, no deviation occurs when the head is at rest. The only portion of the trapezius that is involved in paralysis of the external part of the eleventh nerve stretches from the occipital bone to the acromion. The normal contour of the neck is lost in such cases, and the ability to raise the arm is interfered with because the trapezius cannot fix the scapula, the fulcrum of the deltoid. Bilateral paralysis may occur as in progressive muscular atrophy; if both sterno-mastoids are involved, the head falls backward; if both trapezii, it falls forward.

The **treatment** is that of the underlying cause. If the lesion is nuclear, practically nothing can be done. If the condition is due to pressure, in some cases relief may be obtained. Electricity and massage should be employed during the recovery of the nerve.

DISEASES OF THE HYPOGLOSSAL NERVE.

The nucleus of the twelfth cranial nerve is in the most posterior portion of the floor of the fourth ventricle. It is said by some observers that the nuclei of the fibers for the palate and vocal cords that run in the spinal accessory nerve may be in the lower part of the twelfth-nerve nucleus.

The cortical center for this nerve is in the lower part of the ascending frontal convolution, in the neighborhood of the cortical facial center. This propinquity probably explains the simultaneous involvement of the facial and lingual muscles in some cases. The hypoglossal is the motor nerve for the tongue and for most of the muscles attached to the hyoid bone. Spasm or paralysis may follow disease of the nerve.

Spasm may be either unilateral or bilateral. It is probably met with most commonly in hysteria, or as a part of some general convulsive condition, as epilepsy or chorea. It may also be associated with facial spasm, as mentioned above. Irritation of the fifth nerve (dental caries, ulceration of the gums) seems to be responsible for some cases. "Paroxysmal clonic spasm" is a form in which the tongue is rapidly thrust in and out. Various sensations in the affected region may precede the attack. A rare form—*aphthongia*—is induced when an attempt to speak is made. The prognosis in this condition is good, and a general tonic treatment is indicated.

Paralysis may result from supra-nuclear, nuclear, or infra-nuclear lesions.

Supra-nuclear.—The lesion may be anywhere between the cortex (lower part of the ascending frontal gyrus) and the medulla, and causes paralysis on the opposite side. In this condition the affected muscles do not atrophy nor do they show any electric change.

Nuclear.—The lesion is usually degenerative. It may either be of sudden onset (vascular), less rapid, but still acute (inflammatory), or it may be chronic, as in bulbar palsy or tabes dorsalis. The nuclei are so close together that the condition is almost invariably bilateral.

Infra-nuclear.—The fibers may be injured by the pressure of neoplasms or by the products of meningitis or of syphilis. Disease of the bone may also involve the nerve in its passage through the foramen. More rarely, some traumatism or disease of the upper cervical vertebræ may simultaneously injure the eleventh and twelfth nerves.

Symptoms.—Paralysis and atrophy of one or both sides of the tongue and fibrillary twitchings may be noted, and if the condition be unilateral, the tongue when protruded deviates toward the affected side. Articulation, mastication, and swallowing are but very slightly interfered with. In the bilateral form, however, these are very much impaired; the tongue cannot be protruded and lies motionless on the floor of the mouth. The atrophy is muscular. This throws the mucous membrane into deep folds. Sensation and taste are unaltered.

Diagnosis.—If the lesion is supra-nuclear, there is hemiplegia on the same side as the lingual paralysis, without atrophy of the tongue-muscles. When nuclear, it is, as has been said, generally bilateral and forms part of a bulbar paralysis. There is also wasting of the lingual muscles. When the fibers are involved in the medulla, there is paralysis of the tongue on one side, of the limbs on the other, and the tongue deviates from the paralyzed side of the body. Outside the medulla the condition is, as a rule, unilateral, and the spinal accessory fibers are frequently involved. In the nuclear and infra-nuclear varieties there is wasting of the muscles.

The **prognosis** is usually unfavorable, and the **treatment** consists of a course of general tonics and of mercury and the iodids, with counter-irritation. Electricity may also be tried.

DISEASES OF THE SPINAL NERVES.

DISEASES OF THE CERVICAL PLEXUS.

Phrenic Nerve.—This nerve is usually involved as a result of some lesion of the ganglion-cells in the anterior gray horns at the level of the third or fourth cervical nerve. The trunk may be damaged by pressure, as by aneurysm or neoplasms, or by traumatism, or it may be the seat of neuritis. More or less immobility of the diaphragm follows, amounting in some cases to complete paralysis. This is not readily seen with the patient at rest, and in women it is specially hard to observe, as their breathing is chiefly of the costal type. The abdomen moves in in inspiration, and out in expiration, forming the reverse of the normal movements. Immobility of the diaphragm may also occur in peritonitis, diaphragmatic pleurisy, and in large pleural effusions. Exertion readily causes dyspnea, and pulmonary diseases are apt to be exaggerated as the products of secretion accumulate. This is most apt to occur when the condition is bilateral, as it usually is in the presence of cord-lesions. Other muscles always suffer in this form in addition to the diaphragm. When the nerve alone is involved the affection is generally unilateral.

DISEASES OF THE BRACHIAL PLEXUS.

This may either be involved *in toto*, or any of its branches may be affected separately, or the nerve-roots that unite to form the brachial plexus. Isolated disease of any of the roots may be produced by injury, caries of the vertebræ, or meningeal disease. The symptoms will be almost exactly the same as those produced by disease of the corresponding segment of the cord, but are more likely to be unilateral, and to be either purely motor or sensory, unless the lesion is extensive.

The posterior thoracic supplies the serratus magnus muscle. It may be injured directly by pressure, as in the carrying of heavy loads on the shoulder or by a fall or other traumatism. Rarely, it follows exposure to cold. Its involvement may be a part of an anterior polio-myelitis or of a progressive muscular atrophy. When the muscle is paralyzed the posterior edge of the scapula stands out prominently, and particularly when the arm is moved forward. Neuralgic pains in the neck generally precede the neuritis. The course of the disease is always slow. During the early stage counter-irritation, the iodids and mercury internally, and later electric stimulation to keep up the tone of the muscles, constitute the treatment.

Combined Paralysis.—Two or more nerves, or even the entire plexus, may be involved at one time by new growths in the cervical region, neuritis, stretching or rupture of the nerves by wounds, fractures, or dislocations, and particularly by subcoracoid dislocation. Duchenne has described a form of palsy produced in infants during birth by pressure due to some malposition or to injury by the finger or a hook. Brachial neuritis may follow some injury to one of the nerve-branches (ascending neuritis), or it may be primary. The latter variety is rare, and usually occurs after middle life, especially in cases with a gouty history. Paroxysmal or continuous pain, increased by any movement of the arm and tenderness on pressure over the affected nerves, is the chief symptom. If on the left side, it simulates angina pectoris.

Individual Nerves of the Arm.—These may be damaged by pressure due to a tumor, an aneurysm, or to callus. *Sleep-palsy* and *crutch-palsy* are both pressure-palsies. The nerves may also be contused or torn in fractures or dislocations, and palsy may follow a fall or blow upon the shoulder; I have seen it occur in a heavy man after a fall upon the hand. Primary or secondary neuritis may develop, and, very rarely, neuromata appear.

The *supra-scapular* nerve supplies the supra- and infra-spinati muscles. Paralysis causes imperfect outward rotation of the humerus and rotation of the scapula, with elevation and inversion of the lower angle. Various movements of the arm are thereby interfered with, and the limb tires very readily. More work is thrown on the deltoid, and in time it hypertrophies, causing it to stand out more prominently against the infra-spinatus. The skin over the scapula is usually anesthetic.

The *circumflex* nerve supplies the deltoid and teres minor and the skin over the deltoid and the shoulder-joint. Paralysis results in inability to raise the arm and in wasting of the muscles, with or without anesthesia. Adhesions may form in the joint.

The *musculo-spiral* nerve is more often paralyzed than any other nerve of the arm, its position rendering it particularly liable to pressure. It supplies the triceps and supinator muscles, and is the extensor nerve of the arm. It also supplies the skin on the radial side of the dorsal surface of the hand, the back of the thumb, and the index and radial side of the middle finger. A lesion high up results in paralysis of the extensors of the elbow, wrist, and hand, and of the supinators. Probably the point most commonly attacked is about the middle of the humerus. In such cases the triceps escapes. The characteristic symptoms, however, are wrist-drop and finger-drop, consisting of an inability to extend the hand on the forearm, also the first phalanges of the fingers and thumb. In pressure-palsies the power of supination is usually lost also. Sensory symptoms vary, and are seldom pronounced. There may be slight impairment or tingling or burning sensations.

This condition can usually be differentiated from lead-palsy by the rapidity of onset—by the fact that pressure-palsies are almost invariably unilateral, and that the supinators are involved. Lead-palsy has a slow onset and is bilateral, generally without supinator involvement. Loss of sensation precedes the pressure-palsy. The history too will generally throw some light on the case. I have seen a case of right-sided unilateral wrist-drop in a man who worked in lead with his right hand only. Bilateral wrist-drop may occur in any form of toxic neuritis, but the involvement of other nerves, the manner of attack, and the history of the case will serve to simplify the diagnosis.

Recovery follows in almost all cases of musculo-spiral nerve-involvement, though in cases in which qualitative nerve-changes have taken place it is necessarily delayed.

The *treatment* is that of neuritis.

The *median nerve* supplies the pronators, digital flexors, except the ulnar half of the deep flexor, the radial flexor of the wrist, the abductor and flexor muscles of the thumb, and the two radial lumbricales. It furnishes sensation to the radial side of the palm and front of the thumb, and to the front and back of the first and second and half of the third fingers. This nerve may be the seat of an injury or of neuritis, but is seldom involved alone. The most striking symptoms are wasting of the thenar eminence and an inability to oppose the thumb to the tips of the fingers. Further pronation is only possible in so far as the supinator longus subserves that function—viz. the misposition. Ulnar flexion of the wrist alone remains. Flexion of the phalanges is interfered with. Sensation may or may not be lost.

The *ulnar nerve* supplies the ulnar flexor of the wrist, the ulnar half of the deep flexor of the fingers, the muscles of the little finger, the adductor and inner head of the short flexor of the thumb, the interossei, and some of the lumbricales. It supplies with sensation the front of one and a half and the back of two and a half fingers on the ulnar side. Paralysis causes radial deviation of the hand in flexion of the wrist, loss of adduction of the thumb, and inability to move the little finger. The hypothenar prominence disappears.

The first phalanges cannot be flexed, and the second and third cannot be extended. This is exaggerated in old cases, though still it is not so marked as the "claw hand" of progressive muscular atrophy, since the first two lumbricales escape, being supplied by the median nerve. Sensory symptoms vary.

The *diagnosis* is usually easy. It is well to remember that, since this nerve is the lowest in its point of origin of any considered in this group, ascending cord-diseases will involve it before any of the other brachial nerves. It may also be damaged by disease limited to the lowest part of the cervical enlargement of the cord.

DISEASES OF THE LUMBAR AND SACRAL PLEXUSES.

The **lumbar plexus or its branches** may be involved by abdominal growths, enlarged glands, psoas abscess, disease of the vertebræ, neuritis, and rarely by wounds or dislocation of the hip or during parturition.

The Obturator Nerve.—When the power of adduction of the thigh is lost and the affected leg cannot be crossed over the other, outward rotation is somewhat impaired.

Anterior crural nerve paralysis causes loss of power and wasting of the extensors of the knee, loss of knee-jerk, and anesthesia of most of the thigh and the inner side of the leg and foot.

The *superior gluteal nerve* supplies the gluteus minimus and medius muscles. When it is involved adduction and circumduction of the thigh are lost.

The **sacral plexus and its branches** may be damaged by pelvic neoplasms or inflammation, neuritis (generally secondary to sciatic nerve-involvement), pressure during labor, wounds, dislocations, aneurysms, and diseases of the bone.

The *small sciatic nerve* supplies the gluteus maximus muscle. It is seldom involved alone. Lesions cause difficulty in rising from the sitting posture and anesthesia of the back of the thigh and of the upper part of the leg posteriorly.

The *great sciatic nerve* supplies the flexors of the leg and the muscles below the knee, and also sensation to the outer half of the leg, the sole, and part of the dorsum of the foot. Paralysis causes more or less interference with the act of walking, anesthesia in the part supplied, and wasting of the muscles.

The *external popliteal* or *peroneal nerve* supplies the tibialis anticus, the peronei, the long extensor of the toes, and the extensor brevis digitorum; it also supplies sensation to the outer half of the front of the leg and to the dorsum of the foot. Paralysis causes foot-drop and toe-drop, rendering it necessary to lift the leg high in walking, so that the foot will clear the ground; this constitutes the *steppage gait* referred to in the section on Neuritis. The region supplied is anesthetic.

The *internal popliteal nerve* supplies the popliteus, tibialis posticus, the calf-muscles, the long flexors of the toes, and the muscles of the sole. When paralyzed, flexion of the foot and toes is impossible, and sensation is lost over the back of the leg in its lower part and over the sole. In old cases talipes calcaneus results. The plantar nerves are rarely, if ever, involved alone.

ACUTE ASCENDING PARALYSIS.

(*Landry's Paralysis.*)

Definition.—An acute paralysis, beginning in the legs and ascending by way of the trunk and upper extremities, and ultimately involving the *medullary* centers. It usually runs a short course, and, as a rule, terminates in death.

Pathology.—Although in many cases neither gross nor microscopic lesions have been found, either in the cells, peripheral fibers, or muscles, it is believed to be either an acute myelitis or an acute polyneuritis, the weight of opinion seeming to favor the latter view. Ross arrived at the latter conclusion after an analysis of 93 cases. Nauwerck, Barth, and Centanni hold the same belief, and the latter has discovered a bacillus in the lymph-spaces of peripheral nerves. Remlinger[1] has reported a case occurring in a young man in whom paraplegia developed acutely, and eleven days later death resulted from bulbar involvement. Postmortem the cord was found congested in the region of the anterior horns. Microscopic examination revealed the presence of inflammation in the cervical cord. The multipolar ganglion-cells of the anterior horns were degenerated, and between them were found streptococci. Pure cultures of streptococci were obtained from the cord at various levels, but they were non-pathogenic for the rabbit. R. and F. Schultze and Sinkler have also reported cases in which the only postmortem lesion was myelitis, yet, as stated, the majority of observers believe it to be a neuritis. That it is primarily due to some toxemia, however, as originally claimed by Westphal, cannot be gainsaid. The prodromes, when present, are suggestive, and the enlargement of the spleen, which is a constant concomitant, and more rarely the lymphatic enlargement and albuminuria, are all confirmatory. That the poison should have a selective tendency, since the nervous involvement is chiefly or solely motor, is not unique. We meet with toxic paralysis of the motor muscles of the eye, also with lead-palsy.

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[1] *Gazette hebdomadaire de Médecine et de Chirurgie*, No. 27, 1896.

Etiology.—No definite cause is known. It has followed cold and exposure, traumatism, and the infectious fevers, including influenza. Remlinger's case, quoted above, followed malaria. It occurs in males chiefly between twenty and forty years.

Symptoms.—In the most acute cases there are practically no prodromal symptoms other than malaise and possibly chilly sensations. Weakness, followed in a few hours or a day or two by paralysis, develops in the lower extremities. One may be involved a few hours earlier than the other. It spreads toward, and soon involves, the trunk also, and in quick succession the arms. The third and usually fatal stage is reached when bulbar symptoms develop. Very rarely the upper extremities may be first attacked. Death may occur in forty-eight hours. The paralysis is a flaccid one; the muscles can be passively moved without offering any resistance. Wasting sets in, but no electric changes. In less acute cases a decided febrile stage precedes the onset of paralysis, chills, fever, malaise, and possibly formication or even sharp pain. In any case the later symptoms are pre-eminently or solely motor. Sensory symptoms when present are very slight. Sensation may be delayed, and the reflexes are generally absent; accordingly, there is edema or sweating. The bladder and rectum are not implicated, nor do bed-sores develop. As stated, when the bulb is attacked death generally follows, due to cardiac or respiratory failure or to interference with deglutition. There are no cerebral symptoms.

Course.—Death may occur in from forty-eight hours to a few weeks. A few cases of recovery have been reported, however, in some of which paralysis had been widespread, even reaching the bulb, judging from the labored respiration. When improvement takes place, it does so in the reverse order to the onset, so that the part last affected is the first to recover. It is much slower than the invasion.

Diagnosis.—The rapid onset of a paralysis that usually ascends, the relaxation of the muscles, slight wasting, if any, and the absence of electric changes and of sensory symptoms, with or without fever, serve to make the diagnosis, and to distinguish Landry's disease from poliomyelitis, neuritis, and spinal hemorrhage. For the differential diagnosis between Landry's paralysis and acute myelitis, see page 1076.

Prognosis.—Always grave, particularly if bulbar symptoms occur, and especially if they appear early.

The **treatment** is essentially the same as that for any acute disease of the cord or nerves—*i. e.* rest, freedom from all excitement or worry, moderate purgation and diaphoresis; ergot, belladonna, and iodids internally. Should the patient survive, electricity and massage should be administered.

II. DISEASES OF THE SPINAL CORD AND ITS MENINGES.

DISEASES OF THE MENINGES.

MENINGITIS is very rarely a primary condition. Both the dura and pia may be involved. In the former case the inflammation is usually due to some morbid condition of the vertebræ, while in the latter it is secondary to some infection, as in pyemia, sepsis, pneumonia, typhoid, or the acute exanthemata. It may be part of a tuberculous condition (*vide* Tuberculosis, p. 282) or of epidemic cerebro-spinal meningitis. Injuries also lead to inflammation of the meninges of the cord.

PACHYMENINGITIS.

Definition.—Inflammation of the dura mater. The dura may be involved on its outer or inner surface (*pachymeningitis externa* or *interna*), or the loose connective tissue between the dura and bony canal may be the seat of a peripachymeningitis.

Pachymeningitis externa is always secondary, and usually results from syphilitic or carious affections of the bone, or from pressure due to tumors or to traumatism. It may either be acute or chronic. Of the latter type, those cases due to Pott's disease are most common. The membrane is involved to a greater or less extent. The internal surface may escape entirely, or it may be slightly roughened and adherent to the arachnoid; externally, however, the dura is usually thickened, rough, and covered with a cheesy material.

Pachymeningitis interna was first described by Charcot in 1871, and named "*pachymeningitis cervicalis hypertrophica.*" It is of obscure origin. The dura is generally much thickened, and gives the impression of being made up of a number of concentric layers. The pia is only involved to a slight degree as a rule. Areas of degeneration may occur in the cord, as may also dilatation of its central canal. As implied by the name, this variety of pachymeningitis is found chiefly in the cervical region, and the clinical symptoms result from involvement of the nerve-roots. It is a chronic process, and has been divided into three periods, as follows: (*a*) *The painful period*, lasting, as a rule, two or three months, in which severe neuralgic pains exist, their location being determined by the roots involved. They are mostly in the occiput and upper extremities, however. Early there may be hyperesthesia, numbness, tingling, and, rarely, an herpetic eruption. (*b*) *The Paralytic Period.*—As a result of compression of the motor roots an atrophic paralysis of the upper extremities develops. A peculiar selective tendency is manifested, the radial nerve being spared, while the median and ulnar nerves are involved. This results in a modified "claw-hand" deformity and in an over-extension of the wrists, with flexion of the fingers. Anesthesia may be noted. (*c*) *Spastic Paraplegia.*—This results when the compression has produced degeneration of the cord. Generally, there are paresis of the lower extremities and increased reflexes, but no muscular wasting, since the trophic centers are intact. Occasionally, however, anesthesia and paralysis of the legs and bladder develop, bed-sores following, and finally death from exhaustion.

The *prognosis* is unfavorable, practically all cases terminate in death, but the duration is variable.

The *diagnosis* must be made from amyotropic lateral sclerosis, syringomyelia, and from pressure by tumors. The latter and cervical spondylitis often give rise to almost identical symptoms. Amyotrophic lateral sclerosis does not give rise to sensory disturbances; bulbar symptoms are often present, the lower extremities atrophy, and the bladder functions are preserved. Syringomyelia induces characteristic changes in thermic sensibility, and often anesthesia, but rarely severe neuralgic or radiating pains.

Treatment is not of much avail. Potassium iodid and electricity are the chief measures. In cases otherwise hopeless an exploratory operation is sometimes justifiable.

Pachymeningitis hæmorrhagica interna, or *hematoma of the dura mater,* may occur in any part of the cord, and is usually associated with a similar condition in the cerebral dura. Cysts may be found in the inner surface of the dura, containing broken-down blood-cells and hematoidin crystals, and in their neighborhood an increase of fibrous tissue may be noted. The condition occurs most frequently in alcoholics or general paralytics.

LEPTOMENINGITIS.

Definition.—Inflammation of the pia mater. This may be either acute or chronic.

ACUTE LEPTOMENINGITIS.

(*Acute Spinal Meningitis.*)

Pathology.—The vessels are injected, the membrane becomes cloudy, a sero-fibrinous or purulent exudate either surrounds the cord or may only exist in patches, and in the more severe cases the cord itself is involved (*meningomyelitis*). The spinal meninges alone may be involved to a greater or less extent, but as a rule, the cerebral meninges are similarly involved. It should be remembered that many cases presenting clinically the picture of meningitis shows absolutely no gross *postmortem* lesions of the cerebral or spinal membranes. This is especially true of pneumonia and influenza. In other cases the chief alterations are found in the ganglion-cells of the cord, or no lesions, not even microscopic, are found. These are spoken of as *meningismus.*

Etiology.—This is always microörganismal, and a great variety of bacteria have been discovered. The most common is the pneumococcus, in which case the disease may or may not be associated with pneumonia; next in frequency is the meningococcus; and then the various pyogenic cocci, the influenza bacillus, the typhoid bacillus, etc.

Symptoms.—These are chiefly pain in the back, often excruciating, with fixation, retraction of the head, tenderness on pressure along the spine, tremors or spasm of the muscles, and various sensory disturbances. Reflexes are early increased, and later diminished or absent. Should the cord be involved, paralysis, incontinence of urine and feces, and even bed-sores, may develop. The symptoms are more fully discussed in speaking of the tuberculous and epidemic varieties.

Diagnosis.—It is often very difficult to differentiate the several varieties of spinal meningitis, and equally so to decide whether the case is actually meningeal when some other disease is present. Even bulbar symptoms may be present without postmortem lesions; I have seen this exemplified in a case of Bright's disease. The tuberculous form is readily diagnosticated, especially if any collateral evidence of tuberculosis exists. It is a point of some value in the diagnosis to note the absence of marked leukocytosis in tuberculous and its presence in purulent meningitis. The presence of *Kernig's sign* is in favor of cerebro-spinal meningitis.

Spinal paracentesis or *lumbar puncture*, first introduced by Quincke of Kiel in 1891, is a most valuable diagnostic measure and simple of application. He was first led to adopt it by the knowledge that a free communication exists between the subarachnoid spaces of the brain and spinal cord through the foramen of Magendie; hence he conceived the idea of a lumbar puncture supplanting the older method of tapping the lateral ventricles in cases of hydrocephalus. Later, he used it in meningitis. Therapeutically, it is of little value, but it often serves to confirm the diagnosis. The patient should be in a sitting posture with a slight forward inclination of the trunk. The puncture is then made between the third and fourth lumbar vertebræ and a little to one side of the middle line. Absolute cleanliness should be observed, and the needle introduced slowly until the fluid begins to flow by its own pressure.

The **prognosis** is unfavorable as a rule, particularly in the tuberculous form.

The **treatment** is the same as that of cerebro-spinal meningitis (*vide* p. 133).

CHRONIC LEPTOMENINGITIS.

This disease may follow the acute form or be due to chronic alcoholism, syphilis, trauma, or disease of the cord.

Pathology.—The pia is cloudy and swollen, and often adherent to the arachnoid, or all three membranes may be glued together. They are usually injected. Usually there is considerable proliferation of fibrous tissue. The periphery of the cord is also occasionally affected.

Symptoms.—These are not well marked. Unless the nerve-roots are involved the symptoms are slight or none at all exist; however, pains of a radiating character, stiffness, tremors, hyperesthesia, herpes, and even paralyses, may occur. The course is slow, and may extend over many years.

The *prognosis* is unfavorable ultimately.

The *treatment* consists in the use of iodids and mercury internally, and the application of baths, and counter-irritation along the spine.

HEMORRHAGE INTO THE SPINAL MENINGES.

(*Meningeal Apoplexy; Hematorrachis.*)

(*a*) **Extrameningeal hemorrhage** occurs when the blood is between the dura and spinal canal.

(*b*) **Intrameningeal hemorrhage** is that in which the bleeding takes place within the dura.

Large hemorrhages are more common in the extrameningeal form; they result from trauma or rupture of an aneurysm. The peridural space will accommodate a large amount of blood without giving rise to pressure-symptoms. Caries of the vertebræ or carcinoma may cause hemorrhage by erosion and rupture of a blood-vessel. The intra-meningeal form is common, and may result from meningitis, from trauma, or may occur as a complication of any of the infectious or hemorrhagic diseases. In such cases the hemorrhages are small and scattered. It may also occur in convulsive disorders or in strychnin-poisoning. Rupture of an aneurysm at the base of the brain may give rise to extensive hemorrhage, and in a case of syphilitic ventricular apoplexy in a young man I found *postmortem*, that the blood had leaked out and infiltrated the spinal meninges for some distance.

Symptoms.—When the hemorrhage is large enough to cause pressure, the symptoms are very acute, apoplectiform indeed, but consciousness is preserved. Generally, however, they are quite indefinite. In any case they depend upon the degree and location of the compression. At first they are irritative—viz. hyperesthesia, paresthesia, neuralgic pains that are radiating in character, herpes, muscular irritability, tremors, or contractions. Later, paralytic symptoms may develop, as anesthesia and bladder- and bowel-symptoms, girdle pains, or, when the lesion is high up, interference with respiration, and pupillary changes.

The **diagnosis** is often difficult, unless the onset is sudden and explosive.

The **prognosis** depends on the cause and extent of the hemorrhage. If small in amount, absorption is usually prompt, with little or no disturbance of function remaining.

The **treatment** consists of rest, ice to the spine, counter-irritation, wet- or dry-cupping, leeches or venesection, ergot, opium or gallic acid internally, and later the iodids and electricity. Hypodermic injections of gelatin have recently proven of value in cases of persistent internal hemorrhage, and should be tried if there is any evidence that the effusion of blood continues. In certain cases operative procedures, with a view to removing the clot, may be justifiable.

DISTURBANCES OF CIRCULATION IN THE CORD.

THESE include qualitative and quantitative changes in the blood, and morbid conditions of the vessel-walls.

The blood-vessels may be the seat of peri- or endarteritis, and rarely miliary aneurysms may develop. Embolism and thrombosis also occur, the former much less frequently than the latter, which is prone to follow sclerotic changes in the vessels, giving rise to ischemia and ultimately to softening.

Congestion.—We are justified in noticing this as a possible cord-lesion, but it is questionable if it has any clinical significance. It is safe to assume that it occurs in the general stasis of circulatory disorders, yet no characteristic symptoms develop. It is very rarely met with *postmortem*.

Anemia.—This condition, like the preceding, rarely gives rise to symptoms. Dr. William A. Hammond has described a certain group of symptoms as due to spinal congestion, and another to spinal anemia, but his teachings on this point are not generally accepted. Simple anemia of the cord, *per se*, cannot be recognized clinically. Recently Lichtheim, Petren, Nonne, and others have called attention to the fact that in certain cases of profound anemia, and in some of the profound cachexiæ, nervous symptoms are present. These consist of weakness of the lower extremities, incoördination, and usually increased knee-jerks, although the latter may be diminished or absent. The lesions found in these cases are those of combined sclerosis, sometimes with, sometimes without, involvement of Lissaeur's zone.

Treatment.—Comparatively little can be done in these cases by drugs. Even if the anemia can be benefited by the usual methods, the spinal symptoms continue. The ataxia, however, can often be greatly improved by the Frenkel exercises. The danger of causing a polyneuritis by a too prolonged administration of arsenic should not be forgotten.

HEMORRHAGE INTO THE SPINAL CORD.

(*Hematomyelia; Spinal Apoplexy.*)

THIS is a very much less frequent occurrence than cerebral hemorrhage. It is usually due to traumatism, but may follow cold or exposure or some severe strain or over-exertion (in the latter probably only when the vessels are atheromatous). Hemorrhage may occur in cases of myelitis, epidemic cerebro-spinal meningitis, syringomyelia, tumors of the cord, convulsive disorders, and infectious diseases; it is, however, usually small. If the hemorrhage is extensive, disruption of more or less cord-substance necessarily follows. An area may exist large enough to cause distention of the cord without rupture, and from this extravasations may take place in the cord-substance above and below. Unilateral hemorrhage may occur, the gray matter being chiefly involved. If of recent origin, fresh blood will be found *postmortem;* but if of long standing, a brown or brownish-yellow area will be noted, consisting of disintegrated blood-corpuscles, cell-detritus, and hematoidin crystals.

The *symptoms* necessarily vary according to the region involved. The hemorrhage may be sudden, giving rise to an apoplectiform onset; or gradual, with slowly increasing symptoms. There is generally a backache, followed by paralysis, loss of sensation and of the reflexes, and in some cases loss of control of the bladder and bowel. In less grave cases the early symptoms will be those of irritation, while later paralytic symptoms supervene. If the hemorrhage is slight, absorption soon takes place, with complete recovery; but quite often more or less paralysis remains. Myelitis develops in some cases, the patient growing progressively worse and dying of exhaustion. Dr. C. E. Riggs has reported a rather unique case in a woman forty-five years of age, who developed paraplegia after a nervous shock three years before coming under his observation. When he first saw her she had impaired sensation of the lower limbs and of the trunk as far up as the xiphoid cartilage. The legs were spastic, with increased reflexes. She had neither lancinating pains nor ataxia, but was profoundly anemic, and grew progressively worse until death ensued from exhaustion. *Postmortem*, an area of extravasated blood was found in the mid-dorsal region of the spinal canal, and hardening degeneration was noted in the anterior and crossed pyramidal tracts, direct cerebellar and posterior columns, and in Lissauer's tract. The degeneration extended from the first cervical to the fifth lumbar vertebra. This case was remarkable—first, from the fact that the hemorrhage of the cord was due to anemia; secondly, on account of the extent of the degeneration, and particularly because of the fact that Lissauer's column was involved.

The *diagnosis* is always difficult, for when of sudden onset, unless aided by the etiology, it will be impossible to diagnose the condition from spinal meningeal hemorrhage. In other cases it must be differentiated from myelitis and multiple neuritis.

Treatment.—Rest, ice locally, and the internal use of ergot and opium make up the treatment.

ACUTE MYELITIS.

(*Myelitis; Acute Diffuse Myelitis; Transverse Myelitis; Spinal Malacia.*)

Definition.—An inflammation, with softening, of the cord, giving rise to various groups of symptoms depending upon the region or regions involved, and not, therefore, as constant in its symptomatology as the systemic nervous diseases (tabes dorsalis, lateral sclerosis).

Pathology.—The cord may present little or no change to the naked eye, or in the most acute cases it may be diffluent. Between these extremes many grades exist in which the pia will be found congested and adherent, the cord being more or less ingested and areas of softening, and even cavities, being found. Three forms of softening are spoken of by some writers—the red, yellow, and gray—depending upon the predominance of blood, fat, or connective tissue respectively. The *postmortem* finding depends upon the duration of the disease; the more chronic the course, the greater the amount of nervous connective tissue (neuroglia), and in consequence sclerosis will be the predominant feature. The nerve-cells and fibers are found in various stages of disintegration, the former being swollen, vacuolated, granular, and their processes broken and in many cases missing; while the latter swell, the myelin breaks up, undergoes fatty change, and is removed, and the axis-cylinders finally break up and disappear. A single area of degeneration may exist centrally, in one half of the cord, transversely, or many localized or widely-disseminated areas may be found; but above and below all of them will be found degenerated fibers—ascending and descending degeneration—due to a solution of continuity between the cell-body and its axis-cylinder process.

Etiology.—Myelitis may follow exposure (especially in alcoholics), the infectious fevers (chiefly measles and small-pox), and it may be due to traumatism or disease of the vertebræ (caries, malignant disease). Syphilis is also said to cause it, though it may only act as a predisposing agent. It has also been described as following peripheral neuritis, ascending neuritis, and we meet with some cases in which pregnancy seems to act as the predisposing cause. Embolism and thrombosis may rarely cause it. It is most common in males, generally from fifteen to thirty years of age.

Symptoms.—These will vary according to the seat and extent of the lesion. In the most acute form the course of the disease is quite rapid, reminding one of hemorrhage into the cord or membranes; the onset, however, is not so explosive, and, though rapid, it is not sudden. It is most apt to follow cold or exposure. The most acute case I have ever seen occurred in an alcoholic who had lain out one night in a drunken stupor. There may be chills and fever, malaise, backache, pains in the limbs, and, rarely, convulsions; quite often, however, there is no warning. Motor weakness develops, and is rapidly followed by paralysis. Some irritative sensory symptoms appear, as hyperesthesia and paresthesia, and then more or less complete anesthesia supervenes. The reflexes are generally lost; there is incontinence of urine and feces, and bed-sores and cystitis develop with frightful rapidity. The temperature now rises to 105° F. (40.5° C.) or even higher, and typhoid symptoms, exhaustion, and death close the scene. I have seen a case that developed in a woman a few days after delivery and proved fatal in six days.

Acute transverse myelitis is the type most frequently met with, however, the lesion being generally situated in the dorsal cord. The constitutional symptoms marking the onset are more pronounced than in the previous type and are of longer duration; but they are much less pronounced in the later stages. They are apt to simulate a rheumatic attack, with malaise, fever, muscular pains, anorexia, chills, and possibly sweating. In from a few days to a week spinal symptoms reveal themselves, the motor generally appearing before the sensory symptoms, though they may be contemporaneous, or the sensory symptoms may even appear first. In any event, they are apt at first to be irritative. The limbs will feel tired and heavy and drag in walking, and tremors or twitching occur, even cramps, and later paralysis, partial or complete, in the region involved. The lower limbs may alone be involved, or when the lesion is in the cervical region paralysis and atrophy of the upper with a spastic condition of the lower extremities may develop. The breathing is generally diaphragmatic in cases in which the intercostal muscles are involved. If the lesion is still higher up, death will quickly take place from failure of respiration. Such cases, however, are more apt to occur in the type known as *disseminated myelitis*, in which bulbar symptoms are prone to appear. The sensory symptoms at first are those of a tingling or burning character, or formication. Later, certain or all forms of sensation may be lost, and, roughly speaking, the upper level of anesthesia corresponds to the level of the cord involved. This "boundary region" is apt to be hyperesthetic, and in it the "girdle-feeling" is experienced. The reflexes may be lost at first, but soon return, and become exaggerated below the lesion. The condition of the trunk-reflexes may enable one to locate the position of the cord-lesion. There is not much wasting of muscles, as a rule, nor does the reaction of degeneration develop, unless the lesion is in the lumbar or cervical cord, when both will occur. Loss of control of the bowel and bladder may be among the earliest symptoms, though this is not the rule. While superficial ulceration may occur in any neglected case, the most marked trophic changes take place in those in which the lumbar cord is involved, either directly or by extension. In such cases, despite the most assiduous attention, extensive bed-sores develop. The course of the disease depends on the cause and the extent of the lesions. Death may occur in a few weeks from exhaustion, heart or respiratory failure, or from kidney-disease secondary to cystitis. Recovery is the rule, though with more or less permanent damage due to degeneration of some of the paths of conduction.

Diagnosis.—The distinction from hemorrhage into the cord or membranes has already been mentioned. From Landry's paralysis it can be separated by a reference to the subjoined table:

ACUTE MYELITIS.	LANDRY'S DISEASE.
Paralysis is sudden and generally becomes complete.	Paralysis begins in the feet and rapidly spreads to the muscles of respiration and deglutition.
Wasting and bed-sores are marked.	Trophic disturbances are absent.
Reactions of degeneration are distinct.	No reactions of degeneration.
Early involvement of the sphincters.	Bladder and rectum are not involved.
Girdle-pains sometimes mark the height of the lesion.	Girdle-pains are absent.

Anterior poliomyelitis is not accompanied by sensory symptoms. In *peripheral neuritis* pain of a shooting character is more apt to be present, and is almost invariably the first symptom to appear. Motor symptoms may not appear for some days. This is not the case in myelitis. In *compression of the cord* sufficient collateral evidence can usually be obtained to differentiate it from myelitis. *Hysteric paraplegia* is occasionally misleading. The character of the patient and the previous history should be thoroughly considered; moreover, in this form there are no trophic changes, and as a rule no bladder-symptoms; at any rate, there is no cystitis. Retention of urine may occur, but not incontinence. The diagnosis of myelitis can usually be made without great difficulty from the motor and sensory symptoms, the preservation of the knee-jerk, the vesical, rectal, and trophic symptoms, and often from the presence of the girdle-sensation in addition.

Prognosis.—The most acute cases are fatal in from three days to a week. Less acute cases generally recover with more or less loss of motor power.

Treatment.—Very little can be done to arrest the process in acute myelitis. Absolute rest should be enjoined, and the patient given a nutritious liquid diet with free stimulation. An ice-bag may be applied to the spine. The patient should be placed on an air- or water-bed. Trophic changes should be looked for daily, and at the first sign of their appearance alcohol or some stimulating liniment should be employed. If the skin is broken, absolute cleanliness must be observed, and the wounds dressed antiseptically. It is well, also, to change the patient's position from time to time to avoid too long-continued pressure in any one spot. Ergot or ergotin should be given internally, and, especially in specific cases, potassium iodid. A general tonic and supportive treatment is indicated, and later massage, electricity, and baths.

CHRONIC MYELITIS.

THAT there are both a subacute and a chronic form of myelitis is generally conceded, though these types are not sharply circumscribed. As has been previously mentioned, it is quite likely that many cases exist in which the clinical symptoms do not seem to warrant the diagnosis of myelitis, and yet extensive areas of degeneration may be found *post-mortem*. Even some cases of supposed hysteria may have a distinct pathology.

Pathology.—The lesions are most apt to be disseminated or diffuse, though there may be a single focus. Histologically, the chief differences from the acute variety consist in the greater amount of sclerosis, the thickened blood-vessels with contracted lumen, and an entire absence of recent hemorrhage. In some cases also the pia is much thickened in patches and firmly adherent. The nerve-cells are either seen to be in advanced stages of degeneration or they have actually disappeared. Secondary degenerations, above and below, proceed from the primary foci.

Etiology.—This is not clearly known; an acute attack may terminate and the tissue of the cord become sclerosed with persistence of the symptoms; or the disease may commence insidiously as the result of the existence of some chronic infectious process, such as syphilis; or it may be the sequel of an acute infection, such as typhoid fever.

Symptoms.—Any symptom occurring in the acute may be duplicated in the chronic form, though the onset of the latter is gradual. The symptoms are more or less obtrusive according to the region of the cord that is affected. If the cervical and lumbar regions are not implicated, no definite symptoms will be present, and probably there will be nothing more than subjective sensations and progressive weakness, with possibly some muscular wasting. The most characteristic features of a well-marked case are the irregular and successive involvement of various parts. There will be motor weakness, possibly of an arm, followed sooner or later by sensory impairment. Then one of the lower extremities may become involved, and ultimately paralysis will supervene. When the lesion is single this irregular onset is less apparent. In chronic transverse myelitis of the lumbar region, for instance, there will be paresis of the lower extremities, simultaneously or successively involved. The onset, however, is gradual, and months may elapse before the paraplegia will be complete. A girdle-sensation is apt to be present, together with lowered sensibility, and loss of sensation is very rarely absolute. The knee-jerk is increased, ankle-clonus is present, and in time the muscles become spastic. The sphincters are frequently implicated. Atrophy of the muscles is most pronounced when the anterior gray matter of the cervical or dorsal region is involved, but this may occur in any case. The reactions of degeneration can rarely be elicited.

Diagnosis.—The gradual, and in many cases the irregular, onset characterize this disease. In its various phases it may simulate almost any spinal-cord disease, and it is most apt to be confounded with tumor, pressure (carious or malignant), primary lateral sclerosis, progressive muscular atrophy, and syringomyelia. *Pressure*, whether due to a tumor, to caries, or to malignant disease, is apt to cause pain radiating in character, and the last two usually present collateral evidences in the deformity and cachexia. The symptoms, too, are always bilateral, while those of myelitis may be unilateral. From *progressive muscular atrophy* it may generally be diagnosed by the irregular course it pursues. Apart from the painless ulcerations and the dysesthesia that usually occur in *syringomyelia*, it may be impossible to diagnose it from the latter disease.

The **prognosis** is necessarily grave. Recovery may be possible, but it is extremely rare. The process, however, may be arrested in some cases, and the strictly focal forms are less apt to prove fatal than the disseminated or diffuse.

Treatment.—More can be expected from general hygienic measures than from the use of drugs. In the early stages rest is indicated, but it is well also to employ passive exercise, to prevent, if possible, a too great contraction of the muscles. As soon as expedient—each case being judged on its merits—the patient should be taken out of doors. Change of air and of scene is advisable, as are also baths and massage. Mild counter-irritation may be applied to the spine, but care should be taken to avoid the areas of anesthesia. General tonics, iron, quinin, arsenic, and strychnin, should be given, also mercury or the iodids. The greatest possible care of the bladder should be taken in order to avoid cystitis.

ANTERIOR POLIOMYELITIS.

ESSENTIAL PARALYSIS OF CHILDREN.

(*Atrophic Spinal Paralysis.*)

Definition.—A febrile disease of more or less rapid onset, associated with muscular paralysis and atrophy, occurring chiefly in children, and most frequently in those under three years of age.

Pathology.—The condition is generally unilateral, and is a true focal myelitis; hence we find congestion, softening, and even cavity-formation. Microscopically, the chief feature observed is the destruction of the multipolar ganglion-cells of the anterior horn. If the examination is not made until months or years have elapsed since the onset, the condition will be about as follows: More or less asymmetry of the cord in the region affected, with sclerotic changes at the site of the lesion, and probably in the pyramidal tract also. The anterior nerve-roots of the same side will be found atrophied, and the muscles wasted, having undergone fatty degeneration and fibrous change.

Etiology.—The precise cause is not known, but the following predispose to the affection—viz. age, exposure, acute diseases (particularly those known to be infectious), and warm weather. The disease may occur at any age, but by far the greatest number of cases occur before the third year of life; they are about equally distributed between the two sexes. Later in life the condition is more common in males, chiefly between the ages of ten and twenty-five. It is rare after this period. Epidemics have been described, and, notably, one occurring during the summer of 1894. Dr. Caverly of Rutland, Vt., then reported 126 cases occurring in Otter Creek Valley, a limestone region of Vermont. At the same time domestic animals—horses, dogs, and hens—were affected with a paralytic disease, this fact still further supporting the idea of an infectious origin. A similar epidemic has occurred in Ohio.

Symptoms.—The onset is generally acute, and may be sudden. Constitutional symptoms are present as a rule. Generally, the sequence is as follows: Fever (usually slight), malaise, possibly vomiting (especially in children), muscular twitching, headache, and restlessness. In a few hours, or after one or two days, paralysis supervenes and quickly spreads, involving a greater or less area; it then remains stationary for from two to four days to from five to eight weeks, when improvement takes place, beginning in the part last affected. In some cases, after a most trifling indisposition over night, paresis is met with in the morning. In a few weeks only that portion remains paralyzed that is to be permanently damaged. Wasting of the muscles will be noticed a week or two after the onset of paralysis; these become flaccid and give the reactions of degeneration. Sensory symptoms are very rarely present—so seldom, indeed, that they need not be reckoned with. The reflexes are lost, both superficial and deep, and later contractures develop and result in various deformities. The growth of bone is seriously impaired in some cases. Complete recovery rarely takes place, nor is it to be expected when we consider the destruction of the neuron-body.

Diagnosis.—Usually this is not difficult, except, possibly, for the first few days in some cases. Close scrutiny will enable one to differentiate between this disease and a pseudo-palsy the result of pain on active or passive motion, as seen in rickets, scurvy, and in hip-joint disease.

Prognosis.—Some impairment of motion and more or less wasting of the muscles almost invariably remain. Danger to life, however, is very remote, though the subjects of infantile paralysis are predisposed to intercurrent affections, since their natural degree of resistance is lowered. The more rapid the loss of faradic irritability the less the extent of recovery.

Treatment.—There can be no longer doubt of the infectious nature of the disease, but unfortunately there is no specific treatment. During the acute stage a brisk calomel purge, followed by a saline, is of benefit; and it is necessary to support the general condition. For this reason absolute rest should be enjoined; the diet should be liquid and nourishing; and stimulants should be given freely if necessary. Pyrexia should be met with cold, an ice-bag may be applied to the head and spine, and cold sponges used if necessary. The coal-tar antipyretics should never be used. The affected parts should be wrapped in cotton. As soon as possible the child is to be taken into the fresh air. It is of vital importance to keep up the general systemic tone, and hence the necessity for fresh air, change of scene, and for nourishing but easily digestible food. During this period massage and electricity should be employed, together with the administration of strychnin. In the later stages, when contractures have set in, mechanical appliances may be necessary to correct deformity and to give support.

ACUTE, SUBACUTE, AND CHRONIC POLIOMYELITIS IN ADULTS.

1. **Acute atrophic spinal paralysis of adults**, as the acute form is called, has essentially the same symptomatology as the corresponding disease in children, except that the onset is apt to be more pronounced. Convulsions, however, scarcely ever occur. When pain is a prominent symptom we should be guarded in making a diagnosis. Initial pain is significant of a nerve-lesion, particularly if sensory disturbances can be found, and such cases would indicate a neuritis and not a poliomyelitis. Presumably the incidence of the poison has been on the axon, and not on the neuron-body, this view being consonant with the complete recovery that is sometimes seen in adults. When true poliomyelitis has existed complete recovery probably never occurs.

2. The **subacute form** has been described by Duchenne as "*paralysis général spinale antérieure subaiguë.*" It comes on, as a rule, without apparent cause, and the initial symptoms are very slight. In a few weeks failure of power is noticed in the limbs and paralysis gradually supervenes. After lasting for some time partial recovery follows, the paralysis and muscular atrophy remaining in a limited region only.

3. That **chronic poliomyelitis** exists has been proved by Oppenheim and other observers; yet it is probable that most cases described under this heading have been due to peripheral and not to central lesions. In neuritis, however, the paralysis is either unilateral or bilateral, and in the latter case it is symmetric, differing in this point from the irregular distribution of centric disease. Pain is common, and there is also tenderness along the nerve-trunks as a rule.

Recovery from neuritis may be perfect; at all events, it does not present the tendency that is met with in poliomyelitis to clear up perfectly, except in a limited area.

Treatment.—The general line of treatment that I have given for the infantile type is equally applicable in these forms. Ergot and belladonna may be used in the early stages, and, later, mercury or the iodids in small doses. Electricity and massage are of the greatest value.

ABSCESS OF THE SPINAL CORD.

It is rare for inflammation of the cord to give rise to pus, yet a few cases have been described. The suppuration is necessarily micro-organismal in origin, and as a rule is either due to some septicemia or traumatism, or secondary to purulent meningitis. The *symptoms* are those of myelitis, but may be masked by any associated condition.

UNILATERAL LESION OF THE SPINAL CORD.

(Brown-Séquard's Spinal Paralysis.)

THIS is not a distinct disease, but rather a grouping of certain symptoms, first studied by Brown-Séquard,[1] and hence bearing his name. It is met with particularly as a result of injuries (knife-thrusts and the like), though it may also be due to tumor or caries of the cord, to syphilis, or to any process causing compression of one-half of the cord. Such lesions intercept the motor impulses of the same side; the fibers having crossed in the medulla, the sensory fibers cross in the cord soon after entering, and hence sensation will be absent on the side opposite to the lesion (*vide* Fig. 72). A lesion in the cervical cord above the arm-nuclei causes motor paralysis of both arm and leg of the same side (spinal hemiplegia) and sensory paralysis on the opposite side. If in the dorsal or lumbar cord, the leg on the corresponding side is paralyzed, while that of the other is anesthetic. Lesions are seldom strictly confined to one side of the cord, but overlap a trifle, so that there is apt to be some loss of power on the anesthetic side; this, however, may be due to the redecussation of a few motor fibers at a lower level. The side of the lesion is hyperesthetic—a fact for which no satisfactory explanation has ever been advanced. Muscular sense is diminished or lost on the same side. Above the hyperesthetic region an area of anesthesia commonly exists, and above this, again, an area of hyperesthesia. The reflexes are increased on the side of the lesion (inhibition being removed), and the temperature of that side is usually higher. On the anesthetic side the motor power, reflexes, muscle-sense, and temperature are all normal. Sometimes the sensory symptoms are limited to loss of pain and temperature sense.

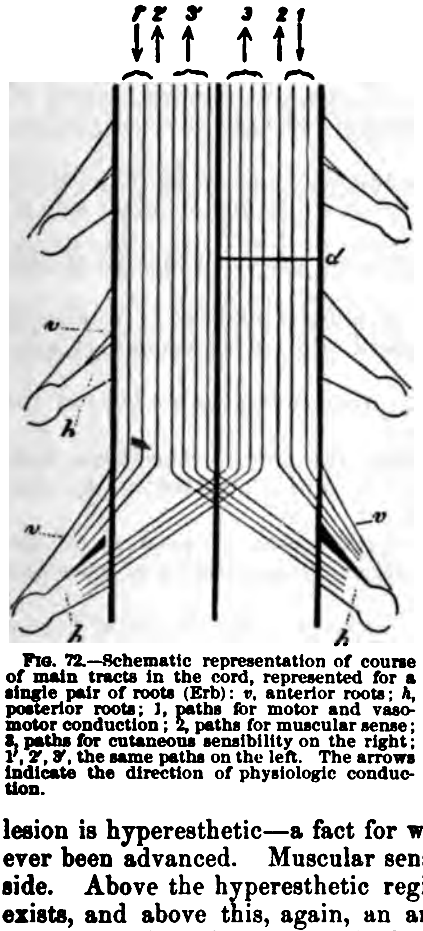

FIG. 72.—Schematic representation of course of main tracts in the cord, represented for a single pair of roots (Erb): *v*, anterior roots; *h*, posterior roots; 1, paths for motor and vaso-motor conduction; 2, paths for muscular sense; 3, paths for cutaneous sensibility on the right; 1', 2', 3', the same paths on the left. The arrows indicate the direction of physiologic conduction.

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[1] *Med-Chir. Trans.*, 1889.

SEGMENTAL LESIONS OF THE SPINAL CORD.

The following table, adapted from Wichmann, gives the principal symptoms for lesions of the segments of the spinal cord:

Cervical cord: paralysis of the trunk and lower extremities, loss of sensation bounded by a horizontal line at the level of the lesion.

C. 1–4. Death as a result of paralysis of the diaphragm.

C. 5. Complete paralysis of the arms, including the deltoids. Anesthesia from neck down.

C. 6. Slight flexion and elevation of arm still possible (paresis of deltoid and biceps), hyperesthesia instead of total anesthesia on the posterior surface of arm and forearm.

C. 7. Paralysis of forearm except thumb, which is paretic, paresis of arm. Anesthesia below line at level of axilla and on ulnar side of arm and forearm.

C. 8. Paralysis of small muscles of hand, paresis of muscles of arm and shoulder. Anesthesia as above.

D. 1. Paralysis of trunk, paresis of muscles of hand. Anesthesia from level of first dorsal spine and third rib. Hyperesthesia of arm. Disturbances of the pupil (myosis).

D. 2–12. Paralysis and anesthesia corresponding to the level of the lesion.

L. 1. Paralysis of the legs including the psoas. Anesthesia from level of first lumbar spine and anterior superior spines of the ilia. Reflexes absent or increased.

L. 2. Paralysis of the legs except the psoas. Anesthesia of the anterior and posterior surfaces of the thighs, hyperesthesia of inner and outer sides. Reflexes lost.

L. 3. Paralysis of the legs, of the rotation of the thighs, of flexors of legs; paresis of the other muscles. Anesthesia of the sacral region, of the genitalia, of the posterior surface of the thighs, and of the legs. Reflexes lost.

L. 4. Paralysis as above, except internal rotation of thigh is possible. Anesthesia as above, except inner surface of thigh is hypesthetic.

L. 5. Paralysis of biceps, glutei, flexors of toes, paresis of other muscles. Anesthesia as above.

S. 1. Paralysis of rectum and bladder; paresis of muscles of legs. Anesthesia of sacral region, genitalia, and posterior surface of thigh; posterior and external surface of the legs, and external half of the foot.

S. 2. Paralysis of the muscles of the anus and of the bladder. Paresis of the legs. Anesthesia of sacral region, genitalia, and posterior surface of thigh.

S. 3. Paralysis as above, no paresis of legs. Anesthesia of sacral region, region of anus, and posterior surface of scrotum and penis (or of labia).

S. 4. Paresis of sphincter ani and bladder. Small anesthetic area over sacrum and about the anus.

S. 5. Small area of anesthesia over the coccyx.

LOCOMOTOR ATAXIA.

(*Tabes Dorsalis; Posterior Sclerosis.*)

Definition.—A systemic sclerosis affecting the posterior columns of the cord. In many cases foci of degeneration occur in the basal ganglia. The disease is characterized by a loss of coordination, absence of the knee-jerk, fulgurant pains, and the Argyll-Robertson pupil.

Pathology.—Macroscopically, it may be observed—1. That the posterior roots are more or less atrophied and grayish in color.

2. There is a thickening and adhesion of the spinal membranes, with some degree of congestion, particularly noticeable in the posterior region (not a constant change).

3. There is a slight change in the shape of the cord, and the affected regions assume a grayish tint. Change of color is well seen after the cord is hardened. Microscopically, degeneration of the peripheral sensory nerves will be found in certain cases to be more marked at the periphery and to diminish as the main trunks are reached. Rarely, changes in the motor nerves will be met with also, but only in cases in which the anterior horns are affected. The spinal ganglia are usually normal.

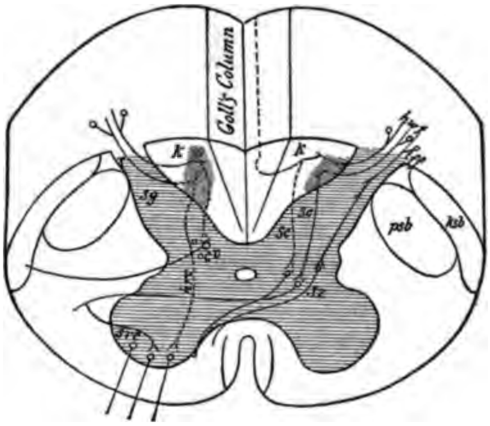

FIG. 73.—Diagram of primary degeneration-areas and secondary degeneration of the fibers in the beginning stage of tabes (Leube): *psb*, pyramidal tract; *ksb*, cerebellar tract; *hwf*, posterior root-fibers; *lf*, lateral entrance of delicate root-fibers; *k*, area of earliest degeneration; *r*, marginal zone; *sg*, substantia gelatinosa; *cv*, Clark's columns; *i*, anterior zones (remaining free); *sc*, sensory collateral fibers; *hrc*, collateral reflex of posterior column; *src*, collateral reflex of the lateral column; ———, healthy fibers; ------, degenerated fibers.

4. There are degenerative changes in the posterior, and occasionally in the anterior roots (*vide* Fig. 73).

5. Cord-changes are present, consisting in the early stages of a degeneration of the fibers of the Spitzka-Lissauer column, of the post-root zone of Charcot, of the fibers going to the column of Clark, and of the comma tract. As the disease progresses more and more of the posterior columns—Goll and Burdach—is involved with the fibers of Gower's

column, the intermedio-lateral tract, and even the direct cerebellar tract. This latter is only affected, however, when the cells of Clark's column are involved.

While the chief incidence of the poison, whatever this may be, is upon the nerve-fibers, yet we do meet with cases in which the posterior root-cells are diseased; as already stated, the cells of the anterior horn may be diseased also. There is an overgrowth of neuroglia that takes the place of the degenerated fibers, and when the membranes are thickened the strands of connective tissue dipping into the cord take on added growth.

6. There are cerebral and medullary changes. There may be some change in the nuclei of the columns of Goll and Burdach and in those of some of the cranial nerves. In addition to changes in the nervous system, certain cases present some morbid condition of the osseous system, consisting of erosion of the interarticular cartilages and atrophy and absorption of the bony articulating surfaces.

Etiology.—*Race.*—White races are more susceptible than negroes; and the disease is less frequently met with among the Jews than among other white classes. *Sex.*—Males are more liable to the disease than females, in the proportion of 10 to 1. *Age.*—Most common between the ages of thirty and forty. *Syphilis.*—Since Fournier in 1875 first pointed out the relationship between these two diseases, the opinion has steadily gained ground, despite the view of Leyden and other German authorities, that a large majority of tabetic cases (observers differ as to the proportion) have an antecedent history of syphilis. It must be clearly borne in mind that locomotor ataxia is not syphilis of the cord and brain, but a distinct entity, in most cases of which, however, syphilis stands as a predisposing factor. It will be remembered that in the description of anemia of the cord, lesions resembling those of tabes are found as a result of various toxemias, and it was suggested that this might throw some light on those cases in which no syphilitic history can be obtained. Exposure and sexual excess are possible factors; likewise traumatism. Alcohol is said to cause tabes, but this is very doubtful; it may certainly give rise to pseudo-tabes, the peripheral form. In England, Gowers has noted that locomotor ataxia occurs more frequently among urban than among rural populations.

Symptoms.—These may be grouped into various stages: the prodromal, preataxic, ataxic, and paralytic. The *prodromal stage* may extend over a number of years, the symptoms are slight and often make very little impression upon the patient. They consist of occasional pains, usually in the legs, of transient disturbances of the ocular muscles leading to ptosis, diplopia, etc., occasionally of slight diminution of vision, most noticeable at night, and of more or less pronounced impairment, very rarely exaltation, of sexual power. The symptoms become characteristic only in the *preataxic stage.* The pains in the legs become more frequent and assume the typical fulgurant or lancinating type; that is, a stabbing or boring sensation, shooting along the limbs and lasting for a brief interval of time. The pupils give the Argyll-Robertson phenomenon (failure to contract to light), there is permanent myosis, nyctalopia, and the paresis of the eye-muscles may be still present or may have disappeared. There is usually some disturbance of motion, chiefly manifested at night, and ataxia may be revealed by the finer tests (having the patient hop backward on one leg). The patella reflexes are diminished or absent. There is now distinct impairment of sexual power and difficulty in urination. The sensory symptoms belong to this and the following stage.

The dominant symptom of the *ataxic stage* is the incoördination of movement. This gives rise to the *ataxic gait.* The legs are kept far apart and are lifted higher than is necessary from the ground, they are brought down violently, and the gait is of a peculiar stamping, irregular, slightly staggering character. Ataxia of the arms occurs later and is manifested by difficulty in grasping objects or in accomplishing finer coördinated movements. Ataxia of the lower extremities may be tested by directing the patient to touch with his toe an object held above it, or, when lying down, to place the heel of one foot upon the knee of the other; of the upper limbs by directing him to touch rapidly the tip of the nose with the forefinger, or to spread the arms apart and bring the forefingers rapidly together. Loss of station, or *Romberg's symptom,* is tested by directing him to stand with the feet close together and to close the eyes. The swaying of the body will vary from several inches in either direction to falling over. The reflexes are now completely abolished, and there is often some wasting of the muscles. The sphincters are involved, there is often difficulty in voiding the urine, associated with incontinence, and as a result of careless catheterization cystitis is often acquired.

The *sensory symptoms* are various: in addition to the fulgurant pains, there are visceral crises, characterized by sudden severe pain and disturbance of function. The most common seat is the stomach, and the crises are associated with vomiting of acid material. Crises may also involve the larynx, liver, kidneys, clitoris, and kidneys. Diminished sensation affects the organs, nerves, and areas of the skin. There is loss of sensation in the testicles or breasts, and severe blows in the pit of the stomach cause no distress. *Biernacki's symptom,* loss of sensation in the ulnar nerve, is present. Areas of anesthesia or hyperesthesia, usually segmental in type, can be detected upon the trunk or less frequently upon the extremities. Astereognosis, or the loss of the ability to recognize objects, may be present on one or both sides, or the stereognostic sense may be impaired. The *girdle pain* is a feeling of constriction that may be very uncomfortable about some part of the trunk. The *paralytic stage* inaugurates the termination of the disease. Locomotion becomes impossible or can only be accomplished with the aid of two canes, loss of control over the bladder is complete, the patient is querulent or even demented, and muscular wasting and bed-sores may appear. In either this or the ataxic stage the optic nerve may atrophy, and this is often associated with a remarkable improvement in the ataxia that is at present inexplicable. Death usually occurs as a result of infection, either through the bladder or lungs, more rarely as the result apparently of exhaustion.

Course.—Rarely the disease runs a very rapid course. The preataxic symptoms—pain, loss of knee-jerk, Argyll-Robertson pupil with or without ptosis and diplopia—may only exist a few weeks before incoördination develops. The latter will then reach its acme in twenty to thirty days. This is very unusual, however. As a rule, the first or preataxic stage extends over a period varying from months to even as long as twenty-five years. Dr. Wm. Egbert Robertson has related to me the case of a man aged fifty-eight who for fifteen years has had fulgurant pains and an absence of the knee-jerk, but neither ocular nor any other symptoms. In some cases the first stage may be absent. The second or ataxic stage—that of incoördination—is generally slowly progressive, finally reaching a point at which it remains; rarely, more or less improvement may follow. When optic atrophy develops, ataxia either does not appear, or, having done so, fails to advance. The final stage in a few cases is only reached when the patient has become paralyzed and bedridden.

Diagnosis.—This is readily made when we have a combination of the absent knee-jerk, fulgurant pains, and the Argyll-Robertson pupil. However, the loss of knee-jerk, associated with one of the other symptoms in an otherwise healthy man, is, to say the least, highly suggestive of the disease; the addition of incoördination serves, of course, to clinch the argument.

Differential Diagnosis.—*Peripheral Neuritis.*—The symmetric distribution of symptoms, tenderness in the muscles, frequent herpetic rashes, motor weakness and wasting, pain (not fulgurant in type), greater prominence of parasthesia, absence of the Argyll-Robertson pupil, knee-jerk often increased (absent in diphtherial form, but other symptoms and history serve to distinguish it), and later, either diminished or absent, and the history of the case, are sufficient. *Alcoholic* and more rarely *arsenical* poisoning give rise to a condition closely resembling true tabes in that there is the loss of knee-jerk, often sharp pain, and incoördination, though the latter symptom is never as marked as in advanced tabes. The gait, however, is totally different, and consists of the high "steppage" gait described in the discussion of Peripheral Neuritis.

General paralysis of the insane may present much difficulty. Spinal symptoms may occur in general paresis, and conversely in certain cases of tabes symptoms of general paresis develop. Time alone will solve the problem.

Ataxic Paraplegia.—Apart from the absence of pain and anesthesia, incoördination is followed by a spastic condition. The knee-jerk is much exaggerated and the so-called ankle-clonus develops.

Cerebellar Disease.—The incoördination does not resemble that of ataxia; optic neuritis is present; also headache and vomiting appear in well-marked cases. The knee-jerk is usually present.

There are certain conditions, already described under *Anemia of the Cord*, in which lesions of the posterior columns of the cord occur. Some of them are very much like tabes, but do not present the "combination of symptoms" seen in locomotor ataxia. As a rule, the Argyll-Robertson pupil is absent, and less frequently the lightning pains also.

The crises may be mistaken for *disease of the various organs involved*. Repeated attacks of acute pain, tabetic in character, and particularly in adult males, should, however, excite suspicion, and an absence of the knee-jerk and other characteristic evidences will always be present in ataxia.

When the chief lesion is in the dorsal region the pain may be mistaken for that of *spinal caries* or even *neuralgia* or *rheumatism*. From caries it may be differentiated by the fact that in vertebral disease the pain is more or less localized, and that it is much increased by movements. Moreover, the other symptoms of ataxia are wanting—*e. g.* ocular troubles, incoördination, and absence of the knee-jerk. The latter point also holds good in cases of rheumatism and intercostal neuralgia. For the diagnosis from hereditary ataxia *vide* p. 1088.

Prognosis.—The outlook is not particularly bright. While, as already stated, the disease does not cause death, perfect recovery is never obtained. Of course the prospect is much brighter the earlier the case is taken in hand, and some improvement may be expected in most cases. The fact that the patient has had syphilis does not modify the prognosis one way or the other.

Treatment.—Rest (first suggested by Weir Mitchell) is imperative when the patient commences treatment, and especially when pain is early complained of, massage and electricity being employed meanwhile to keep up the tone of the muscles. In my opinion the rest-treatment retards the progress of ataxia more effectively than any other measure, but it cannot be used with the expectation of producing a cure. The bowels should be moved daily, and the urinary functions especially looked to. In certain cases catheterization is necessary. The patient should then be taught, first, what surgical cleanliness means; and secondly, how to use the instrument. Urotropin in doses of gr. v—0.3, three or four times daily, is a valuable prophylactic against cystitis. Counter-irritation along the spine and suspension are useless. The diet should not be heavy, and if gastric crises occur special care should be taken in this direction.

Electricity is of doubtful utility. The galvanic current is to be chosen, and Erb advises placing the medium-sized cathode over the cervical sympathetic, and the larger anode near to the spinal column on the opposite side, moving it at brief intervals in the downward direction. This method must be continued for many months. Frenkel has recently advocated the systematic education of the muscles in coördinated movements. Hydrotherapy is a serviceable measure if judiciously employed. Neither cold nor hot baths are free from deleterious effects, but tepid baths (80°–90° F.—26.6°–32.2° C.), combined with gentle friction of the body-surface, are signally useful. Among the numerous natural springs enjoying more or less popularity there are two in especially high favor—the carbonic-acid thermal saline springs of Oeynhausen-Rehme in Minden and Aix-la-Chapelle in Germany. The chief benefit may, after all, be credited to the invigorating effect of the changed environments.

The fulgurant pains, or those of the various crises, are occasionally so severe as to require bromids, codein, or even morphin, though the use of the latter agent is always to be postponed until other means are exhausted. Antipyrin or salol and phenacetin may also be tried in this connection. Heroin is often almost a specific. In any case the patient should live a simple, regular life, avoiding excesses of all kinds, and particularly sexual and alcoholic indulgences.

Recently it has been discovered (Frenkel) that the *ataxia* can be greatly improved by systematic exercises designed to train the muscles in coördinated movements. The important points are to avoid fatigue and irritation; and to increase gradually the complexity of the tasks.

HEREDITARY ATAXIA.

(*Friedreich's Disease.*)

Definition.—An hereditary disease, first described in 1861 by Friedreich. The symptoms are primarily manifested in early life, and the disease is characterized by ataxia, defective speech, nystagmus, absence of the knee-jerk, and more or less secondary deformity, as spinal curvature or talipes.

Pathology.—The postmortem findings are essentially those of locomotor ataxia and ataxic paraplegia. The spinal membranes are somewhat thickened and adherent, especially over the posterior part of the cord, and that, too, chiefly in the lumbar region. The posterior nerve-roots are generally atrophied and sclerosed. The columns of Goll and Burdach are degenerated, particularly in the lumbar region, and to a lesser extent in the cervical. Degeneration is also found in the lateral, and to a slight degree in the anterior, columns. The chief microscopic change is a marked neurogliar overgrowth, as shown by Dejerine. The nerve-cells of the cord are generally normal. Frequently the cord is abnormally small. Cerebral lesions also have been found in this disease.

Etiology.—1. Family tendency (heredity) has a strong influence. A single case, however, may develop in a family.

Age.—Most commonly the disease appears between the third and twelfth years, though it may appear earlier.

Infectious fevers (in particular) and other acute diseases frequently precede the evolution of this complaint. Trauma and many other conditions have been described as exciting causes.

Symptoms.—The earliest evidence of the disease is impaired coördination, first in the legs, and, later, in the arms; it is most marked when the eyes are closed. Attention is often called to this symptom by the fact that the child stumbles, ambles, and staggers, and cannot walk properly. The gait, however, lacks the pronounced stamp of true ataxia. Rutimeyer has pointed out that in many cases the great toes are turned upward. Some children never learn to walk. Romberg's symptom is generally present. Movements of the arms, when these are ataxic, are irregular and jerky, and jerky movements of the head may also be observed. Bilateral nystagmus develops and the speech becomes affected. At first there is a mere impediment (a stuttering), but later syllables, or even whole words, are omitted and an unintelligible jargon results. The knee-jerks are almost always absent. There is no optic atrophy, nor are any sensory symptoms present as a rule. The sphincters are not involved. There are no trophic changes in the skin or the joints, and no visceral crises. Vaso-motor symptoms—flushing, sweating—are sometimes observed. There is no mental change.

Talipes and spinal curvature are generally met with after the disease has existed for some time. In old cases muscular weakness and wasting are present, but the muscles do not give the reactions of degeneration.

The **course** is always slow. It may last for many years, thirty or even more.

Diagnosis.—Usually this is not difficult, and especially when more than one case exists in a family. The age, incoördination, shambling gait, nystagmus, scanning speech, and deformity are strikingly characteristic.

Differential Diagnosis.—*Locomotor ataxia* appears later in life, and the preataxic stage (pain, absent knee-jerk, and ocular symptoms) is generally well marked. It is absent in hereditary ataxia, nor does the latter present the sensory and visceral symptoms met with in the true form. Further, the gait is very different.

Ataxic paraplegia shows an exaggerated knee-jerk, the presence of ankle-clonus, and an absence of the ocular symptoms, nystagmus, and the scanning speech.

Disseminated Sclerosis.—Tremors are almost always present, but these are fine and never coarse as in hereditary ataxia. There may be nystagmus, incoördination, and imperfect articulation, but the cases are isolated (*i. e.* they do not run in families).

The **prognosis** is necessarily bad. The disease is progressive, though it does not kill directly. It may last thirty years or more.

Treatment.—Little or nothing can be accomplished. The same general treatment should be pursued as for locomotor ataxia.

SPASTIC PARAPLEGIA.

(*Primary Lateral Sclerosis; Spastic Spinal Paralysis.*)

Definition.—A disease of the spinal cord characterized by loss of power, contractures, exaggerated reflexes, a peculiar gait, and by precipitate micturition. Spastic paraplegia (spasm plus motor paralysis) is met with as the result of the various pathologic substrata. Any transverse cord-lesion above the lumbar region may cause motor paralysis, spasticity, exaggerated knee-jerk, and ankle-clonus. The same condition results from a lesion in any part of the upper segment, from the cortical motor cells to the terminal arborization of the axon in the cord.

It is believed that fibers of the pyramidal tracts may be primarily involved, and, since they course chiefly through the lateral cord-region, the resulting condition has been named primary lateral sclerosis. This is purely hypothetic, however, for only two uncombined cases have been found. This may be due to the fact that the disease does not tend to shorten life, and that therefore the same condition that caused degeneration of the pyramidal fibers may subsequently act on other fiber-systems. Since in the case of the lower segment it is the peripheral portion of the axon that, in many cases at least, first yields to the morbific influence, so may it be with the upper segment. In such an event, however, the degeneration would be an ascending one, and the converse of that which is usually met with in the motor tracts. Thus we see that the same clinical condition may be etiologically quite different. The following are the chief varieties:

PRIMARY LATERAL SCLEROSIS.

That this condition exists alone is questioned, as I have already stated. Von Stofella has reported a case, but no microscopic examination was made. Morgan's and Dreschfeld's case, published in 1881, seems to be the only one that may be regarded as a true type. The only pathologic change observed was in the pyramidal tracts of the anterior and lateral regions.

Etiology.—It is most apt to occur when there is a ***neuropathic family tendency***. *Age*, generally between twenty-five and forty, exerts an etiologic influence. *Exposure*, *acute disease*, and ***traumatism*** are all predisposing causes. ***Syphilis*** has been said to predispose to the condition, but if so it is rather rare.

Symptoms.—In typical cases the onset is slow. The patient complains of feeling tired, and is less capable of exertion than formerly. Weakness of the legs develops, and with it increasing difficulty in walking. Even at an early stage some rigidity of the muscles will be present when the limb is extended; later this becomes a prominent symptom. The spasm is at first of little moment. It may only be noticed in the morning. When the disease has advanced, however, it becomes pronounced, so that it may not be possible to flex the limb, or, if flexed and an effort is made to extend it, it will often spring forward like a knife-blade in clasp-like rapidity. This spasticity is often so marked that in walking, so long as the ball of the foot touches the ground, clonic contractions occur; these also appear when the individual is in a sitting posture unless his legs are extended. The gait is characteristic: the legs are stiff, and move with an evident effort, while the toes scrape the ground. In some cases the adductor spasm is so great that the legs not only cannot be separated, but are actually overlapped in walking (*cross-leg progression*). In course of time the power of walking may be lost. The flexor muscles are usually weakened. The knee-jerk is very much exaggerated, a mere tap causing a sharp, quick response. Ankle-clonus can always be elicited. Pains and other sensory manifestations are often absent, though dull and fleeting pains in the back and limbs may be complained of. The arms are frequently unaffected. The sphincters are rarely involved, and ocular symptoms do not occur, though nystagmus is occasionally present. Seguin states that the ability to retain the urine is lessened and precipitate micturition results.

The **diagnosis** is not difficult. Certain hysteric cases may occasionally simulate it very closely, but these do not present the characteristic spasticity of the true form, nor is the knee-jerk increased quite as much, and ankle-clonus is either slight or absent. Then, too, in hysteria spots of anesthesia are commonly met with. Drs. Bastian and Russell Reynolds have described "paraplegia dependent on idea," in which no hysteric element entered.

SECONDARY SPASTIC PARALYSIS.

As I have already mentioned, transverse lesions above the lumbar region (caries, tumor, sclerosis, myelitis) are followed by degeneration of the pyramidal tracts, and as a result there are weakness in the limbs, increased reflexes, and more or less rigidity. If the lesion is total the paralysis is usually flaccid and the reflexes are lost.

CONGENITAL SPASTIC PARAPLEGIA.

This condition, the symptomatology of which is practically that of the adult types previously described, is almost always the result of some injury at birth, either instrumental or due to a malposition, as first pointed out by Dr. Little and since abundantly confirmed by Spencer, Dr. Sarah McNutt, Sachs, and others. The disease is probably always due to meningeal hemorrhage. In recent cases more or less extravasated blood is always found over the central convolutions and often at the base. Later, cases show atrophy and sclerosis of the motor region, the blood having been absorbed. Nothing abnornal may be noticed for a few days or weeks, though rarely convulsions, or even bulbar symptoms, may early manifest themselves. Generally, the child is several months old when the mother first notices some impairment of movement, and not until the child tries to walk will she observe anything out of the way. The abnormality varies from a slight difficulty in walking, in which the toes barely scrape the ground, to a total inability to walk, owing to the high degree of adduction spasm. Between these extremes are various grades of talipes equinus and cross-legged progression. Sensation is usually normal. The bladder and rectum are not implicated. Some cases present evidences of impaired cerebral development—idiocy and imbecility. Some observers have also described what they believe to be an hereditary form of spastic paraplegia (notably Drs. Gee and Sachs).

ATAXIC PARAPLEGIA.

This name was given by Gowers to a condition in which spastic paraplegia and ataxia coexist, owing to simultaneous involvement of the lateral and posterior columns. The posterior root-zones escape, and hence the retained reflexes. This same morbid condition may be met with in Friedreich's disease (hereditary ataxic paraplegia), or primary lateral or posterior cases may extend and involve the posterior or lateral columns respectively. Disseminated sclerosis may possibly present the same symptoms. The type Gowers describes occurs chiefly in males of middle age. Traumatism and exposure seem to predispose to the disease, as does syphilis very rarely.

Symptoms.—These develop insidiously. The patient tires rapidly, and some impairment of the power of walking is observed. In turning quickly he stumbles, and there is difficulty in walking in the dark, or even in standing when the feet are close together. The reflexes are increased at an early date, and spasticity supervenes and is progressive, though it never becomes as marked as in uncombined lateral sclerosis. The gait is somewhat similar to that met with in locomotor ataxia, but it lacks the forcible stamp already described. When the arms are involved the same ataxia, with weakness, spasticity, and increased reflexes, is met with. Sensory symptoms are generally absent, and fulgurant pains are never present. When pain occurs at all, it is of a dull character and often in the sacral region. Optic atrophy does not occur. Nystagmus is often seen, though other eye-symptoms very rarely appear. Sexual power is lost. The sphincters are not usually involved, though retention of urine may occur. Ultimately, the case generally partakes more of the nature of a lateral sclerosis, but the features of a posterior sclerosis may rarely predominate. Mental symptoms often develop in the late stages.

The *diagnosis* is easy in typical cases. The ataxia, with myotatic irritability and spasticity in the absence of sensory and ocular symptoms, is characteristic.

COMBINED SYSTEM SCLEROSIS.

Ormerod and Dana have published valuable treatises on this subject. In 1891, Dr. James Putnam of Boston described a group of system scleroses, with diffuse collateral degeneration, occurring in enfeebled persons past middle life, and more particularly in women. He had had 8 cases, and made autopsies on 4. In the white columns of the cord he found both recent and old degenerations and disintegration of the cells of the gray matter. In 1 case he found some degeneration in the peripheral nerves. The chief symptoms were motor weakness of all four extremities, but especially the lower, with some impairment of sensation and general muscular wasting. In 3 cases there was an exaggerated knee-jerk with ankle-clonus; in 1 lancinating pains, and in another incoördination. The fatal cases ran a course of three or four years. Several of them showed lead in their urine, and Putnam thinks that this may have been an etiologic factor in some instances.

The *symptoms* of combined sclerosis partake of the nature of locomotor ataxia and spastic paraplegia, but are less marked than either of these diseases. The onset is slow, there is more or less incoordination, and Romberg's symptom can be elicited as a rule. There is loss of motor power, and the sensory symptoms are slight. There may be dull sacral pain. Optic-nerve atrophy very rarely occurs, though there are certain eye-symptoms. The reflexes are generally exaggerated, and "ankle-clonus" is present.

The *diagnosis* is based upon the presence of paraplegia with increased reflexes, associated with sensory symptoms—paresthesiæ—and rarely pain.

REFLEX PARAPLEGIA.

Since this was at one time so warmly put forward by Brown-Séquard as a distinct entity, it seems justifiable to speak of it, though in the light of our present knowledge we are not disposed to give it any nosologic distinction. It was supposed to be due to anemia of the cord, and to be the result of irritation reflected from a sensory nerve to vaso-motor nerves. The so-called "urinary paraplegia" was included in this category.

INTERMITTENT PARAPLEGIA.

Romberg was the first to call attention to this condition. His original case was that of a woman aged sixty-four, in whom paraplegia developed suddenly with involvement of the sphincters. The sensations were normal. In about twenty-four hours she was so much better as to be able to walk; micturition was normal, but there was some weakness. Next day, however, the paraplegia returned. These attacks, with almost normal intervals assuming a periodic character, induced him to give quinin, which he did. Recovery was the prompt result. Erb and others have since reported cases, but it is now believed that they are due to involvement of the peripheral nerves rather than of the cord.

Treatment of Spastic Paraplegia.—In general the treatment is the same as that of locomotor ataxia. This is especially true if syphilis is suspected. Little can be done, as a rule, for the disease is usually progressive in spite of all treatment. Belladonna or hyoscin seems to lessen the spasm in some cases. Attention should be given to the bladder and bowel, particularly to the former. In the congenital form operative measures are often requisite to overcome deformity.

MULTIPLE SCLEROSIS.

(*Insular or Disseminated Sclerosis.*)

Definition.—A disease due to the development of sclerotic patches, occurring in an irregular manner throughout either or both the brain and spinal cord. It is characterized by paresis, intention-tremors, scanning speech, and mental disturbances.

Pathology.—The sclerotic tissue occurs especially in the white matter, though any part of the cerebro-spinal axis may suffer. The cortex is rarely implicated. The spots are usually well circumscribed, gray or grayish-red in color, and on section may be level with, raised from, or depressed beneath the normal line of section according as to whether it is in the early, hypertrophic, or cirrhotic stage. The cranial nerves may be involved at their origin, the first, second, and tenth being particularly vulnerable. The medullary sheath of nerve-fibers in the affected region degenerates early, but the axons are markedly resistant.

Since they are not cut off from their trophic center, secondary degeneration is rarely met with. The blood-vessels show more or less proliferation of the adventitia, and endarteritis is not an uncommon condition. Whether this vascular change is primary or secondary is unknown. Microscopically, the sclerotic areas are made up of an overgrowth of neuroglia-cells and fibers and of the ordinary connective tissue. In certain cases these patches exhibit some tendency to involve special parts of the nervous system, as the lateral or posterior columns.

Etiology.—There is no definite and known etiologic factor. Among the possible predisposing causes may be mentioned emotions, trauma, heredity, exposure, infectious and exhausting diseases of any kind, and perhaps hysteria. It is important to remember, moreover, that it is frequently impossible to diagnose this disease in its early stage from hysteria. This point is dwelt upon particularly by Buzzard and Bastian, and many cases of supposed hysteria have subsequently proved to be cases of multiple sclerosis. The difficulty is manifestly greater when the patient is a woman. Age is also, in a sense, a predisposing cause. The majority of cases occur between twenty and thirty years of age, though the condition may occur in children. Pritchard has collected over fifty published cases occurring between the ages of fourteen months and fourteen years, and about equally divided as to sex. Among adults disseminated sclerosis is met with somewhat more frequently in women.

Symptoms.—These may be described under two headings: first, the general symptoms, or those common to all cases of the disease, and not explicable from the position of the sclerosis; and, secondly, those dependent on the locality of the lesions. The disease is always chronic, and either remissions, or one or more intermissions occur, and in some cases may extend over several years. The first evidence of the disease is *loss of power*, first in one, then in the other, lower extremity. Later, paresis develops in the upper extremity. Sooner or later other general symptoms appear—viz. tremors, nystagmus, scanning speech, increased reflexes, and optic-nerve atrophy. The *tremor* is volitional (*intention-tremor*), and when the patient is at rest no abnormal movement is manifest, as a rule. On attempting to use the hands, or in walking, a fine, trembling motion of the limbs results. The head may be similarly involved, and some incoördination is commonly associated therewith. The *nystagmus*, too, is brought out when the eyes are in use. It is more marked in lateral than in vertical movements. *Speech* is slow and deliberate (staccato or scanning), the tendon-reflexes are increased, ankle-clonus may be present, and optic-nerve atrophy is of frequent occurrence. No alteration of sensation occurs, other than perhaps some numbness or tingling. There is no wasting of, nor electric change in, the muscles, nor do bed-sores occur. Vertigo is usually present. The mental phenomena are at first hysteroid, and they may never progress beyond this point. In other cases dementia, or even acute maniacal outbursts, are met with, but these are rare. During this stage epileptiform or apoplectiform attacks may occur. The symptoms directly resulting from the local lesions cannot be given in detail. Certain types result, however, that depend upon the tendency of the sclerotic areas to involve certain tracts, and these are—first, a form resembling lateral sclerosis, due to implication of the lateral tract; and, secondly, a form similar to locomotor ataxia, in which the posterior columns especially suffer.

The **diagnosis** is generally easy after the disease has lasted some time. The intention-tremor and the gradual and progressive loss of power, with increased reflexes, scanning speech, and mental deterioration, are sufficient. The following table gives the differential points between this disease and *paralysis agitans*, *locomotor ataxia*, and *hereditary ataxia:*

Disseminated Sclerosis.	Paralysis Agitans.	Locomotor Ataxia.	Hereditary Ataxia.
Rarely occurs in children. Generally between the twentieth and thirtieth years.	Occurs in persons over forty years of age.	Rarely before the twentieth year.	Usually before the twentieth year. Generally affects several in the same family.
No sensory symptoms, as a rule. Sight may be impaired, the hearing less frequently. The Argyll-Robertson pupil is absent.	No sensory or special-sense symptoms of any importance. Argyll-Robertson pupil is absent.	Fulgurant pains an early symptom. Sight and hearing are commonly affected. Often diplopia and Argyll-Robertson pupil are present.	Sensory symptoms are rarely present. Diplopia and Argyll-Robertson pupil are absent.
Nystagmus is present, as a rule.	No nystagmus.	No nystagmus.	Nystagmus is frequent.
Reflexes are exaggerated; ankle-clonus is present. There may be muscular rigidity.	Reflexes are normal; very rarely they may be plus. Permanent muscular rigidity.	The knee-jerk, ankle-clonus, and rigidity are all absent.	The knee-jerk is lost in the course of the disease; it is rarely increased. No rigidity.
Scanning speech.	Speech is slow and deliberate on commencing a sentence, but soon it becomes hurried.	No speech-defects.	Speech is slow and irregularly scanning.
A tremor is generally present on voluntary movements only. If the tremor occurs during rest, it is fine, Oscillations of the head are frequent; of the trunk, less so.	Tremor when at rest. Voluntary movement may make it cease temporarily. The head may shake, with rather a vertical than an oscillatory movement.	No tremor. Incoördination is marked. No oscillations of the head or trunk. Romberg's symptom is present. Trophic disturbances are common.	Incoördination is present, but is not increased by closing the eyes. Static ataxia may be noted.
Mental disturbance is frequent.	No mental phenomena.	Mental disturbance is rare.	No mental disturbance.
Gait is usually spastic and paretic, and often uncertain.	The head is bent back and arched; the face is immobile and masklike. The gait is propulsion, festination, retropulsion, or lateropulsion.	The gait is stamping in character; the legs are moved stiffly. There is difficulty in urination.	The gait is swaying and irregular, like that of a drunken man. The legs are not kept wide apart as in locomotor ataxia.

The **course** usually extends over five to ten or even fifteen years, and death is generally the result of some intercurrent affection, though it may occur during an apoplectiform or convulsive attack. Rarely it is due to failure of the heart or respiration.

The **prognosis** is always bad.

Treatment.—No remedy is of any avail. Silver nitrate, mercury, the iodids, and arsenic may be tried. Rest and easily assimilable food are also of prime importance.

PSEUDOSCLEROSIS.

In 1883 Westphal described a case characterized by disturbance of speech, slowness of the movements, decrease of both intelligence and irritability, apoplectiform attacks, pronounced tremor, spasticity and increased reflexes, slight disturbance of sensation, and no involvement of the sphincters. The autopsy was entirely negative. Since then similar cases have been reported, especially by Strümpell. The disease usually begins in childhood and causes death in the course of several years.

The *diagnosis* cannot be made from multiple sclerosis during life.

Treatment is useless.

BULBAR PARALYSIS.

(*Glosso-labio-laryngeal Paralysis.*)

Definition.—An acute or chronic disease, due to involvement of the motor nuclei of the medulla oblongata. It is generally secondary to some condition affecting other portions of the motor path, and is characterized chiefly by a difficulty of speech or of deglutition. Three varieties have been described:

1. **Sudden or apoplectiform**, this being due to hemorrhage, embolism, or softening. The onset is always sudden, often with vertigo, and possibly vomiting, with or without loss of consciousness. The power to articulate is impaired or lost. The lips and tongue are involved, and hence the pendulous lower lip, the dribbling of saliva, and the atrophy of the lingual muscles. There are dysphagia and generally frequent attacks of choking.

The *symptoms* are less characteristic than those of the degenerative form. They are less regular in type, and usually are widespread at first; later, some improvement takes place. In other cases, after more or less of a respite, degeneration sets in and they grow progressively worse.

The *diagnosis* of this type is not usually difficult. "Pseudo-bulbar paralysis" must be borne in mind, however, and is a condition due to a bilateral lesion of the motor cerebral cortex in the lower frontal parietal region or of the motor fibers in the course. There is great danger to life for some little while in these sudden cases. Later the prognosis is rather more favorable than in the other forms.

2. **Acute Inflammatory.**—Here the onset is less abrupt, requiring a

few days to a week to develop. But for this fact the symptoms are much the same as in the preceding form.

3. **Chronic Bulbar Paralysis.**—This condition occurs chiefly in males beyond middle life. The cause can seldom be discovered, though certain cases seem to be of toxic origin. It may develop in the course of progressive muscular atrophy, amyotrophic lateral sclerosis, insular sclerosis, or other disease of the cord.

The *symptoms* are bilateral, the tongue being usually the first to suffer. The patient may notice that he cannot speak for any length of time without fatigue, and that he will then articulate indistinctly. Soon he observes that there is a marked and progressive *impairment of speech.* The muscles of the lips and other muscles of the lower part of the face atrophy. He can no longer whistle. Speech is rendered still more defective, owing to paralysis of the lips. The lower lip drops, and the saliva constantly dribbles from the mouth and may be greatly increased in amount. *Difficulty in swallowing* is always present to a greater or less degree. Owing to the lingual paralysis, the tongue can neither be protruded nor can it be used to manipulate the food and make a bolus. It is atrophied and the mucous membrane is wrinkled. *Fibrillar tremors* are present. The *larynx* is involved, so that phonation is imperfect, but it is not so marked as the implication of other parts. Particles that enter the larynx cannot be ejected, owing to motor paralysis. There are no sensory symptoms, and the power of taste is normal. The mind generally remains clear, though the patient is often emotional, and cries or laughs without apparent cause. This type of bulbar palsy is particularly liable to develop in the course of progressive muscular atrophy. The *course* of the disease is slow, and death is usually due either to inspiration-pneumonia or to interference with respiration or circulation.

The **diagnosis** is not difficult, as a rule, the bilateral character of the symptoms rendering them distinctive. In the *pseudo-bulbar form* previously mentioned the limbs are often paralyzed also (double hemiplegia). *Tumors* rarely, if ever, give rise to such regular bilateral symptoms. I have met with 2 cases of chronic bulbar palsy, and 1 occurring in the course of Bright's disease, in which no postmortem lesion could be found that would account for the condition. In neither of the cases was there much atrophy, though otherwise they conformed to the regular type.

Treatment.—The disease is incurable. Hypodermics of strychnin, or of strychnin, morphin, and atropin, are of value in controlling the salivary flow. Electricity is of no value. Semi-solid food is probably the most readily taken, and it is often necessary either to use an esophageal tube or to employ rectal alimentation.

AMYOTROPHIC LATERAL SCLEROSIS.

(*Charcot's Disease.*)

Definition.—A disease of the entire motor system, from the cerebral cortex to the muscles: characterized by loss of power, spastic symptoms, and muscular atrophy. The first clear and thorough description of the clinical symptoms and pathological anatomy was given by Charcot in 1872.

Pathology.—The pyramidal tracts are degenerated, the process commencing either in the cortex, crura, or medulla, and extending to the termination of the neurons in the cord. The ganglion-cells of the anterior cornua are atrophic, there is degeneration of the anterior roots and of the muscle-fibers, the blood-vessels in the affected parts are dilated, and in the early stages granular cells are present.

Etiology.—The disease is more frequent in males and usually begins in early adult life. Exposure has sometimes been noted in the previous history, but neuropathic heredity does not appear to have any influence.

Symptoms.—Three stages are generally recognized: (1) The involvement of the upper extremities. (2) The participation of the lower extremities. (3) The appearance of bulbar symptoms. At first there are weakness of the upper arms, atrophy of the muscles, and moderate exaggeration of the reflexes; in the course of a few months the symptoms of spastic paraplegia develop, all the reflexes are greatly increased, and there are chin- and ankle-clonus and dragging of the toes. The wasted muscles show fibrillary twitchings and give the reactions of degeneration. Contractures then occur, the forearms are flexed on the arms, the hands are held in pronation, and the proximal phalanges of the fingers bent backward, giving rise to the so-called *claw-hand.* From time to time there are tonic spasms in the muscles, particularly in the calves (*spinal tetanus*). Sensation is not disturbed, excepting for the occurrence of slight paresthesia from time to time, and the sphincters continue to functionate normally. Finally the bulbar symptoms appear, and there is paralysis of the lower part of the face, which becomes rigid and expressionless, with the mouth partly open and saliva dribbling from the angles. Deglutition and articulation become difficult or impossible, and death finally occurs from exhaustion or inspiration-pneumonia. During the course of the disease the intellect is slightly involved. Memory is impaired, the conduct becomes childish, and there is a tendency to weep or laugh without cause. Atypical cases occur in which either the lower extremities are first involved, or the paralytic symptoms are more prominent than the spastic symptoms, or the bulbar symptoms appear very early. The course is steadily progressive, and death usually occurs within two years.

The **differential diagnosis** is to be made from *multiple sclerosis* by the absence of nystagmus, of the intention-tremor, and of sensory disturbances, and by the degenerative changes in the muscles; from *transverse myelitis* by the absence of sphincter disturbance and of pain, and the involvement of the upper extremities and head; from *progressive spinal muscular atrophy* by the presence of spastic symptoms; from *syringomyelia* by the absence of sensory disturbances, trophic lesions of the skin and joints, and the greater regularity of the course; from *pressure upon the spinal cord* by the absence of pain and sphincter disturbance and the involvement of the head and upper extremities. It must be remembered that amyotrophic lateral sclerosis may be associated with multiple sclerosis or infantile spinal paralysis.

Prognosis.—It will be understood from the foregoing description that death is the invariable termination. The course is progressive, and even temporary amelioration rarely occurs.

Treatment.—The patient should be rendered as comfortable as possible. Arsenic and mercury are useless, but the hypodermic injection of strychnin (see Progressive Spinal Muscular Atrophy) may be tried.

SYRINGOMYELIA.

Definition.—A neurogliar overgrowth of more or less vertical extent, and situated in the gray matter of the cord in the neighborhood of the central canal. Its symptomatology is not constant, but the following have come to be looked upon as typical of most cases: viz. progressive muscular atrophy and dissociated anesthesia (*i. e.* impairment or loss of temperature—and pain-sense, with retention of the tactile and muscular sense and trophic and vaso-motor disturbances).

Pathology and Etiology.—Tubular cavities of greater or less extent are met with in the cord as a result of two conditions existing separately or in conjunction—viz. (1) *hydromyelia*, a dilatation of the central canal (proved by the cubical cells lining it). This is either (*a*) congenital, according to Leyden, or (*b*) acquired, due to pressure (tumor), dilatation taking place above the point of obliteration. (2) *Syringomyelia*, a name given by Olliver to a neurogliar overgrowth situated within the gray matter of the cord. In this cavity-formation takes place as a result of hemorrhage or degeneration. The cavity is entirely without the central canal; it never possesses an epithelial lining, and is not, therefore, as Leyden supposed, the remains of congenital hydromyelia. While the new growth in many instances is gliomatous, being probably a rejuvenescence of some vestigial remnant, with subsequent hemorrhage or degeneration and cavity-formation, yet in others the structure is not identical with such neoplasms. The latter have been described particularly by Joffroy and Achard. They speak of it as a gliosis, a secondary overgrowth, and sclerosis of the neuroglia. In any case, however, the disease is most prone to develop in the cervical and upper dorsal region, growing and invading the posterior and postero-lateral tracts. Breaks and crevices in the diseased material radiate from the main cavity. The onset of the trouble generally takes place somewhere between the fourteenth and twenty-first years of age.

Symptoms.—Owing to the fact that different levels of the cord are involved, and that the extent claimed by the process varies in different cases, it will readily be understood that no account, however concise, will fit every case. The disease is of *slow onset.* Neuralgic *pains* develop in the muscles, and the latter progressively *waste.* The *reflexes* are increased and more or less spasticity is present. The lower extremities usually escape, though they too may be involved, when the condition presents much the same appearance as amyotrophic lateral sclerosis. The *temperature-* and *pain-sense* are lost, but the *tactile* and *muscular* senses are preserved.

The *special senses* and the sphincters are usually normal. *Ocular symptoms* develop only when the cervical cord is extensively involved. *Joint-changes* may be met with, and various *ulcerations*, *bullous eruptions*, or *wounds* may be present, the latter often being received without the patient's knowledge, since loss of sensation is complete. These constitute a special feature of the disease originally described by Morvan of Brittany in 1883. He had observed many cases prior to that time, but his attention was specially called to the matter by a case of whitlow which he incised, but to his surprise no pain whatever was experienced. He described *the disease as affecting the upper extremities*, with neuralgia, progressive paresis and wasting, dissociated anesthesia, and, later, painless whitlows and necrosis of the phalanges. Joffroy and Achard have made three autopsies upon cases dying of this disease, and in each syringomyelia was found. In Gombault's case neuritis was present, and the current view is that *Morvan's disease* is a combination of syringomyelia and neuritis.

Diagnosis.—The loss of pain and thermic sense, with preservation of the muscular and tactile senses, in association with the muscular wasting, which is most marked in the upper extremities; and with the spasticity of the lower extremities, and the trophic changes, especially in the fingers, constitute a group of symptoms that has come to be regarded as typical.

Differential Diagnosis.—*Hypertrophic cervical pachymeningitis* may be mistaken for this disease, and *vice versâ*. In this case, however, the pain is usually greater, the tactile sense is apt to be lost, and possibly the other senses also; but there is not the dissociation met with in syringomyelia. *Amyotrophic lateral sclerosis* presents neither sensory nor trophic symptoms, other than the muscular wasting. *Disseminated sclerosis*, apart from the tremor that is always present, presents less trophic disturbance. The neural form of *leprosy* may present a clinical picture that cannot be differentiated. There are dissociation of sensation, trophic changes in the fingers, and muscular degeneration. Even spasticity of the lower limbs may occur, although this is rare.

The **prognosis** is always unfavorable, though the disease runs a very chronic course, lasting even fifteen or twenty years.

Treatment.—Nothing can be done, except by attention to hygienic and dietetic details.

COMPRESSION OF THE SPINAL CORD.

It is of importance to be able to recognize this condition. To be sure, it is not always possible to diagnose it with certainty, but when there is a reasonably surety the question of operation may arise. Since it has so many features in common with myelitis, the necessity for reserve and caution in arriving at a conclusion is manifest, since the latter condition would not be benefited by any operative procedure.

Pathology.—The postmortem findings will depend upon the degree and duration of the pressure. The cord will be more or less flattened and distorted at the seat of pressure, and in the early stages hyperemic, and possibly softened. Later it is hard, sclerosed, and of a grayish color, and above and below the compressed region degenerated areas will be seen on sectioning the cord. Microscopic examination reveals the same changes as those met with in any other form of myelitis. The nerve-roots will be more or less damaged by compression.

Etiology.—We may class the causes of compression under three headings—(*a*) traumatism (fractures), (*b*) inflammatory disease (caries of the spine), and (*c*) neoplasmata (including various tumors, gummata, and aneurysm), but these will receive separate consideration (*infra*).

Symptoms.—These will vary according to the site of the lesion and the extent of involvement—*i. e.* the vertical extent, the degree of pressure exerted, and the amount of inflammation present. Two groups of symptoms are present in typical cases—first, those due to involvement of the roots, and, second, those dependent upon involvement of the cord itself—*ascending* and *descending* degeneration. The former gives rise to *pain*, neuralgic in character and radiating along the course of the nerves. The parts supplied are usually *tender*, and there may be *paresthesia* and *formication*. These irritative symptoms are followed sooner or later by those of *paralysis*, and hence the anesthesia. Areas of *hyperesthesia* may accompany the anesthesia (*anæsthesia dolorosa*). *Motor symptoms* are also irritative and paralytic, and hence the early twitching, or even spastic condition, and later the loss of power, or paralysis. The muscles of the affected parts waste, and qualitative and quantitative electric changes can be elicited. The *second group*, due to cord-changes, then develops, and its symptoms may set in either rapidly or slowly. If myelitis promptly supervenes and is extensive, *cord-symptoms* of a pronounced type develop quickly. The parts below the lesion will become weak, there will be girdle pains, and a sense of constriction or pain in the legs. Sensory symptoms may be absent. The reflexes are usually increased. If the tumor or other cause of compression ceases to fail to act for a time, some improvement takes place, due possibly to the subsidence of the myelitis. If the pressure is of *slow onset*, great tolerance is manifested. As is usually the case, sensation is recovered before motion. In certain cases, however, motor power is regained, while the muscular and tactile senses do not return. In such instances, in which the posterior columns bear the brunt of the trouble, incoördination results and secondary ataxia is met with.

Diagnosis.—If the combined symptoms of peripheral and central origin develop slowly in the order named, compression is likely. *Myelitis* gives rise first to cord-, and only later to peripheral symptoms; hence the difficulty in cases in which myelitis develops quickly. Extensive root-symptoms are suggestive of *meningeal involvement*. In any event, too much stress should not be placed on the nervous symptoms alone. The spine should be carefully examined and palpated for points of tenderness. Careful note should also be taken as to whether there is any limitation of movement or deformity. The family history may suggest *tuberculosis* (caries of the spine).

The **prognosis** depends entirely upon the cause. Having ascertained this, it then depends upon the possibility of its removal.

Treatment.—In general the treatment is that of myelitis. In certain cases a surgeon should be consulted, though operative cases are the exception rather than the rule, and most may be expected in cases of caries. It is well to impress upon the patient and relatives the chronicity of the condition, but faithful and persistent efforts will yield good results. Rest is of vital importance, particularly when the disease is active. The patient should be kept in bed in a recumbent position until consolidation has taken place. Extension may be necessary. Good and easily assimilable food, and cod-liver oil and alteratives should be given. The nutrition of the muscles may be improved by gentle friction (massage). As soon as possible a plaster jacket should be put on the patient, and he should be taken into the open air and sunlight.

TUMORS OF THE SPINAL CORD AND ITS MEMBRANES.

A GREAT variety of neoplastic formations, both primary and secondary in nature, may occur. The location is of course the most important clinical feature; nevertheless, if we would institute successful medical or surgical treatment it is desirable to determine as nearly as possible the nature of the tumor.

Extradural tumors may be, though rarely, exostoses from the periosteal lining of the spinal canal. More frequently lipomata or even an abnormal development of fatty tissue between the bone and the membrane is present. Of the parasitic growths, the hydatid cyst is apparently the only one that occurs in this situation.

The *tumors* of the *dura mater* itself are chiefly sarcoma, gumma, and myxoma. Calcareous plates may occur in the arachnoid. Occasionally growths from the bones, particularly carcinomata and sarcomata, may extend to the membranes. Inside the dural sac both hydatid cysts and the cysticercus may be found. Tuberculosis may occur either in the form of miliary tubercles or as tuberculous masses, particularly in the cervical region. In the pia mater and arachnoid, myxoma, lipoma, fibroma, endothelioma, sarcoma, and angio-sarcoma occur. Occasionally cylindroma and osteoma are also found. A specimen with bony plates in the dura was obtained from an old woman who had syphilitic lesions in other parts of her body.

In the cord itself the commonest tumors are glioma and sarcoma. Gummata also occur (*vide* Syphilis, p. 330). Cysts are sometimes found. The simplest form is the dilatation of the central canal known as hydromyelia; the commonest are those that occur as a result of gliomatous softening in syringomyelia. Finally multiple neuromata may occur upon the nerve-roots. The changes that take place in the cord are destruction of the nervous tissue at the side of the tumor, pressure-myelitis, and systemic degenerations in the various columns. Curiously enough, complete restoration of function can occur, even when the symptoms of degeneration in the pyramidal columns are pronounced, and there is wasting of the cord macroscopically, as in the case recorded by Gowers and Horsley.

The **etiology** of these conditions varies, of course, with the nature of the growths. Certain tumors, as lipoma and glioma, are more apt to occur in advanced life; others, as the tyroma, somewhat earlier.

The **symptoms** depend upon the particular segment or segments affected, and the situation in the cord itself or its membranes. In general the dorsal or lower cervical region is most frequently involved.

Disturbances of Motion.—If the tumor attains sufficient size to exert considerable pressure, paraplegia always occurs. This usually commences upon one side, and then more or less rapidly involves the other. *Exaggeration* of the *tendon reflexes* of the leg is almost invariable in tumors above the first lumbar segment. Whenever this condition is at all advanced there are disturbances of the functions of the bladder and rectum. *Spasms* are sometimes the earliest motor changes. Ordinarily they appear in the muscles of the trunk governed by the segment that has been involved; but sometimes they appear in the legs, and are usually more severe in one than in the other, and they may be due either to pressure upon the motor roots or to pressure upon the motor columns of the cord. It is not therefore permissible to draw definite conclusions from their location as to the site of the tumor. *Paresis* is commonly an associated symptom, and gradually deepens into paraplegia. In the latter stage *contractures* may also develop.

Special Motor Symptoms.—If the tumor is situated in the lower portion of the cervical region, there are often disturbances of *motility* in a certain definite group of muscles that are supplied by the brachial plexus. These disturbances may be *ataxia*, *tremor*, *spasm*, or *paralysis*. If the tumor be in the dorsal region, the cramp of the intercostal or abdominal muscles at a particular level may give rise to a *girdle-sensation;* if in the lumbar region, to disturbances of motion similar in character to those described in the arm. In this locality, however, as the nerve-roots are longer, more of them are apt to be involved in the pressure, and the symptoms are more extensive.

Sensory Symptoms.—*Pain* is usually the earliest symptom. It is, as a rule, sharply localized, severe, and paroxysmal, with symptoms of dull neuralgic aching between the exacerbations. Certain forms of pain are very common. Tumor in the cervical region gives rise to severe pain in one or both arms and to neuralgic pains in the neck and occiput. Tumors in the dorsal region cause the girdle-sensations before described, or intense backache, often associated with tenderness over the site of the tumor. Tumors in the lumbar region induce pain in one or both legs, often of a burning character, and sometimes referred to the soles of the feet. *Hyperesthesia* of the skin is usually found at a level supplied by the segment in which the tumor is situated. Other sensory disturbances are tingling, numbness, and total anesthesia. A rare combination, in the early stages of the tumor of the spinal cord, is the presence of anesthesia in one-half the body below the tumor and of paresis or paralysis in the other half—that is, the *symptom-complex of Brown-Séquard:* it almost invariably disappears in a short time The reflexes are increased below the level of the tumor. If this is situated in the lower cervical region, then the cutaneous reflexes of the thorax and abdomen are prompt and vigorous; if in the dorsal region, they are normal above the site of the tumor, abolished in its neighborhood, and increased below, and at the same time there is an enormous exaggeration of the skin- and tendon-reflexes of the legs; if in the upper portion of the lumbar region, there may be an abolition of the knee-jerk, while ankle-clonus is exaggerated; but ordinarily all reflexes are abolished.

The muscles frequently degenerate, and the electric reactions of degeneration are found in those regions that are supplied by the anterior horn, or the anterior roots that have been destroyed by the tumor. In those tumors situated in the lumbar region, involving a number of nerve-roots, the wasting of the muscles of the legs is usually very marked. *Trophic disturbances* occur late in the course of the disease, when extensive bed-sores may develop, exactly as in transverse myelitis. Occasionally vaso-motor disturbances (*tâche spinale*, localized edema) may be observed in the early stages.

Course.—Tumors usually grow slowly, and therefore the symptoms are gradual in their development. Ordinarily there are periods of arrest or even improvement that are followed subsequently by further advance. The *duration* of spinal tumors is variable. Those of malignant nature or rapid growth may produce death in a short time; those that simply exert pressure and enlarge very slowly may not produce total disability for several years. In general it may be said that from five to ten years is the ordinary limit after the first appearance of motor disturbance. Some tumors, however, particularly lipomata, produce only slight disturbances throughout life, or else no symptoms at all, remaining entirely latent.

The **diagnosis** involves three points: first, the recognition of the presence of the tumor; second, of its site; and third, of its nature. The prodromal symptoms of spinal tumor are often confounded with *neuralgia* or *lumbago*. It is sometimes possible to make a differential diagnosis by means of the presence, in neuralgic conditions, especially of intercostal nature, of the sensitive points along the course of the ribs, and of the existence, in the case of tumor, of exaggerated knee-jerks and sensitiveness over certain portions of the vertebral column. In the paraplegic condition it may be confounded with a *neuritis*, such as one of alcoholic origin. In these cases the diagnosis is more difficult if the tumor is situated in the upper portion of the lumbar cord; nevertheless, the sensory disturbance is slight in alcoholic neuritis, whilst it is considerable in tumors in the lumbar region, and often presents the form of *anæsthesia dolorosa*, that is, diminished sensibility associated with considerable pain. There are also apt to be disturbances of the sphincters.

The *intrinsic diseases* of the spinal canal give rise to much greater difficulty, especially *myelitis* and *pachymeningitis cervicalis.*. From the former the correct diagnosis may sometimes be suspected, because in tumor there are severe radiating pains and the symptoms are more pronounced on one side than on the other. Moreover, the symptoms of segmentary involvement are sharper and the root-symptoms more characteristic. From pachymeningitis cervicalis, a central tumor in the cervical region can be usually distinguished by the fact that the radiating pains are less severe and the symptoms not so distinctly bilateral. It may be impossible to distinguish a central tumor from *syringomyelia* unless the symptoms of root-pressure are quite distinct. *Pott's disease*, in its early stage, may also give rise to some difficulty. However, the rapid development of the kyphosis, and particularly the pain that is elicited by sudden pressure upon the head, renders it possible, after a reasonable period of observation, to recognize the true nature of the case.

The *diagnosis of the position of the tumor* has been largely discussed in the Symptomatology. The symptom-complex may, however, be considerably disturbed by the presence of multiple tumors. In these cases the majority ordinarily remain latent. It must not be forgotten, however, that the absence of the knee-jerk does not localize the tumor to the lumbar region of the cord, for it may be abolished when the tumor is situated in the dorsal region and compresses the posterior columns. In general, it may be said that the presence of root-pains suggests a meningeal seat, whilst pronounced paraplegia, or the Brown-Séquard symptom-complex, points to the presence of a tumor in the substance of the cord itself.

Finally the recognition of the *nature of the growth* can often be made from the history of the existence of a tumor or an infectious process in other parts of the body. It must be remembered, however, that it does not always follow that a tumor in the spinal canal is similar to that found elsewhere. The presence of cerebral as well as spinal focal lesions points very strongly to syphilis.

The **prognosis** depends upon the severity of the symptoms, the rapidity of their development, and the nature of the growth, if this should be known. Complete subsidence of all the symptoms may occur, even after a spastic paraplegia has existed. Of course this is only likely in those cases in which the tumor can be removed by operation or absorbed through the action of drugs.

The **treatment** depends wholly upon the recognition of the nature of the tumor; if this be syphilitic, mercury and potassium iodid should be given in full doses. If, on the other hand, it is not specific, and appears to be extradural, operation would seem to offer a possibility of cure, the famous case of Gowers and Horsley having demonstrated the practicability of removal. As the prognosis is, in general, unfavorable as to cure and often gloomy as to life, the clinician should not hesitate to recommend surgical interference.

LESIONS OF THE CONUS TERMINALIS AND THE CAUDA EQUINA.

As the spinal cord terminates at the second lumbar vertebra, tumors or injuries below this point produce symptoms only in so far as they compress or destroy the lumbar roots. This destruction may be partial or complete. If partial, there are *paralyses* of various groups of muscles, and circumscribed areas of anesthesia. There may or may not be a disturbance of the functions of the bladder and sphincters. If this be *total*, there are complete anesthesia, complete paraplegia, flaccid in character, with reactions of degeneration in the muscles, loss of the knee-jerk, and rectal and vesical incontinence. If only the *cauda equina* is involved, there may be isolated paralysis of the bladder and rectum. These lesions may consist of tumors, such as are found in the membranes of the cord or on the nerve-roots, and it should be noted that, probably on account of greater space for their development, tumors in this situation are apt to be larger than those in other parts of the spinal canal. They may also consist of fractures or lesions occurring as a result of congenital anomalies, such as spina bifida.

III. DISEASES OF THE BRAIN AND ITS MENINGES.

DISEASES OF THE DURA MATER.

Inflammation.—This may be met with on the outer or inner surface (*pachymeningitis externa* or *interna*). Of the external variety the chief causes are (*a*) traumatism, (*b*) disease of the bone, (*c*) syphilis, and (*d*) middle-ear disease. That due to traumatism is often seen, and in the mildest form is of little moment. When severe and accompanied by fracture with or without displacement, infection of the membranes may either take place at once or later from diseased bone. That form due to caries or any other form of osteitis is always dangerous, owing to the possibility of infection of the diploë. The brain-sinuses will then become affected, and infected emboli may pass into the circulation, with the development of pyemia. In the syphilitic variety the inner table of the skull is thickened and roughened, and more or less pus and granular material is found between it and the dura (see also Syphilis of the Nervous System). Sinuses may communicate with the exterior.

The **symptoms** are indefinite in mild cases, and may consist only of *headache*. In the severe forms there are *headache*, *malaise*, *chills*, *fever*, *drowsiness*, and later *stupor*, and rarely *convulsions* and *hemorrhage*, or other symptoms of compression. The ophthalmoscope will reveal more or less evidence of *choked disc*. Rigors are suggestive of the onset of pyemia.

The **treatment** varies with the cause. Antiphlogistic measures and counter-irritation are of value, and in the severe grades operative interference may be necessary. The internal variety either occurs as a simple inflammation or may be so acute as to cause extravasation of blood. This may organize, and, together with the products of inflammation, cause a pseudo-membrane. Rarely is pus found.

Hemorrhage.—Hemorrhage may be (1) extradural—(*a*) traumatic and (*b*) due to rupture of a vessel by erosion, the result of caries; or (2) intradural—into the so-called arachnoid sac—(*a*) very rarely traumatic; (*b*) due to injuries at birth; (*c*) due to pachymeningitis interna; (*d*) met with in general paralysis of the insane; (*e*) occurring in the course of anemia, scurvy, or some other profoundly altered blood-condition; (*f*) in cardiac, renal, or pulmonary disease; (*g*) the result of strain—*e. g.* whooping-cough.

The **symptoms** will depend upon the circumstances, whether the amount of blood is small or large, whether the onset is *gradual* or *abrupt*; they may be further obscured by the primary disease, or by *shock* if the cause is some trauma. In the slight forms absolutely nothing characteristic exists. In others there are *headache*, *vertigo*, *vomiting*, and possibly *mental confusion*, *convulsions*, or *coma*; in fact, the ordinary symptoms of apoplexy.

The **treatment** is that of cerebral hemorrhage; in some cases operation is justifiable.

Internal hemorrhagic pachymeningitis or h[illegible] of the dura mater is ch[illegible]rized by the formation of a fibr[illegible] upon the inner surf[illegible] capillari[illegible] quently rupture. It is found most commonly among the insane or epileptic. It is rare in childhood.

The **symptoms** are variable. The entire course may be without symptoms, or they may be masked by the existence of other conditions. More frequently there are *headache* and *convulsions*, followed later by *paralyses*, coma, and death. The location of the lesion causes considerable modification of the symptomatology. In the milder form recovery frequently occurs, or the case may become chronic. If the onset is sudden, the symptoms may resemble those of hemorrhage.

The **diagnosis** is always difficult. In children muscular contractions and convulsions are frequently met with; in adults the slow onset may be the only difference between this condition and an attack of *grand mal*. Of course, there is a greater periodicity in epilepsy; but a repetition of the attacks occurs in hematoma, and, as already stated, the repeated hemorrhages are believed by some to be the cause of the lamination of the false membrane.

The **prognosis** is extremely unfavorable in children, but is much less so in adults.

The **treatment** calls for the use of leeches behind the ears and over the temples, the ice-cap, and counter-irritation. Free movement of the bowels should be promptly secured, and later the iodids or mercurials should be administered.

Tumors are considered in connection with Tumors of the Brain.

DISEASES OF THE PIA.

Inflammation (*Leptomeningitis*).—This is met with in the following conditions: (*a*) When tubercles develop on the membrane (*vide* Tuberculous Meningitis); (*b*) During, or as a sequel to, some acute febrile disease, as pneumonia, erysipelas of the head and face, small-pox, measles, scarlet fever, typhoid fever, ulcerative endocarditis, and pyemia; (*c*) Cerebro-spinal meningitis; (*d*) Cachexia; (*e*) Gout and Bright's disease; (*f*) Exposure to the sun; (*g*) Traumatism, even when not accompanied by fracture; (*h*) Disease of the bones—caries, or secondary to middle-ear disease; (*i*) Extension from syphilitic involvement of the meninges.

Inflammation of the pia (non-tuberculous) is more common in males than females, and occurs, as a rule, before the twentieth year. After that time it is rather rare.

Pathology.—In the extent and degree of the inflammation, great variations exist. Is may be either (1) limited to the convexity, with or without involvement of the sides; (2) limited to the base; or (3) general, involving both convexity and base. In the early stages and in the mild forms there may be no more than an injection of the part. Later, inflammatory products are met with and more or less adhesion exists. Pus is also present in some cases. This form of leptomeningitis, unlike
is prone to attack the convexity of the brain. I
one child of twenty months in which the in-
'ie convexity, and still less on the sides, but
rticularly about the Sylvan and cerebellar
m. A granular ependymis was found

limited to the body of the lateral ventricles, and reminding one very much of a similar condition met with in paralysis of the insane. The ventricles were dilated, and contained an excess of fluid, due probably to occlusion of the foramen of Magendie, as Gee and Barlow have pointed out. No tubercles were found, nor did the family history lead me to suspect tuberculosis.

Symptoms.—These are very varied, and naturally depend on the *seat* and *extent* of the inflammation. Those cases in which symptoms pointing to involvement of the base occur need not be discussed here, since they are considered in detail under the tuberculous variety. In any case *headache*, localized or general, is usually present. In children too young to talk its presence is often indicated by crying or putting the hand to the head. *Delirium*, *insomnia*, and *coma* are also met with in different cases. There are more or less *fever*, *constipation*, a *coated tongue*, *vomiting*, a *rapid pulse*, and the *tâche cérébrale* may be elicited. Spasmodic movements may occur, or even general *convulsions*. Of course, in cases of inflammation of the base, the cranial nerves become affected, and we have *ptosis* or *strabismus*, *facial spasm* or *palsy*, and, if the fifth nerve is involved, *sensory* and *trophic* changes. The head is usually retracted until it seems to bore into the pillow; the muscles of the back of the neck are tense; the spine is often rigid; the abdomen retracted, and the limbs flexed. The tendon reflexes are exaggerated and cutaneous irritability greatly increased. *Kernig's sign* consists in the inability of the patient to straighten the leg, when the thigh is flexed to a position of 90 degrees to the axis of the body. It is very constant, and therefore of value in diagnosis. It must be borne in mind that meningeal symptoms are frequently simulated by the *infectious diseases*, and particularly by pneumonia (of the apical type generally; more especially in children and old people), typhoid fever, and influenza. Such cases are most likely due either to vascular disturbance of the meninges (congestion) or to a toxic encephalopathy. This has been described by the French writers under the name *meningism*.

Diagnosis.—Where no etiologic hint can be obtained the diagnosis is generally in doubt for two or three days. There may be nothing more than a reflex irritation (dental or gastro-intestinal), or possibly one of the infectious fevers. The symptoms should be studied in their entirety; one or two supposedly pathognomonic signs should not be allowed to cloud our vision. Having made the diagnosis of meningitis, it becomes important to differentiate the *tuberculous* from the *non-tuberculous* variety. The family history is of importance. In *tuberculous meningitis* the focal symptoms usually appear late, and are due to involvement of the cranial nerves at the base of the brain, chiefly those controlling the eye. The eye-grounds often show a slight perineuritis without choked discs, and perhaps one or more miliary tubercles. The leukocytes are slightly, if at all increased. There is rarely rigidity of the neck. In other forms of meningitis this appears early. The optic nerve shows intense inflammation and there is usually pronounced leukocytosis. Quincke, in 1890, introduced his method of *lumbar puncture*. This is of comparatively simple application, and has become a well-recognized measure. The tubercle bacillus and streptococcus pyogenes, also the pneumococcus and micrococcus meningitidis, have been found in the fluid withdrawn.

Prognosis.—This is always grave. A percentage of cases of epidemic cerebrospinal meningitis, varying with the nature of the epidemic, may recover. In all other forms any termination, except in death, is exceedingly exceptional. Remissions frequently occur in the symptoms, and the course may be very prolonged. That even tuberculous cases may recover is proved, however, by that of West, and even more strikingly by that of Baumann and Senlie, who found the tubercle bacillus in the cerebro-spinal fluid. The case recovered, but died some months later from an intercurrent affection; the diagnosis gained additional confirmation from the *postmortem* findings.

Treatment.—This has been previously considered. It is only necessary to add that we are able to do no more in this form than in the tuberculous. We have no specific, and all that can be done is to meet the symptomatic indications. In certain cases—*e. g.* those secondary to middle-ear disease—operation may seem justifiable. When in doubt the physician should not delay action until too late, but should call in a surgeon while some benefit may still be hoped for.

DISTURBANCES OF CIRCULATION OF THE BRAIN.

HYPEREMIA.

Definition.—An abnormal increase in the amount of blood in the cerebral capillaries. The condition is not in any way associated with the primary phenomena of inflammation.

What has already been mentioned in the case of hyperemia of the cord is equally true in this case—viz. that while congestion undoubtedly may take place, there is nothing symptomatically pathognomonic in the fact, and hence we do not recognize it as a definite clinical entity. "Congestion of the brain" is rather a "diagnostic haven" and satisfies the patient, while at the same time, provided the assumption is not made on too superficial evidence, it harms no one. Two forms of hyperemia have been described:

1. **Active hyperemia** is met with in men more frequently than in women, and results from over-action of the heart and widespread obstruction to the circulation, as when the surface capillaries contract, or there is arterial dilatation, due to excessive mental activity from any cause or to drugs—alcohol, amyl nitrite, nitroglycerin.

2. **Passive congestion** is met with in cases of obstruction of the cerebral sinuses and veins, and is due to pressure on the superior cava or the innominate or jugular veins by tumors or aneurysms; also in suffocation and strangling, in cases of excessive strain, and in tricuspid insufficiency.

Pathology.—There are no marked changes in the brain in these cases. In the active form the gray matter will appear somewhat darker than normal, and a macroscopic section of the white matter shows the puncta vasculosa to be increased. It often leaves no trace *postmortem*. In passive congestion the veins and sinuses are engorged and more or less edema may be present.

The **symptoms** are described under two headings—1, those of *irritation*, and, 2, of *depression*. Among the former are *headache*, *vertigo*, *irritability*, *rapid pulse*, *restlessness*, *insomnia*, and special nervous phenomena, as *flashes of light*, *hyperacusis*, and even *convulsive movements*. The latter is manifested by the obtunding of the senses; in fact, the antithesis of the other. The cerebral symptoms met with in febrile processes are probably due either to faulty metabolism or to some toxin, and should not be regarded as the result of hyperemia.

Treatment.—The recumbent posture is of great importance. Leeching, wet-cupping, and venesection are sometimes employed. Cold applications to the head, bromids internally, and attention to the bowels will be of assistance. Freedom from annoyance and worry of all kinds is necessary.

ANEMIA.

Definition.—A condition in which an insufficient amount of blood circulates in the cerebral capillaries.

It is due to exhausting discharges (diarrhea), an abnormally slow pulse or weak heart, to hemorrhage, obstructive endarteritis of the vessels supplying the brain, and to syncopal attacks and dilatation of the intestinal vessels, owing to the too rapid withdrawal of ascitic fluid.

Pathology.—The gray matter is quite pale; the puncta vasculosa are diminished, and sometimes cannot be seen; the cerebro-spinal fluid is frequently increased.

Symptoms.—The most exaggerated type is met with after a profuse hemorrhage. There are *pallor*, *weakness*, *vertigo*, *headache*, *flashes of light*, *subjective noises*, *rapid respiration*, *cool skin*, possibly profuse sweating, and in extreme cases *coma*. *convulsions*, and *death*. We are more familiar with the ordinary fainting-attack. When cerebral anemia is brought about more slowly "irritable weakness" results. The patient is either *somnolent*, *dull*, and *apathetic;* or he may be a victim of *insomnia*. *Headache*, *vertigo*, *tinnitus aurium*, *musca volitantes*, and lowered muscular power are present. The patient becomes irritable on the slightest provocation. Marshall Hall has described a group of symptoms as "hydrocephaloid" from their resemblance to hydrocephalus; they occur especially in young children after diarrhea. There are *pallor*, *hebetude*, *contracted pupils*, and *depressed fontanels*. The somnolence may deepen into a coma that often becomes more profound until death results.

The **treatment** varies with the cause. The recumbent posture is always indicated, and in some cases it is necessary to depress the head, administer stimulants, and even transfuse or inject a normal saline solution. A light and easily assimilable diet should be given during convalescence.

EDEMA OF THE BRAIN.

Definition.—An infiltration of serum into the subarachnoid space and a greater or less increase of ventricular fluid, with or without infiltration into the brain-substance.

Pathology.—The fluid is chiefly in the meshes and beneath the membrane. The ventricular fluid is increased in amount; the brain-substance is pale, and in some cases infiltrated and softened. Microscopically, lacunæ may be seen in the cerebral tissue, the perivascular spaces are dilated, and some slight degree of nerve-cell degeneration is often present.

Etiology.—Edema is met with in Bright's disease, in senile cerebral atrophy, and as a result of active or passive hyperemia.

Symptoms.—In general the symptoms are those of *anemia* though nothing definite is known of them. Since the condition is always secondary, it may be that symptoms directly referable to the edema are masked by the primary condition. Cases of apoplexy are seen occasionally, in which the only postmortem finding is an effusion of fluid into the pia and ventricles. This has been termed "serous apoplexy."

The **treatment** is that of the primary condition.

EMBOLISM AND THROMBOSIS.

(*Cerebral Softening.*)

Embolism.—**Definition and Etiology.**—The obstruction of arteries or capillaries by material brought to the spot from some other part by the blood-current. The material, generally fibrin, usually comes from the heart, and is either a vegetation of a recent endocarditis or, more commonly, of chronic valvular disease; it may possibly be a fragment of the valve plus the fibrin in ulcerative endocarditis. In the latter case the plug is generally septic, giving rise to suppurative processes. An embolus may be washed from the auricular recesses, from an aneurysm of the aorta or carotid, or from atheromatous patches; rarely from the pulmonary veins.

In puerperal women, and in certain febrile processes (diphtheria and pneumonia) the coagulability of the blood is increased. Heart-clots form, and fragments may be washed into the cerebral vessels. Owing to the direction of the vessels the embolus most frequently enters the left carotid, whence it usually passes to the left middle cerebral. Almost any cerebral artery may be obstructed, but the cerebellar very rarely. Embolism occurs most frequently between the tenth and fortieth years of life. The middle cerebrals are most frequently involved, and next in order the internal carotid and anterior cerebrals.

Pathology.—That region of the brain that is cut off from its blood-supply by the embolus undergoes softening. The cortical changes are less marked than those of the central ganglia, since in the former case more or less anastomosis exists, and none in the latter. When the embolus is septic one or more metastatic abscesses result. The degree of softening varies in different cases within wide limits. There may be nothing more than a slight diminution in the consistence, the affected area being somewhat paler than normal, or absolute dissolution may occur, the myelin breaking up into granules, while the tissue becomes infiltrated with serum, and the vessels undergo hyaline or more often fatty change. The color of the part varies with the amount of blood. In recent cases it is red. As the hemoglobin is absorbed a yellow color appears, and soon predominates. Red and yellow softening are found chiefly in the cortex. The so-called white softening is met with particularly in the white matter. A variety of red softening in which numerous small hemorrhages exist has been termed capillary apoplexy, while *plaques jaunes* is the term given by the French to a form of yellow softening often seen in the cortex of old people. The ultimate changes depend in a great measure upon the extent of the lesion. If this is small, the granular débris is absorbed, and the proliferation of connective tissue results in the formation of a scar. On the other hand, if large the solid elements are removed, and the cavity that remains contains more or less fluid (a cyst). In many instances fibers, trabeculæ, and even vessels that have escaped destruction, pass through the cyst.

Thrombosis.—**Definition.**—Obstruction of a vessel due to clotting *in situ*. This may occur (*a*) in the arteries or (*b*) in the veins and sinuses.

IN THE ARTERIES.—**Etiology.**—Thrombosis results from disease of the vessel-wall, atheroma, endarteritis, or syphilitic arteritis, extension from surrounding diseased areas, traumatism, in aneurysms, in depraved blood-states, and at the seat of lodgement of an embolus. Thrombosis of a cerebral vessel may rarely follow ligation of the carotid. In general we may say thrombosis results from (1) changes in the vessel-wall, (2) retardation of the blood-current, and (3) hypercoagulability of the blood. It occurs most frequently in the middle cerebral, basilar, internal carotid, and vertebral arteries.

Pathology.—The changes in the brain-tissue are precisely those described under Embolism. Within the vessel a clot is found adherent to the vessel-wall, and extending from the nearest large branch on the proximal side to the contracted branches on the distal side. If of recent and rapid formation, it is always of a red color. The slower the formation the paler the color. Such clots are often laminated. The ultimate changes are contraction and atrophy, or, more rarely, calcification, or even softening and removal, the vessel again becoming patulous.

IN THE VEINS AND SINUSES.—**Etiology.**—Thrombosis may be (1) primary, due to general causes, or (2) the result of local changes.

Primary thrombosis is less common than the secondary variety. It is met with in marasmic children (one of the causes of infantile hemiplegia—Gowers), in which the clot is called marantic thrombosis, cachexia, phthisis, carcinoma, and in blood-dyscrasiæ (anemia, chlorosis).

Secondary thrombosis usually results from an extension of neighboring forms of inflammation, caries of the bone, middle-ear disease, or meningitis. It may also be due to fracture of the skull or compression of a sinus by a tumor.

Pathology.—In primary thrombosis the most common seat is the superior longitudinal sinus. From this it spreads into the veins of both sides, and frequently also into the lateral sinuses of one or both sides. In secondary thrombosis the sinus nearest the local disease suffers. The veins emptying into the sinus involved become distended, often rupture, and in consequence the brain-tissue and pia become infiltrated. When the veins of Galen are blocked serum escapes into the ventricles. Red, yellow, and white softening is met with as a final result of the extravasation. Secondary thrombi are usually septic, and give rise to abscess formation.

Symptoms.—**Following Embolism or Thrombosis of Arteries.**—The symptoms necessarily depend upon the position and extent of the lesion. Often it is discovered *postmortem*, not having been suspected during life. We meet with many such cases occurring in late adult life. Then, too, extensive lesions may occur in those portions of the brain that never yield any localizing symptoms—the frontal region, for instance.

Apart from the etiologic differences, the clinical pictures of embolism and thrombosis differ as follows: In the former the *onset* is sudden, without premonitory signs, and is in many cases accompanied by loss of consciousness. In addition to symptoms arising directly through implication of the particular part involved, there are those of *shock*. In the less severe cases consciousness soon returns and the apoplectic symptoms pass off. When more severe, *coma* supervenes and may prove fatal. When hyperemia occurs in or about the motor region the irritation may give rise to *convulsions*. In other cases *delirium* is a prominent feature; hence three varieties of softening are described by some writers—the *apoplectic*, *convulsive*, and *delirious*, from the prevailing feature. Thrombosis may commence abruptly, but as a rule the onset is slow, the patient meanwhile complaining of vague pains, numbness, tingling, headache, and vertigo. It is observed that a gradually increasing impairment of the mind is going on, and that motor weakness, slight at first, increases until the function is lost. The special symptoms are, as stated, dependent upon the location of the obstruction. If this is in the middle cerebral artery, the most common seat, there will be *hemiplegia*, owing to destruction of the internal capsule. The trunk may be spared and one of its branches stopped. The latter run to the third frontal, ascending parietal, supramarginal, angular, or temporal gyri. Thus, then, we can account for the aphasia so often met with in these cases by the plugging of the branch that supplies the third frontal convolution of the left side. If both middle cerebrals are plugged, symptoms develop that are indistinguishable from hemorrhage into the ventricles. This condition is generally fatal. Thrombotic obstruction of the anterior and posterior cerebral arteries rarely causes symptoms, owing to compensatory circulation. " Hebetude and dulness of intellect may occur " (Osler), with obstruction of the anterior cerebral. Hemianopsia may arise from a lesion of the posterior cerebral, since it supplies the cuneus. The left cerebral is more often involved than the right. In either case bulbar symptoms develop.

Cerebellar softening is rare. When it does occur it is usually in the region supplied by the posterior cerebral artery. There may be no symptoms if only one hemisphere is involved; otherwise they are similar to those of cerebellar disease due to other lesions.

Thrombosis in veins and sinuses causes variable symptoms. Those directly due to the vascular disturbance are severe headache, optic neuritis, delirium, or convulsions, and, later, great depression. Hemiplegia may result. When the superior longitudinal sinus is affected, epistaxis is common, while in lateral-sinus involvement post-auricular edema occurs. In secondary cases, moreover, we have to reckon with the cause. Since this is so often septic, septicemic symptoms are the rule.

Treatment.—*Of Embolism and Thrombosis of Arteries.*—Very little can be done in brain-softening. In the early stages, however, while it is absolutely impossible to repair the tissue already damaged, an effort should be made to prevent the spread of the process. Rest in bed with the head slightly elevated should be insisted on. When shock is present it must be met by gentle stimulation, ammonium carbonate, and even by alcohol and digitalis in some cases; hot-water bottles may be applied to the body. Venesection is contraindicated. The bowels should be made to move gently and purgation should be avoided. Later, as stated, symptoms of irritation often appear. In such cases the bromids should be given, and also a diaphoretic mixture, or ice should be placed to the head. In any case in which syphilis, rheumatism, gout, chorea, or other malady capable of causing or adding to the trouble exists, the original disease should be treated promptly and thoroughly. In the mean time efforts should be made to improve the patient's general tone by the strict observance of hygienic and dietetic rules.

Of Thrombosis of Veins and Sinuses.—Treatment in these cases depends largely on the cause. In the primary form it is that of the systemic disease. Good, wholesome, and easily assimilable food should be given, together with a tonic treatment. In secondary thrombosis careful search should be made for pent-up inflammatory products, which should be liberated at the earliest possible moment. The emunctories must act freely. Counter-irritation, applied to the neck, is of questionable value, but internally quinin, iron, and strychnin, and, if stimulation is necessary, ammonia and alcohol, will all be useful.

VASCULAR DEGENERATION.

Arterial.—The cerebral arteries undergo a more or less decided degenerative change in the majority of people past middle life (Bichat said seven-tenths). It is met with much earlier, however, as a result of disease. Bright's disease, rheumatism, gout, alcoholism—in fact, any irritation of the vessel-wall, whether autogenous, the result of faulty metabolism, or whether introduced from without, as alcohol—is capable of bringing about a change of the inner seat of the vessel, to which Virchow gave the name "*endarteritis deformans.*" The circle of Willis and its branches are the most frequent seats. Various stages may be met with in different vessels or even in the same vessel—viz. hyaline degeneration, fatty degeneration, liquefaction-necrosis, atheromatous ulcers, and calcification.

Syphilitic arteritis is not a true degenerative process. It is rather a proliferative process in which both intima and adventitia are involved.

Venous.—The veins are less liable to disease than arteries, possibly because they are more yielding, yet the same pathologic changes may be met with in them. They are more commonly damaged by extension of inflammation from neighboring tissues or by pressure.

Aneurysm.—Dilatation of a vessel results from any of the causes above mentioned. The aneurysms may be very small—miliary—or often as large as a filbert-nut, and rarely as large as a hen's egg. They occur more commonly in males than in females. The middle cerebrals and basilar are most frequently attacked, and next come the internal carotid, the vertebral, and the anterior and posterior cerebrals. Miliary aneurysms are frequently found in enormous numbers upon the basilar branches of the cerebral arteries.

Symptoms of Aneurysm.—There may be none; but in any case they are due to pressure exerted by the mass, and are therefore comparable to tumors of the brain. In many cases the first evidence of any trouble is an *apoplectic attack*, and it is scarcely necessary to add that this is usually fatal. In other cases *headache*, *vertigo*, and *optic neuritis* are present, and more rarely a *subjective murmur*. Still more rarely an *objective murmur* may exist.

INFLAMMATION OF THE BRAIN.

(*Encephalitis.*)

Definition.—Encephalitis, strictly speaking, is an inflammation of the brain-substance, and does not include inflammation of the meninges, though in many instances the two conditions coexist as the result of a common cause, or one may precede and give rise to the other. Encephalitis is met with in two forms—(*a*) Focal, and (*b*) Diffuse.

FOCAL ENCEPHALITIS.

(*Abscess.*)

Pathology.—In very acute cases no time is given for encapsulation; when of longer duration, however, the abscess is well circumscribed, having a well-defined wall, within which there are cell-detritus, pus, and sometimes more or less altered blood. It may be offensive. About it the brain-substance is generally softened and edematous. The abscess is generally single, except in pyemic cases, and varies greatly in size in different instances.

Etiology.—Abscess of the brain is a more or less circumscribed process, due to (1) *Injury.*—In the majority of cases of abscess following head-injuries either a compound fracture of the skull exists, with or without *hernia cerebri* (fungus cerebri), or a punctured wound has been made. Less commonly it may follow a simple fracture, and rarely it is said to occur when neither a fracture nor even an abrasion of the scalp has been produced. Meningitis is an almost invariable concomitant. (2) *Extension from some neighboring inflammatory focus*, as from orbital, nasal, or aural disease, in which the bones have usually become affected. (3) *Pyemia*, in which case the abscesses are apt to be small and multiple. It is also met with occasionally in gangrene of the lung, bronchiectasis, ulcerative endocarditis, suppurative hepatitis, or bone-disease, and rarely in chronic septic processes. (4) *Congenital Heart-disease.*—Little is known of this condition. Within the past two or three years Northrup, Packard, Sir Dyce Duckworth, and Osler have reported such cases. (5) *Obstruction of an artery, vein, or sinus*, whether of spontaneous origin or the result of ligature, may give rise to abscess. Generally, however, the cerebral change is that of softening, and not of true suppuration. (6) *Intracranial tumors.* (7) *Infectious fevers.*

Symptoms.—These at first are generally vague; but in traumatic cases, and more especially in those in which a compound fracture of the skull has resulted, the course may be most acute, and *fever*, *headache*, *delirium*, and possibly *vomiting* may be seen quite early. These are followed by other evidences of irritation, soon by compression with *convulsions*, and then by coma and death. In the more chronic cases the symptoms depend upon the size and location of the abscess and whether or not a vent exists. In such cases an intermission in the symptoms is occasionally met with, due to filling and emptying of the sac. Apart from the headache, pyrexia (not always present), twitching, and drowsiness, that occur in the course of these cases, more or less *hemiparesis* commonly exists, except in abscess of the frontal lobes. The latter are spoken of as "silent regions." An abscess may be "latent," however, in almost any region, these latent abscesses being typified in certain cases of congenital heart-disease. I do not think they were suspected during life in any of the cases reported thus far, and therefore optic neuritis has not been looked for; in other cases this latter symptom is commonly present.

Diagnosis.—In the acute cases following injury little difficulty presents as a rule, though even in this group a latent period may exist. With such a history, however, the onset of headache, fever, delirium, and convulsive movements is decidedly suspicious, and, should optic neuritis also exist, practically no doubt can remain. When dural or nasal disease exists the head-symptoms should be carefully studied, since they are prone to develop in ear-disease soon after a cessation in the discharge.

Differential Diagnosis.—Brain-tumor usually runs a more chronic course, and is seldom accompanied by fever, at least not until its final stage. It may be impossible to differentiate cerebral abscess from meningitis, and the two conditions often coexist, as already stated.

The **prognosis** is always grave.

Treatment.—When an abscess is diagnosed immediate operation is indicated. Suspected cases may be treated symptomatically unless focal symptoms develop. It must be remembered, however, that in a great many cases no localizing symptoms appear, and, since we know that most abscesses occur either in the temporo-sphenoidal lobe or in the cerebellum, when we have reason to suspect the presence of one, these regions should be explored.

DIFFUSE ENCEPHALITIS.

A good deal of doubt exists in the minds of some as to whether diffuse encephalitis ever exists except as a result of traumatism. Certain it is that it is less common and much less is known about it than of inflammation of the cord. We meet with it especially in the frontal regions in certain cases of general paralysis of the insane. The changes often escape the unaided eye. Microscopically, the vessels will be found injected and the perivascular spaces distended with leukocytes; these latter escape into the surrounding tissue, which becomes softened and edematous.

Symptoms.—It is manifestly impossible to give a definite symptomatology in the present state of our knowledge. Excepting the traumatic cases the symptoms are, as a rule, chiefly psychic.

Acute hemorrhagic encephalitis is a condition described by Strümpell. The brain and soft membranes are hyperemic and slightly edematous, and throughout the brain-substance there are numerous punctate hemorrhages. Perivascular leukocytic infiltration is also present. The *etiology* is unknown; but in some cases the disease appears to follow influenza. The *symptoms* are grave from the first. There are intense headache, fever, unconsciousness, disturbances of motility that are usually hemiplegic in type, and finally death. In less acute cases there may be delirium, rapid emaciation, and symptoms of involvement of the cranial nerves. The *diagnosis* can rarely be even suspected. The *prognosis* is invariably fatal, and no *treatment* is of any avail.

Acute polioencephalitis, so-called, is a disease of childhood of doubtful pathology. In some of the cases after recovery from the acute process insular sclerosis or porencephaly is present, with secondary degeneration in the pyramidal tracts. The *etiology* is obscure. The *symptoms* resemble those of acute poliomyelitis of the cord; but contractures develop very rapidly in the paralyzed limbs, and reactions of degeneration are absent from the muscles. The *prognosis* and *treatment* are the same as for the disease of the cord.

Non-suppurative encephalitis is by this time well established as a disease entity, but the symptomatology is not yet clearly defined. The *etiology* is indefinite; influenza has preceded some of the cases. The *pathology* is variable; there is usually focal softening, often associated with some vascular alteration, but there may be even cyst formation. The symptoms are those of focal disease of the brain, usually coming on gradually—that is, without apoplectiform attack, and, as a rule, subsiding without permanent alteration. There may be, therefore, monoplegiæ and disturbances of sensation. The general symptoms are headache, delirium, or coma; fever, rarely exceeding 103° and sometimes absent; and convulsions, often focal in character. The *diagnosis* is difficult; in many cases it cannot be distinguished from meningitis, cerebral hemorrhage, or tumor except by the outcome. The *treatment* is confined to rest, and sedative and antipyretic measures. Operation is never indicated.

CEREBRAL HEMORRHAGE.

(*Cerebral Apoplexy.*)

Definition.—Hemorrhage into the brain-substance; bleeding into the meninges is generally embraced in the definition.

Pathology and Etiology.—In intracerebral hemorrhage the blood will be found to have infiltrated the brain-substance, and, if extensive, it may have penetrated into the ventricle. In such cases the white matter is torn asunder, leaving a ragged space that is more or less filled with recent clot and fragmentary gray matter; if the ventricles have been entered, blood may escape from the lowest into the subarachnoid space. In less severe cases the territory involved is less extensive, and the blood may occupy a single space or several small spaces, forming mere separations of the nerve-fibers. Other changes take place according to the duration of the case. The blood changes color and gradually grows lighter, while reactive inflammation about the lesion results in the formation of a wall. The cyst —for such it has become through fatty degeneration of its contents—may remain as such, or when the lesion is a small one connective tissue may form within and a scar result. The larger arteries are generally atheromatous, and an aneurysm is occasionally met with; many miliary aneurysms may be seen in the course of the smaller vessels. It is very seldom that the actual source of the hemorrhage can be discovered.

Secondary degeneration follows a lesion occurring in the motor region (the cortex or internal capsule), so that sclerotic changes can be traced from the cortex through the corona radiata, internal capsule, crura, pons, and medulla to the termination of the fibers in the cord.

Cerebral hemorrhage is generally of arterial or capillary origin. It is rarely venous, and in the latter case is due almost always either to traumatism or to rupture. Spontaneous rupture generally results from extension of some neighboring focus of disease to the vessel-wall.

Andral states that varicose veins occur in the pia and that they occasionally rupture. Capillary hemorrhage may follow the plugging of a large vein, and of the larger vessels any one or more may be involved, but it has been observed that hemorrhage tends to take place at particular places. In more than one-half of all cases the lenticulo-striate artery (Charcot's artery of cerebral hemorrhage) gives way, and damages the lenticular nucleus and internal capsule. Other regions in the order in which hemorrhage occurs are as follows: centrum ovale, cortex, pons, peduncle, cerebellum, optic thalamus, and the posterior and anterior parts of the hemispheres. Hemorrhage into the cerebrum occurs twenty times more often than hemorrhage into the cerebellum; it may take place into the brain-substance, into the ventricles, or into the meninges, the latter form having already been considered. Ventricular hemorrhage in a great number of cases is caused by a more or less extensive laceration of brain-matter, thus permitting the blood to escape into the ventricles. Not only the lateral ventricles, but the third and fourth also, may contain blood.

Symptoms.—Generally the patient is seized without any warning, but in other cases *headache*, *depression*, possibly *choreiform movements*, and more or less *paresthesia* precede an attack. The loss of consciousness is usually the first manifestation, though for a few moments before, motor weakness, with or without spasmodic movements, may exist. In very slight attacks consciousness may be preserved throughout. The cause of the *unconsciousness* is still an open question. Niemeyer regarded it as due to pressure acting either directly upon the convolutions or by limiting the blood-supply. This view is scarcely tenable, however, for unconsciousness occurs even when the hemorrhage is too small to exert pressure, and, moreover, the hemorrhage and loss of consciousness are usually simultaneous. The symptoms are in direct proportion to the extent and position of the hemorrhage. The patient falls, the face is usually congested, one side often expressionless, and the cheek flaps during respiration. Breathing is stertorous and, in grave cases, of the Cheyne-Stokes type; the pulse is generally feeble for a few moments, but soon becomes full and bounding in character. The pupils vary, and may either be contracted or dilated. There is frequently a relaxation of the sphincters, and on raising the limbs it will be found that those of one side offer absolutely no resistance. The *temperature* is slightly lowered at first, but after a few hours rises to, or just above, normal. In grave cases it will either remain low or will mount up to 106° F. (41.1° C.) or even higher. Such cases are usually fatal. *Conjugate deviation* of the head and eyes takes place in some cases, the deviation being toward the lesion and away from the paralyzed side; in pontine hemorrhage the opposite to this occurs. As a rule, the symptoms that we group under the term apoplexy—viz. loss of consciousness, motor power, and sensation, with or without relaxation of the sphincters—pass off in twelve to twenty-four hours. In fatal cases the coma deepens, but death rarely ensues under twelve hours. In hemorrhage into the medulla or ventricles it may be more rapid.

In from twenty-four to forty-eight hours after the onset *febrile reaction* sets in, with irritative symptoms due to the inflammatory changes occurring about the original lesion. There are fever, often delirium, twitchings or spasmodic movements of a more pronounced type, and sometimes rigidity in the affected limbs. The temperature of the paralyzed side is often from one-half to two degrees higher than the temperature on the sound side. *Trophic changes* in the form of vesicles, or even sloughing, may occur. Death may take place during this stage. Cases are generally fatal also in which a second "stroke" follows closely upon the first, indicating a fresh hemorrhage. After the reactionary period a stationary period follows; sooner or later control of the damaged membranes is then gradually, but not perfectly, regained. The degree of recovery is dependent upon the resumption of function of slightly damaged tissue or upon the compensatory activity of the other side of the brain. In well-marked cases the movements of the affected side are subsequently ataxic. In certain cases the structural damage has been too great, and permanent paralysis remains, with rigidity, wasting, and secondary contractures.

Ingravescent Apoplexy.—In certain cases the onset is slow, consciousness being lost gradually. Coma deepens and the case, as a rule, terminates fatally.

Simple Apoplexy.—The term "simple apoplexy" is almost obsolete.

Serous Apoplexy.—The cases present clinical evidences of apoplexy, but the only *postmortem* finding is an excess of serum, and this is in no way responsible for the apoplexy. These cases probably belong in the same category as those just mentioned, but occur in old persons whose brains have atrophied.

Hemiplegia.—When this is complete one side of the face and the arm and leg of one side, generally the same, are all involved. The facial palsy is not complete, the frontalis and orbicularis oculi escaping. The tongue when protruded deviates toward the paralyzed side. As a rule the arm is affected to a greater extent than the leg, and, indeed, in some cases the face and arm may alone be paralyzed. The trunk-muscles always escape, possibly owing, as Broadbent suggested, to the functional unison of the spinal nuclei of *the two sides* that preside over them, and, since they habitually act together, he supposed that they might be stimulated from either hemisphere.

Sensation is, of course, absent during the period of unconsciousness. Subsequent sensory disturbances are not constant for all cases. Spots of anesthesia often exist for a brief period, but hemianesthesia is rare. Occasionally dissociation of sensation is present, tactile sensation being preserved, whilst muscular and thermal sensation is lost or diminished. The stereognostic sense is often seriously disturbed in these cases. A lesion in or about the posterior part of the posterior limb of the internal capsule is specially prone to give rise to disturbances of sensation.

The special senses may be temporarily perverted or their functions in abeyance, but rarely do permanent disturbances occur. Transient crossed hemianopsia usually occurs, even if the optic tract has not been directly involved. More or less mental deterioration may be permanent, however. The deep reflexes are increased on the paralyzed side, and the superficial reflexes are absent.

Crossed Hemiplegia.—When a lesion occurs in the lower part of the pons the fibers of the facial nerve that are involved have already decussated; hence facial palsy occurs on the same side as the lesion. The fibers coming from the cortex are implicated before their decussation, so that paralysis of the limbs occurs on the side opposite to the lesion. Lesion of the crus may lead to oculo-motor palsy of the same side, and palsy of the face, arm, and leg of the opposite side.

Course and Terminations.—As previously intimated, the course depends on the position and extent of the lesion. In the most extensive cases death rarely takes place under several hours. Hemorrhage into the medulla may prove fatal more quickly. In the slightest cases, perfect recovery may take place in a few days or weeks. Generally, however, when little or no improvement occurs in two or three months, permanent changes result. The facial muscles soon recover, and next the leg, while at first the patient is able merely to move the toes. Daily improvement then follows until he can support his weight; dragging of the foot, however, is marked at first, and rarely disappears absolutely. In the mean time a less pronounced change for the better has been taking place in the arm. This member very rarely recovers, however, to the same extent as the leg, and secondary contractures develop in time, the arm and hand becoming flexed, whilst the leg is extended. The hand is usually bluish and cold, and swells if kept in a dependent position. More or less ataxia is constant, and rheumatoid pains are apt to occur during this stage. Other late manifestations that are only occasionally met with are athetosis, arthropathies, post-hemiplegic chorea, and tremors.

There is no wasting of the affected muscles as a rule; nor are there electric changes, except during the irritative period, when the response to stimulation is heightened. Occasionally marked atrophy occurs, and is due in some cases, as Charcot has shown, to changes in the cells of the anterior horns. In others no such change is found, and we are forced to regard the wasting as cerebral.

Differential Diagnosis.—Apoplexy is to be distinguished from other conditions causing unconsciousness, such as cardiac syncope, epilepsy, alcohol or opium-poisoning, insolation, or uremia. If some previous history can be obtained, the difficulty of the diagnosis is lessened, though it may still be great. In *syncopal attacks* the pulse is very feeble and the face is pale, respiration being shallow and often suspended. The sphincters are hardly ever relaxed; the reflexes are usually preserved, and the skin is often moist. In *epilepsy* there is a history of previous attacks, or, failing to obtain this, one can usually learn that a convulsion has immediately preceded the coma. With *alcoholism* the case is more difficult. The odor of alcohol on the breath is of no value, as spirits may have been given by a bystander; moreover, hemorrhage is common in alcoholics (*vide* table of differential diagnosis, p. 969). In *opium-poisoning* the coma comes on gradually, and when not too profound the patient can be aroused when shaken or shouted at. The respirations, which are very slow and deep at other times, become somewhat quicker and shallower when he is aroused. In *insolation* the temperature suffices as a rule, though, as stated, high temperature may occur in apoplexy. The presence of albumin is not conclusive evidence of *uremic poisoning* unless the centrifuge and the microscope reveal the presence of casts or other indications of renal change; even then the case may be one of apoplexy in a subject of nephritis. In the case of *diabetic coma* the presence of sugar in the urine serves to make the diagnosis. When we meet with a comatose case in which there is absolutely no resistance when the limbs of one side are raised, while those of the other still exhibit some tonicity, particularly if the deep reflexes are exaggerated on the flaccid side, the probability is that it is an apoplectic attack. It is generally not possible to tell whether the condition is due to hemorrhage, embolism, or thrombosis, though the tabulated points of distinction (after Leube, *vide infra*) will afford material aid:

EMBOLI.	HEMORRHAGE.
Early adult life.	Late adult life; in early life very rare.
Previous development of atheroma, cardiac disease following acute rheumatism, sepsis, chronic valvular disease, fatty heart, general cardiac weakness, aneurysm.	Atheroma with cardiac hypertrophy.
Detection of emboli in other organs.	History that the patient up to the time of attack was well; also the finding of casts in urine and other symptoms of chronic nephritis.
During the attack there is an absence of congestion of the face; the pulse is normal; in cardiac affections it is accelerated and irregular.	During the attack there are noted flushes (reddish) of the face, pulsating carotids, and slow pulse.
Temperature normal, but shortly after the attack it begins to rise, without making an unfavorable prognosis.	Temperature during the attack is subnormal, but just previous to death there is an *antemortem* rise.
The attack, as a rule, is short; if there is a protracted embolic infarction, the duration is long; usually the circulation adjusts itself promptly.	The duration is, as a rule, longer. Coma of long duration (about two days) gives a very unfavorable prognosis.
Remote effects are infrequent. Hemiplegia is right-sided, with aphasia.	Remote effects quite frequent; alteration in the urine—albuminuria, polyuria.
Ophthalmoscopic Examination.	
At times the ophthalmoscope reveals either a recent or an old embolus in the arteria centralis retinæ.	The retinal arteries may show various stages of atheromatous degeneration; as a result there may be a hemorrhagic retinitis or there may be a thrombus of the central vein of the retinæ. In a few instances, in which the hemorrhage occurred in the ventricle, the pupils were contracted.

It is not an uncommon occurrence to have patients brought to a hospital dazed and smelling of liquor. These should always be carefully watched, for mistakes readily occur, and many such cases have been condemned to a prison-cell when they were really suffering from cerebral hemorrhage.

Prognosis.—Sufficient has already been said on this point.

Treatment.—The patient should be kept as quiet as possible and in the recumbent position, with the head somewhat elevated. The clothing about the neck should be loosened to prevent constriction. An ice-bag may be put to the head and hot bricks or a hot-water bottle to the feet, while sinapisms may be placed on the back of the neck or on other parts of the body. If the pulse is strong, full, and incompressible, and the face is congested—venesection is probably justifiable, particularly if the age and condition of the vessels support the idea that hemorrhage is taking place. It must not be done without consideration, however, since it would be useless in embolism and thrombosis. The bowels should be made to move freely; a cathartic may be exhibited by the mouth (croton oil, gtt. j or ij), and at the same time an enema may be given. When consciousness returns the patient should be kept absolutely quiet for several days, and only liquid food permitted. Later an endeavor should be made to keep up the tone of the affected muscles by massage and electricity. It is questionable if the iodids or any other drugs have an influence over the subsequent changes.

APHASIA.

Definition.—Impairment or total suppression of the power of speech, due to cerebral disease. This is a complex subject, and cannot be more than touched upon here; but the chief disturbances will be briefly mentioned, omitting any further description of the form due to disease of the bulb (*anarthria*), since it has already been dealt with in speaking of Bulbar Palsy (*vide* p. 1098).

The majority of cases of aphasia are met with in connection with hemiplegia. They are apt to be more marked in the beginning, owing to the general obtunding of the psychic processes that is induced by the shock. Speech is the expression of thought in words, and is the result of external stimulation in which the impulse awakens in the mind a recollection of similar impulses that have preceded it—*e. g.* the sight of a dog, sound of a bell, or certain odors (*vide* Fig. 70). Bastian, however, believes that it is not necessary to postulate the existence of a separate center for conceptions or ideas. He believes that a better knowledge of the functional activity of the auditory and visual word-centers obviates it, and gives the following three ways in which the perceptive center may be called into activity: (1) By voluntary recall of past impressions, as in an act of recollection; (2) by association—that is, by impulses communicated from another center during some act of perception or during some thought-process; and (3) by means of external impressions.

Two principal forms of aphasia exist: (1) *Motor*, and (2) *Sensory*. I shall consider these forms of aphasia separately, although before doing so it seems necessary to indicate by means of the accompanying schematic diagram (Fig. 71) the different paths on which the individual acts for the occurrence of speech are fulfilled. This will also serve to furnish an easy explanation of the symptomatology of the individual forms of aphasia and the means by which their discrimination (from each other) is possible.

Motor Aphasia.—As stated, we meet with aphasia most frequently in hemiplegia. It is also met with in certain cases of embolism and thrombosis and as a result of softening from any cause; also in certain cases of brain-tumor and in deaf-mutes. The latter cases are not strictly included in the definition given, for, while brain-injuries at birth may and do occasionally cause aphasia either by damaging the brain as a whole (imbecility) or in a more circumscribed fashion, I refer particularly to those cases in which the child is aphasic because he is deaf, for unless trained through his visual or tactile sense he has no memory-picture of words from which to draw. A child may acquire the power of speech and retain it for several years, and then become deaf. He will then receive no more auditory impressions, and, unless trained as in the preceding group, will become aphasic. Certain cases of congenital speech-defect have been described by Hodden under the name "idioglossia." Here the children utter peculiar sounds, constant for the same words. As a rule, their intelligence is not up to the normal. In hemiplegia the lesion is usually in the motor, or emissive, center in the foot of the third frontal convolution (Broca's region). In right-handed people this is on the left side of the brain, and on the right side in left-handed people. Bastian believes Broca's region to be sensory and not motor (*vide* Sensory Aphasia, *infra*). A lesion in Broca's region causes the loss of voluntary speech, nor can the person repeat words after any one or read aloud; but he can understand when spoken to, and, unless his arm is paralyzed, can usually write from a copy. It is rare, however, for a lesion to involve so strictly limited a portion of the cortex. As a rule, adjacent parts or commissural fibers between neighboring motor centers are involved, resulting in "combined motor aphasia." In either case automatic speech is perfect, and oaths or other expletives may be uttered, songs sung, or poetry recited unless the oro-lingual centers are damaged.

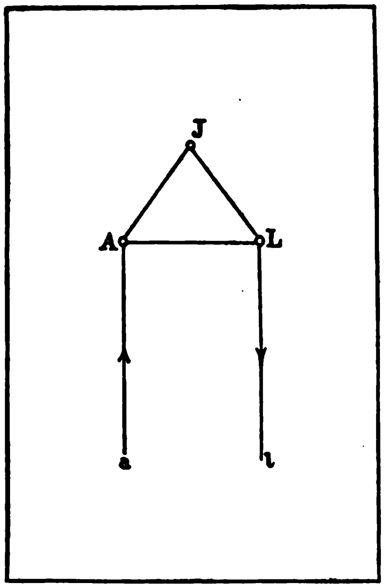

Fig. 74.—Diagram for the explanation of the process of speech (Leube): *A*, auditory perceptive center (center for the recognition of sounds); *a A*, acoustic tract (auditory tract); *L*, center of motor speech; *L l*, motor speech tract (path for the innervation of the muscles of phonation); *J*, center of ideation (higher concept center).

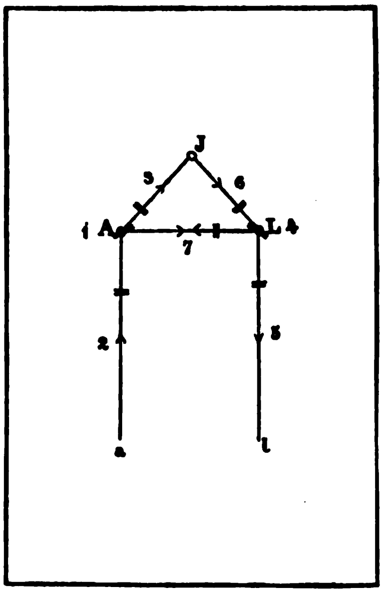

Fig. 75.—Diagram for the explanation of aphasia (Leube): *A*, *a A*, *L*, *L l*, *J*, the same as in Fig. 71; *A J L A*, circle by which speech is controlled; *a A L l*, path used for automatic speech; *J L l*, path used for voluntary speech; *a A J*, path used for the recognition of words; —, interruption of conduction; 1–3, forms of sensory aphasia (1, cortical; 2, subcortical; 3, transcortical); 4–6, forms of conduction-aphasia (1, cortical; 2, subcortical; 3, transcortical); 7, conduction-aphasia (amnesic aphasia).

Some observers claim that a special center for writing exists at the foot of the second left frontal convolution. Dejerine denies this. In some cases the patient comprehends when spoken to, and even speaks himself, but he misplaces words, and hence fails to convey his meaning. The lesion in such cases of paraphasia is believed to be in the commissural fibers uniting the frontal and temporal lobes, and is called "Wernicke's aphasia of conduction."

Sensory Aphasia.—This comprises various auditory and visual disturbances of speech.

(*a*) **Auditory Disturbances.**—The cortical center for auditory impressions is in the posterior part of the first and second temporal convolutions. That this region represents several auditory impressions—viz. that for words, music, and, in fact, sounds of any kind—we have abundant clinical proof. Cases are reported in which the patient recognizes all but spoken words. He can read and write, but when spoken to, hears the words just as one may hear a foreign language, but cannot understand. He cannot, therefore, write to dictation. This is "word-deafness." Prof. Edgren of Stockholm has reported cases in which the patient is tone-deaf. The sound is heard, but is not appreciated as music. While these various auditory impressions may occur singly, we most commonly meet with a condition in which they are all lost—"mind-deafness"—and in every case, except those possibly of the most trivial kind, more or less apraxia (loss of knowledge of the use of things) exists contemporaneously.

Bastian gives four centers for word-memory as follows:

(1) Auditory memory, the memory of the sound of words, situated in the posterior part of the first temporal convolution.

(2) Visual memory, the memory of the visual appearances (printed or written) of words, situated in the angular and supramarginal gyri.

(3) Kinesthetic memory, the memory of the different groups of sensory impressions, resulting from the mere movements of the vocal organs during the utterance of words (impressions from muscles, mucous membranes, and skin). This he places in Broca's region, and terms the glosso-kinesthetic center.

(4) The cheiro-kinesthetic center, not definitely located, he regards as the memory-center of the different groups of sensory impressions emanating from muscles, joints, and skin during the act of writing individual letters and words.

Bastian does not regard these four centers as sharply defined. He says on this point: "Although I am not a believer in the complete topographic distinctness of the several sensory centers in the cerebral hemisphere, I consider it clear that there must be certain sets of structurally related cells or fiber-mechanisms in the cortex whose activity is associated with one or another of the several kinds of sensory endowment." Such diffuse but functionally unified nervous networks may differ altogether from the common conception of a neatly-defined "center," and yet for the sake of brevity it is convenient to retain this word, and refer to such networks as so many "centers."

(*b*) **Visual Disturbances.**—The visual perceptive center of Monk is situated in the occipital lobe in the region of the cuneus and of the calcarine fissure. The visual memory-centers are in the angular and supramarginal convolutions. By the visual memory-center is meant that part in which are stored memories of things, faces, or places. Memory is no longer regarded as a definite and fixed unity, but rather as a number of memories, each being closely related cortically. Of this also we have abundant clinical proof. Kussmaul was the first to point out that blindness for words can exist as a separate entity, and for this he proposed the term "word-blindness." Letter-blindness can also exist alone. Charcot speaks of a patient who knew Latin, Greek, German, French, and Spanish, but lost the memory of some of the Greek and German letters only. In other cases the patient may recognize the letters, but cannot form them into words or sentences. Note-blindness, according to Professor Edgren, is a variety of amnesia in which the patient can no longer recognize musical notes, though he sees them as well as ever. In still other cases the visual memory for objects, faces, or places may be lost. The following case reported by Serieux is interesting in this connection: A woman aged sixty-two had a stroke, followed by paralysis, and after a second attack epileptic convulsions and hallucinations of hearing. She was found to have deafness and paraphasia, word-blindness, agraphia, and object-blindness (apraxia). She could not recognize the friends nor the objects that once were quite familiar to her. Some time later she died with pneumonia, and *postmortem* softening was found in the left first temporal (posterior part) and in the left supramarginal convolutions; also on the right side areas of softening were noted in the posterior parts of the first and second temporal convolutions and in the angular and supramarginal convolutions. This case illustrates the point of which I have already spoken—viz. the mixed character of the symptoms, due to simultaneous involvement of two or more centers. That this is the more common condition is not alone due to the anatomic propinquity of the various centers, but chiefly to the fact that they are all within the area supplied by the Sylvian artery and its branches. The simultaneous loss of these several visual memories results in the condition known as "mind-blindness." These sensory aphasias are sometimes spoken of as "amnesic aphasia."

Eskridge[1] has briefly summarized this subject as follows:

(1) If the lesion is in the foot of the left third frontal (Broca's) convolution in right-handed persons, and in left-handed persons in the right third frontal, the patient will be unable to speak voluntarily, to repeat words after another, read aloud, think in speech, and generally to write voluntarily or from dictation so as to be understood; but he understands the speech of another. He is usually able to use and to understand gesture-expression.

(2) A lesion in the oro-lingual center (lower portion of the central convolutions) will cause paresis or paralysis of the oro-lingual muscles, including the lower side of the face, and imperfect articulation; but the patient will be able, with a decided effort, to repeat words after another, to talk voluntarily, to think in speech, and to write, although the letters may be imperfectly formed. There is, as a rule, no omission or transposition of letters or words, unless the lesion is sufficiently extensive to affect the adjacent cortical centers or their commissural fibers. It is possible that the center is bilateral; that a lesion on either side may give

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[1] *University Med. Magazine*, Jan., 1897.

somewhat similar symptoms; and that the loss of function in the affected muscles will not be completely abolished by a unilateral lesion.

(3) A lesion in the foot of the second frontal convolution on the left side in right-handed persons, or on the right side in left-handed persons (the probable orthographic center), will be attended by disturbances in writing—inability to write, due to inability to spell. In the only case of of this kind that has been reported the patient formed all letters well, but omitted, transposed, and substituted letters to such an extent that his writing was unintelligible. He wrote well when words were spelled for him.

(4) A limited lesion in the posterior two-thirds of the first and second temporal convolutions is attended by word-deafness, and an inability to write at dictation (pure word-deafness). A more extensive lesion in the same region results in mind-deafness with paraphasia, and some disturbance in reading and writing will be added. The greater the extent of the cortical and subcortical areas involved the more marked the paraphasia and other symptoms of sensory aphasia.

(5) A lesion involving the angular gyrus and adjacent parts will cause word-blindness and inability to read, also defects in writing, copying, and speaking. In these cases paraphasia is often present. If the lesion affects the parts posterior to the angular gyrus, mind-blindness may be added.

INTRACRANIAL GROWTHS.

OWING to their close relationship, new growths, both of the brain and membranes, are here considered.

Pathology.—Rindfleisch has classified intra-cranial tumors according to the tissue from which they spring, thus:

1. Having their origin in the *membranes*, either extra-cerebral or intra-ventricular; these include tubercle, gumma, carcinoma, sarcoma, myxoma, lipoma, cholesteatoma, and psammoma; small fibroids have also been described. Enchondroma and osteoma may arise from the falx or from the bones of the skull.

2. From *blood-vessels*: to this group belong aneurysms, tubercles, and gummata.

3. Originating in the *neurogliar tissue*: glioma.

4. Originating in the *connective tissue*: sarcoma.

I will here consider these new growths in the order of frequency with which they are met.

1. **Tubercle** is most common in children and young adults, and is generally multiple (see Tuberculosis, p. 318).

2. **Sarcoma** is usually of the round- or spindle-celled variety; there may also be melanotic lympho- or fibro-sarcomata. Sarcomata are apt to diffuse themselves through the brain-substance quite rapidly.

3. **Glioma.**—Infiltrating tumors, generally single, and showing no definite line of demarcation from the surrounding brain-structure. They may be soft, even telangiectatic, or quite firm. They often run a very chronic course.

4. **Gummata** are generally small and often multiple. They spring

from the membranes or the adventitia of blood-vessels, or from connective-tissue septa. Frequently they are attached to the periosteum of the skull.

5. **Carcinoma** may be primary or secondary, usually the latter. Secondary growths are generally small and round, but in some cases they perforate the bones of the skull, producing a fungus hematoides.

6. **Fibromata** are not common. They either grow in the membranes or aid in the formation of a mixed tumor, as fibro-sarcoma. Other tumors met with less frequently are as follows: 7. **Osteoma**; 8. **Enchondroma**; 9. **Myxoma**; 10. **Lipoma**; 11. **Angioma**; 12. **Cholesteatoma.**

13. **Hydatids** are rare, especially in America. They may develop in any part of the brain or its membranes, and are said to occur most frequently in children. 14. **Cysticerci** may also occur in the brain or its membranes.

15. **Brain-cysts** are probably most often due to absorption of areas of softening from any cause, but they also occur between the dura and skull, as has been described. The lack of cerebral substance, due either to imperfect development or to atrophy following vascular obstruction or injury at birth, has been termed porencephalia by Hesche.

Etiology.—Age and sex are the chief factors; tuberculosis is far more common in children than in adults, while gummata when found appear almost invariably in adults, as do malignant growths. As a whole, new growths are more common between the twentieth and fortieth years, and males are more often affected than females. Heredity and traumatism may be disregarded as causal factors.

Symptoms.—These are (1) General, and (2) Focal.

General Symptoms.—*Headache* varies in degree and character; it is not of any value as a localizing symptom, nor is tenderness on pressure.

Vertigo in a mild form is quite a common symptom. In cerebellar cases it is often very marked.

Vomiting occurs in most cases, and generally bears no relation to the time of taking food; this constitutes an important point in the diagnosis. The vomiting is apt to be exaggerated in cerebellar tumor.

Optic neuritis is present, according to Gowers, in four-fifths of all cases; in 82 per cent. according to Oppenheim, and in two-thirds according to Knapp. Gray believes this to be misleading in a measure. We are so imbued with the fact that optic neuritis is a symptom of brain-tumor that without it we are apt to overlook the possibility of tumor. He says it is unquestionably present as a rule in the later stages, but the practical question he asks is, "How often is optic neuritis in the earlier stages when the diagnosis is obscure?" Headache, vomiting, and optic neuritis are "classical symptoms" of brain-tumor, and when met with simultaneously are quite characteristic.

The *mental disturbance* is usually slight. Dulness and stupor are most reliable evidences of intra-cranial growth, and especially when occurring with any of the above symptoms. The patient may be emotional or hysteric. Pseudo-apoplexy may occur as the result either of the growth or of hemorrhage taking place about it.

Convulsions are focal (Jacksonian), or general convulsions may occur.

Constitutional and other symptoms may include progressive weakness, loss of appetite and of flesh, pupillary changes, and changes in the pulse, respiration, etc., and possibly slight fever. In certain tumors in the basal ganglia hyperpyrexia is met with. High fever is often significant of meningeal inflammation, as in syphilitic cases.

The **focal symptoms** are of two kinds: first, those due to direct local action (irritation or compression), and second, those due to changes occurring about the growth—indirect irritation, hemorrhage or softening, or merely congestion; thus can intermission or remission in symptoms be explained. The chief regional symptoms are as follows in—

(*a*) Tumors in the *prefrontal region*. *Headache*, not limited to the frontal region, with more or less mental impairment and drowsiness (though this is not constant by any means); and perhaps a disturbance of the sense of smell. No motor or sensory symptoms are present as a rule. The tumor may, however, grow backward, and either encroach on the motor region or cause motor symptoms indirectly. Downward growth would result in aphasia.

(*b*) Tumors in the *motor region*. The early symptoms are irritative and, later, paralytic. The former give rise to *spasm*, which is often very localized at first, possibly in a few muscles (Jacksonian epilepsy). More or less *sensory disturbance*—*e. g.* tingling or anesthesia—is generally present also, thus supporting the view that the motor cortex is also sensory. The point of origin and direction of spread of the spasm are valuable localizing symptoms. Sooner or later destruction of the area causes *paralysis*. We may have spasm in one limb and monoplegia of the other on the same side. Involvement of the left third frontal region causes aphasia.

(*c*) Tumors in the *parietal lobes*. There may be no symptoms. When the posterior part is involved (angular or supramarginal gyri) we may meet with word-blindness or mind-blindness. Sometimes there is astereognosis in the opposite hand.

(*d*) Tumors in the *temporal lobes* may be latent, or there may be disturbances of taste and smell. If the posterior part of the first convolution is involved, we have word-deafness or other psychical disturbance of hearing.

(*e*) Tumors in the *occipital lobes*. A unilateral tumor produces *hemianopia*, while a bilateral lesion may cause blindness. In certain cases, too, mind-blindness results, or "soul-blindness," as it was at one time called.

(*f*) Tumors of the *corpus callosum* are often latent; they may, however, cause unilateral or bilateral motor symptoms. Often some mental aberration is noted.

(*g*) Tumors of the *corpora quadrigemina* and crura may be considered together, since in the former case no distinctive symptoms occur when the tumor is small; when large the crura will be implicated. In such cases there will be oculo-motor paralysis on the same side as the tumor, and hemiplegia on the other. Sensory disturbances may occur also.

(*h*) Tumors involving the *base*, when growing in the anterior fossa, give rise to exophthalmos, disturbances of smell and vision, and possibly to mental impairment. When in the middle fossa the symptoms are chiefly those of involvement of the third and fifth nerve, consisting of ptosis and other oculo-motor symptoms and facial neuralgia. When the tumor involves the pituitary gland, temporal hemianopsia, amblyopia or amaurosis, and frontal headache occur. Tumors have been found in this region in cases of acromegaly (*q. v.*). When in the posterior fossa, facial neuralgia, neuro-paralytic ophthalmia, or seventh or eighth nerve involvement and crossed hemiplegia are met with.

(*i*) Tumors in or about the *basal ganglia*, if quite small, cannot be diagnosed. When of larger size they cause hemiplegia and sensory disturbances by pressure upon the internal capsule, and hemianopia by pressure on the optic radiation. They may also cause obstruction and consequent distention of the ventricles (internal hydrocephalus).

(*j*) Tumors in the *cerebellum* may be latent. They give rise to most marked symptoms when the growth is in the middle lobe. In such cases headache, vomiting, vertigo, and ataxy are pronounced. The gait is of a reeling or staggering character, and there is a tendency to fall in some particular direction—forward, backward, or to one side. In addition there may occur optic neuritis, disturbances of vision, pain and stiffness in the cervical region, and possibly deafness.

(*k*) Tumors of the crus often cause a peculiar type of crossed hemiplegia, in which the face, arm, and leg are involved on the opposite, and the muscles supplied by the third nerve (eye muscles) on the same side. There may also be hemianesthesia.

(*l*) Tumors in the *pons* and *medulla* give rise to cranial nerve-lesions, with or without hemiplegia; also to sensory disturbances, and when extensive they may by pressure cause cerebellar symptoms. Tumors in the medulla may cause cardiac or respiratory failure.

It is often important from a surgical point of view to ascertain whether the growth is cortical or subcortical. No absolute means of doing this exists, though Seguin has formulated a fair working rule—viz. when localized clonic spasm occurs, followed in some cases by epilepsy (this primary spasm Seguin has termed the "signal symptom"), and later by paralysis, with local pain and tenderness and increased local cerebral temperature, a cortical lesion is most likely. On the other hand, when the spasm is tonic, particularly if widespread—involving one side, for instance —and when the local pain and tenderness are slight or absent, and the cranial temperature normal, the lesion is more apt to be subcortical.

Course.—Many cases run a very chronic course. Others may have existed months or years without symptoms, and then develop suddenly, owing to hemorrhage, thrombosis, or acute softening about the tumor. Either improvement may take place or the case may speedily progress to a fatal termination.

Diagnosis.—The general symptoms are usually sufficient to warrant a diagnosis. The gradual onset and progressive character without fever, in the apparent absence of any etiologic factor, are, as a rule, enough to indicate that a tumor is present, while its location can only be determined by the focal symptoms.

The **prognosis** is always grave. Syphilitic growths are almost the only kind in which some gleam of hope may be extended, and this only very rarely; tubercle may recover by the growth becoming encapsulated and calcified. Nothing can be said as to the possible duration of life. Several years may elapse between the appearance of the symptoms and their fatal termination, or death may occur suddenly.

Treatment.—In any case recourse should be had to mercury and the iodids, and this treatment should be pushed, since it will certainly benefit syphilitic cases, and it is believed to be of some value even in the non-syphilitic. Other symptoms should be met as they arise. The question of operation must be considered where medical measures have proved of no avail. Macewen and Horsley in Great Britain and Keen in this country have pointed out its justifiability. If the situation of the growth is favorable and the nature of the tumor is not malignant, an operation is likely to be successful. The percentage of recoveries is increasing as the technic becomes more perfect (see Bruns, *Geschwulste der Gehirn*, etc.).

CHRONIC HYDROCEPHALUS.

THIS affection is divided into external and internal hydrocephalus.

EXTERNAL HYDROCEPHALUS.

Pathology.—When the skull is opened the bone is usually found to be thin; the dura is normal; the arachnoid is lifted from the surface of the cortex by a considerable accumulation of clear fluid of low specific gravity; the convolutions may be somewhat flattened, and the cortex slightly thinned. Upon microscopic examination no changes are found in the brain-substance. Sometimes the effusion is general; sometimes it is sacculated. In a case of epilepsy with very marked neuropathic heredity, upon which I recently performed an autopsy, two large subarachnoid cysts were found beneath the temporal lobes, and smaller cysts in other parts of the brain, flattening or separating the convolutions.

Etiology.—External hydrocephalus may depend upon a congenital smallness of the brain or upon a congenital enlargement of the skull. The space between the brain and the bone is filled by an excess of subarachnoid fluid (*vacuum dropsy*), or there may be a wasting of the brain, such as occurs in old age or in chronic cachectic conditions.

The **symptoms** depend upon the form. In cases in which there is hypoplasia of the brain or in which the brain has wasted, no pressure-symptoms are present. All the manifestations are purely psychic in nature, and similar to those of *microcephaly* or *senile dementia*. In cases, however, in which the cranium-cavity is abnormally large, it is probable that the real cause resides in a congenital excess of subarachnoid fluid.

The **prognosis** is gloomy; nevertheless, it is possible that the disease may undergo spontaneous cure as a result of rupture into the nasal fossa.

The **treatment** is the same as for the internal variety (*vide infra*).

INTERNAL HYDROCEPHALUS.

This is a condition in which one or more of the ventricular cavities of the brain are distended by the cerebro-spinal fluid. In the *congenital form* and in that occurring in early childhood, this is associated with more or less enlargement of the skull. In the later *acquired forms* the cranium does not yield so readily, and the enlargement does not exist or is slight.

The **pathology** of the condition varies with its nature. In the congenital forms, upon opening the head the skull is found to be thin. The fontanels and sutures are either still open and connected only by a membrane, or closed by Wormian bones. The dura may be thickened, but usually is normal; the substance of the brain is slightly softened—although this is not invariably the case—and very much thinned. This thinning is, as a rule, particularly noticeable in the corpus callosum and commissures, which may, indeed, either be torn apart or completely atrophied. The enlargement ordinarily affects the two lateral ventricles, the third ventricle, and the aqueduct as far as its entrance into the fourth ventricle, which is commonly less involved than the other cavities. The ependyma is sometimes smooth, but more often shows small projections, which, according to Virchow, are composed of brain-substance, but in some cases are due to proliferation of the glia tissue beneath the ependyma. The enlargement may not be uniform. If due to obstruction of the foramen of Monro, one or both lateral ventricles are usually enlarged, whilst the third ventricle either remains of normal size or is diminished. If due to enlargement of the pineal gland, the aqueduct does not show the funnel-shaped distention. Another cause upon which considerable weight has been laid is the closure of the transverse fissure between the cerebellum and medulla. The quantity of fluid may be enormous, as much as 4 or 5 liters (5 or 6 quarts) having been recorded. The thinning of the brain-substance is also remarkable when one considers that a cerebrum 5 mm. ($\frac{1}{5}$ in.) in thickness is apparently able to perform a large proportion of its ordinary psychic functions. The atrophy seems to affect particularly the white substance, especially the myelin-sheaths.

In cases of the *acquired form*, unless they occur early in life, the enlargement of the skull is not very noticeable; the substance of the brain shows considerable softening; the ventricles are moderately enlarged, and, particularly in the chronic forms due to tuberculosis, are considerably roughened. The most pronounced cases are those that occur when there is a tumor in the occipital fossa which compresses the veins of Galen. In these cases the accumulation of liquid is slower, the brain yields more gradually to pressure, and the dilatation is more pronounced. Ordinarily, there is considerable flattening of the convolutions. In a few of these cases inflammatory changes in the ependyma have led to partial obliteration of the ventricles, particularly in the anterior horns or the lateral ventricles. Occasionally also bands of organized lymph may cross the ventricles in various directions; the liquid is of higher specific gravity and contains more albumin than in the non-inflammatory varieties.

The **etiology** of the *congenital form* is unknown, though the fact that it frequently occurs in several children of the same family has led to the supposition that it is dependent upon some hereditary influence. In some cases it has been referred to emotional disturbances suffered by the mother during pregnancy, and in still other cases an anatomic foundation has been discovered, such as enlargement of the pineal gland It is generally supposed that the immediate cause is chronic ependymitis.

The acquired form is usually secondary to inflammatory conditions (particularly meningitis) or to brain-tumor. Some cases, however, occur in childhood, that are apparently not due to either of these causes.

Symptoms.—The most characteristic appearance in congenital hydrocephalus is the *globular enlargement of the head.* Upon palpation the fontanels are found to be still patulous and usually bulging, and the sutures are open. The head is usually so heavy that it cannot be held upright, but falls backward or to one side. The face appears proportionately very small. *Motility* is usually disturbed, the legs are spastic, and the child either does not learn to walk at all or only long after the usual time. There are sometimes choreic movements of the upper extremities. The *eyes* frequently show nystagmus and conjugate deviation, and often there is either choked disk or atrophy of the optic nerve. Fischer has described a *systolic murmur* that can be heard if the stethoscope is placed over the anterior fontanel. Its cause is unknown *Convulsive attacks* are common; they are epileptic in type, and, as a rule, ultimately cause death. *Intelligence* is usually considerably impaired. and sometimes the children are idiots; more often they merely show retardation of intellectual development. Occasionally—and this even in the most pronounced cases—the intelligence is well preserved. Henoch records the case of a boy three years of age whose head was 75 cm. (29.6 in.) in circumference, and who could speak both French and German. Ordinarily, the children are quiet and apathetic, but they may be querulous. *Nutrition* is commonly seriously disturbed, the children sometimes exhibiting pronounced cachexia. They may, however, be well nourished and, to a certain degree, vigorous. The symptoms of the chronic form in adults are those of brain-tumor without focal symptoms.

The **diagnosis** is ordinarily very easy. Careless observation may lead to confusion with *rachitis* but the square shape of the head and the presence of other rachitic deformities in the skeleton should lead to a prompt recognition of the true nature of the case.

The **prognosis** is extremely unfavorable, the majority of the children dying about the fifth year. A few cases, however, may live until they reach young adult life, and still fewer apparently recover entirely.

Treatment is of course difficult. Potassium iodid and mercury have been employed without much beneficial effect. Cod-liver oil may be given to stimulate nutrition, and purgatives occasionally relieve pressure-symptoms temporarily. Among the mechanical procedures constant pressure upon the head seems the most valuable. This can be obtained by means of strips of adhesive plaster or by the application of an elastic band. Tapping the ventricles is occasionally followed by temporary improvement, but is always dangerous. More satisfactory is probably the lumbar puncture recommended by Quincke. If convulsions develop, they should be combated by bromids and purgatives.

SCLEROSIS OF THE BRAIN.

THIS may be diffuse or focal. The diffuse form is usually a congenital condition, and leads to a hardening of the whole brain, associated with more or less malformation. If it is secondary to some vascular disturbance in early life, it may be restricted to one or more lobes on one side. The focal form occurs in insular sclerosis, and as a congenital lesion. The latter may be associated with overgrowth or contraction of the neurogliar tissue, and is spoken of as hypertrophic or atrophic gliosis. It is usually associated with epilepsy and idiocy. Neurogliar hyperplasia is also met with in areas in which the nervous tissue has been destroyed, no matter what the cause. It occurs, therefore, in both primary and secondary degeneration. The sclerotic areas vary in size from very small and even microscopic plaques to large and widely-diffused ones. These scleroses have been considered under the respective diseases to which they give rise. Focal sclerosis, due to scar-formation and characterized by an overgrowth of connective tissue, occurs after injury or hemorrhage.

GENERAL PARALYSIS OF THE INSANE.

(*General Paresis; Dementia Paralytica.*)

Definition.—A chronic disease involving both the cerebrum and the meninges, and characterized by a gradual loss of power, tremors, and progressive mental decay.

Pathology.—The intima and adventitia of the blood-vessels undergo proliferative changes, and the perivascular spaces are dilated and contain an excessive quantity of fluid, also cellular elements. Obliterative endarteritis occurs also. Atrophy and degeneration of the cerebrum are met with, chiefly involving the cortex, particularly that of the frontal or parietal regions and the anterior basal region. The ventricles are dilated and the ependyma is granular. The membranes are thickened and opaque, and adherent to the surface of the convolutions, so that the cortex is torn upon their removal. Hemorrhage may take place into the subdural spaces, and may vary in amount from a mere stain to the formation of a pseudo-membrane.

Secondary sclerotic and descending degenerative changes may be found in the cord in some cases.

Etiology.—As in locomotor ataxia, a history of syphilitic infection is obtained in a large majority of all cases. The condition occurs more frequently in men than in women, and usually between the thirtieth and fiftieth years. Business or domestic troubles, and, in fact, great anxiety of mind from any cause, also venereal or alcoholic excesses, serve more or less directly to induce the disease. Trauma and heredity play but a minor part.

Symptoms.—The **prodromal stage** may last for months or even years. The symptoms are both mental and physical, either of which may appear first and exist alone for some time, or they may be contemporaneous in point of onset. As a rule, some *alteration of the character* and demeanor of the patient is the first evidence of the trouble. The patient suffers from insomnia and is generally restless, as well as incapable of sustained effort. He will be forgetful and perhaps careless where he was formerly careful and attentive. The sexual passions are aroused, and the patient may suffer from a sort of erotomania, sometimes taking the form of perversion. The *ego* will figure prominently in his sayings and doings. Among the physical signs are frequent twitchings and *tremors* of the facial muscles, particularly of those about the mouth and the tongue. Tremors of the hand and arm seriously interfere with writing; tremor of the lips and tongue renders the *speech* thick, blurred, and hesitating, and syllables are omitted from words, or even whole words lost from sentences; and the *pupils* are frequently unequal and fail to react to light. Frequently they exhibit the Westphal-Piltz reaction—that is, contraction upon attempt to close the eye against resistance. These symptoms extend over a variable period, with one or more remissions as a rule, and sometimes with a complete intermission and an apparent cure. Sooner or later, however, the next stage develops.

Stage of Mania or Melancholia.—The symptoms of this stage are superadded to those of the first, which by this time have grown gradually more pronounced; *loss of power* usually is already a prominent feature. Acute outbreaks of *mania* are most commonly met with, and are characterized by a most remarkable prodigality of thought and speech. The patient believes himself to be possessed of enormous wealth or of great rank and power. He is boisterous, sleepless, and constantly and actively engaged in pursuing his extravagant ideas. In other cases this delusion of grandeur (*expansive delirium*) is absent and the patient is melancholic. This is especially apt to be the case if his physical condition is lowered by some intercurrent disease. Remissions of all these symptoms are not rarely met with. Whether the prevailing type be that of mania or melancholia, *paroxysms of terror* may occur from time to time, or mania and depression may alternate. *Epileptiform* or *apoplectiform attacks* occur, followed by paralysis. In the large majority of cases the mental decay is progressive, until finally complete fatuity is reached; the patient then becomes bedridden, bladder and rectal symptoms develop, and possibly bedsores. Death results from exhaustion or from some intercurrent disease.

Diagnosis.—This is always difficult, and it is often impossible to diagnose the disease in the earliest stages, particularly when the mental phenomena alone exist. The slight change of character and the occasional outbursts of temper or unrestrained jollity may be regarded as mere moods more or less directly dependent upon the daily routine. When to these symptoms are added the tremor, the defects of speech, the inequality of the pupils, and paresis, the clinical picture gradually assumes definite shape, and ofttimes, long before expansive delirium or melancholia develops, a positive diagnosis is made. The tabetic type of the disease presents many points of resemblance to tabes dorsalis. There are ataxia, loss of knee-jerks, disturbance of micturition, fulgurant pains, visceral anesthesia, and Biernäcki's symptom (absence of tenderness over the ulnar nerve). To these are added tremor of the lips, disturbance of speech, and the peculiar mental symptoms.

Differential Diagnosis.—The diseases with which it is most likely to be confounded are—(1) *Disseminated sclerosis*, (2) *Paralysis agitans*, and (3) *Cerebral syphilis*.

(1) In *disseminated sclerosis* the mental symptoms are even less obtrusive in the earlier stages, the first evidence of the disease being paresis in the lower extremities. The tremor too is volitional, the speech is scanning, and nystagmus is present. The reflexes are exaggerated, and there may be spasticity. Mental phenomena generally develop later.

(2) In *paralysis agitans* there are frequently no mental changes, and in any case they consist of nothing more than dulness. The characteristic attitude and gait; the tremor when at rest, which sometimes ceases on movement; the speech, which is hesitating at first, then hurried; the high-pitched voice; the absence of pupillary changes,—all mark paralysis agitans. Remissions are uncommon.

(3) *Cerebral syphilis* may also simulate paretic dementia. The differential diagnosis is comparatively easy in most cases, however, and particularly if the early history can be obtained. In cerebral syphilis the tremor may or may not be present, but no speech-defect occurs; epileptiform attacks are common (usually *petit mal* or Jacksonian), and attacks of headache are frequent and usually severe. The condition often passes into dementia, and then closely resembles general paresis.

The **prognosis** is gloomy, and recovery very seldom occurs. The tendency is toward a fatal termination in from two or three to fifteen years.

Treatment.—Drugs are of no value in a curative sense, except in those cases that are due to syphilis, when the iodids must be pushed. Bromids, morphin, chloral, or, still better, sulfonal or trional, may be used in combating the insomnia and attacks of delirium. These cases cannot be properly cared for at home; indeed, their removal to an asylum is generally imperative. The tendency to bed-sores must not be forgotten, and continuous rest in bed must therefore be postponed as long as possible.

CEREBRAL PALSIES OF CHILDHOOD.

Definition.—This includes hemiplegia, the birth-palsies of Gowers (diplegia), and paraplegia, each being charaterized by a particular palsy, and in certain cases with rigidity, and all showing more or less mental defect.

1. **Hemiplegia.**—*Pathology.*—Practically nothing is known of the early changes that take place in most of these cases. They are essentially chronic, and do not reach the autopsy-table until late in the disease. A minority of cases, due to some vascular disturbance—embolism, thrombosis, or hemorrhage—may be clear enough. The others have been ascribed to some one of the following lesions: (1) Polio-encephalitis of Strümpell, who advances the view that there is an acute inflammation of the motor cortex similar to that of the motor cells of the anterior horns. (2) Congenital encephalitis of Virchow, believed by him to be an interstitial inflammation, with the formation of yellowish bodies in the white substance, due to the deposition of fatty granules. (3) Meningo-encephalitis, with opacity, thickening, and adherence of the membranes. The later changes are as follows:

(*a*) *Atrophy or Hypertrophy and Sclerosis.*—The sclerotic areas vary from small plaques to extensive regions, involving even the entire cortex of one hemisphere. They are hard and firm, and in the hypertrophic variety stand out beyond the normal tissue-line. The blood-vessel walls generally show proliferative changes, and the membranes are generally thickened and adherent, though in some cases they show very little if any change.

(*b*) *Porencephalia.*—This condition, first described by Heschl in 1859, is etiologically very obscure. Among the causes said to give rise to it are arrest of development, vascular disturbances, encephalitis, and hydrocephalus. It consists of a loss of cerebral substance, cysts of various kinds extending into the brain-substance and reaching even to the ventricles. An entire hemisphere may sometimes be wanting, and the cranium is sometimes distorted.

Etiology.—Enough has been said to indicate the uncertainty as to the cause of this condition. Many cases are congenital, and the large majority develop within the first three years of life. There may be a history of dystocia, with or without the use of forceps. Cerebral traumatism and the infectious diseases are cited as having a more or less direct etiologic relationship.

Symptoms.—In many cases the symptoms are similar to those met with in hemiplegia of adults. Without premonitory symptoms a *loss of consciousness* develops suddenly; in other cases local or general *convulsions* precede the unconscious period; while in still another group consciousness is never lost. *Fever* is always present, but does not go above 102° F. (38.8° C.). The *hemiplegia* may be of gradual onset, though usually it is found to be quite pronounced when consciousness returns. The face is generally not involved, and even when it is implicated the upper parts (eyelids and forehead) escape. The right side is affected most frequently, and the upper extremity to a greater extent than the lower. Just as we see in adults, so it is in children. The paralysis may clear up in a few days, leaving little or no trace behind, the rule being for the leg to recover first. The arm may or may not recover. Where permanent damage is done the muscles waste, though usually but little. Neither sensory nor electric changes occur. The reflexes are increased and spasticity may develop (*hemiplegia spastica cerebralis* of Heine).

These children are, as a rule, mentally deficient, varying from what may be regarded merely as stupidity to idiocy or imbecility. Speech may not be acquired until late or not at all. It is probable, too, that the cases of "idioglossia" and congenital speech-defect, first described by Hadden, belong to this category.

Various forms of post-hemiplegic movements are quite common. These may be tremors, choreiform movements, or athetosis, and about half of these children develop epilepsy. Occasionally the latter is Jacksonian in type; usually, however, it is *petit mal* or *grand mal.*

2. **Diplegia, Birth-palsies, and Paraplegia.**—These conditions are described together, since they have the same pathologic substratum. They are characterized by double hemiplegia or paraplegia, increased reflexes, often by spasticity, preservation of sensation, and a lack of mental development. While hemiplegia may or may not be congenital, these cases of congenital spastic paraplegia (or "Little's disease," as it is sometimes called after an orthopedic surgeon, one of the earliest writers upon it) always date from birth, though the condition may not be recognized until months later. Probably most cases result from meningeal hemorrhage, due to the use of forceps or other injury at birth, as pointed out by Sarah J. McNutt, or perhaps to a previous meningo-encephalitis. At all events, whatever the earlier changes be, the later ones are those of atrophy or porencephalia. Van Gehuchten has published some interesting papers on this type of infantile palsy. His conclusions are that it is met with most frequently in children born prematurely, no matter whether the labor be a difficult one or not. He calls attention to the fact that at this early stage the pyramidal tracts are non-medullated, and therefore unable to functionate normally. The pons-cerebellar pathway is the first to myelinate, the last being the fibers of the direct motor path-

way, which become medullated from above downward; hence those to the lumbar and sacral region are last of all, and may never become covered. This is in keeping with our clinical findings. The palsy may disappear from the face, then from the upper extremities, and, finally, or perhaps not at all, from the lower extremities.

Symptoms.—In some cases a history of convulsions with febrile attacks is obtained; in others nothing abnormal is observed until the child commences to walk and to try to use its arms in a definite manner. The limbs will be found more or less rigid, and in a few cases the face-, head-, and neck-muscles will be affected. The *reflexes* will be increased, though sensation is generally unchanged. The *mental condition* is almost always very poor. Various grades of spasmodic incoördination are met with; also choreiform movements and athetosis.

The **diagnosis** is not difficult if we can obtain a definite history; otherwise it may be. The absence of electric changes will serve to differentiate it from anterior polio-myelitis.

Prognosis.—The extent and degree of paralysis and the character of the mental change are important aids in forming a prognosis. Taken as a whole, the outlook is not particularly bright in any case.

Treatment.—Apart from the treatment of the early convulsions, which must be promptly met by hot mustard baths, enemata, purgatives, and the bromids, little can be done. Massage and faradization may be tried. In view of the results of operative measures these are unjustifiable, nor could we expect to gain much, if anything, owing to the nature of these cases. Careful and systematic training is of great importance, for, while some remain imbeciles, others, though very slow to learn, ultimately reward the patience of the teacher.

ACUTE DELIRIUM.

(*Acute Delirious Mania; Typho-mania; Acute Periencephalitis; Bell's Mania.*)

Definition.—An acute maniacal delirium associated with hallucinations, with a febrile course, of limited duration, and of grave prognosis.

Pathology.—Minute pericapillary hemorrhages are almost invariably present. In addition to these there are usually degenerative changes in the ganglion-cells. Often injection of the pia and minute hemorrhages into the gray matter may be observed with the naked eye. Cramer has recently reported a case in which the pericapillary spaces of the brain were filled with mononuclear leukocytes, surrounding which were recent hemorrhages; he also noted the fact that the ganglion-cells, instead of exhibiting normally-formed chromophilic bodies, were filled apparently with fine dust.

Etiology.—The disease occurs in either sex with about equal frequency. Predisposing conditions are neuropathic heredity, nervous disposition, the presence of other nervous diseases, particularly neurasthenia and epilepsy, alcoholic or sexual excesses, and severe prolonged anxiety. It has been supposed (Hertz) that abnormal narrowness of the jugular canal, which has been noted in several cases, bears some etiologic relation to it. It frequently occurs apparently as the immediate result of menstruation, parturition, injuries to the head, sunstroke, acute infectious diseases, particularly pneumonia and typhoid fever, and it may develop in the course of chronic mental diseases. Occasionally, however, it appears to arise without any definite cause.

Symptoms.—The disease usually commences with certain indefinite prodromes. These consist of *restlessness*, associated either with melancholia, preoccupation, or anxiety. The *intelligence* becomes distinctly decreased; the patient loses appetite, is constipated, and commences to emaciate. During sleep unpleasant dreams or nightmares almost invariably occur. Sometimes there is a sense of impending mental disorder. This period gradually changes to one of defiance, which perhaps, even in the prodromal stage, may lead to violence and injury to those in the neighborhood. The prodromal stage rapidly passes to *acute delirium*, in which two steps may be recognized—excitation and collapse. The excited stage commences suddenly; there is great confusion; the patient ejaculates disconnected sentences or words or even syllables. There is great anxiety, and even fear, and the patients exhibit intense excitement, suffering very often with delusions of persecution by their environment, and nearly always having hallucinations, either of sight or sound. Often their minds are occupied by some subject that had previously caused them great anxiety—either disgrace, business, or other misfortune. The mania is often dangerous; indeed, it is likely that the disease known as "running amuck" in the Malay Peninsula is simply one of the forms of acute delirium. The patient soon becomes restless, throws himself from one side of the bed to the other, and makes efforts to rise and escape from the room. The tongue is dry, the pulse rapid and weak. Petechiæ may appear upon the skin, and there is nearly always more or less fever, not rarely rising to 105° (40.5° C.) or even more. Rapid emaciation supervenes. There are all the objective symptoms of irritation of the brain—myosis and increased reflexes, and often hyperesthesia, although the patients pay little attention to any injury they may inflict upon themselves. Sometimes there seem to be curious imperative movements; at others, imperative ideas. In a case that I observed the patient rhymed, very imperfectly it is true, each two successive sentences. This stage of excitation soon passes into one of *stupor* and *collapse;* fever may become even higher, and the pulse still more rapid and weaker. The patient lies in a condition of muttering delirium, with carphologia. All the symptoms are those of profound exhaustion: the eyes are hollow, the lips and teeth covered with sordes, and the emaciation extreme. The skin becomes dryer, and finally cyanotic, the pupils dilate, and there may be marked anesthesia. Death ordinarily occurs at the end of two or three days after the commencement of this condition. Occasionally the course of the disease is interrupted by intervals in which the patients exhibit more or less lucidity. Certain varieties have also been described. Thus in addition to the maniacal form authors speak of the *melancholic* and *paralytic* forms. In the former of these the patients exhibit, in place of excitement, profound depression, with fear of poisoning and positive refusal of all food; slight elevation of temperature, or, indeed, a subnormal temperature, and very rapid emaciation. It is most apt to occur in patients previously debilitated. In the paralytic form there is vasomotor paralysis with cyanosis, depression, and often stupor. From these the patient passes into an algid state, in which death occurs.

The **differential diagnosis** is frequently difficult. In many infectious diseases, particularly *pneumonia* and *typhoid*, hallucinatory delirium may develop. This, of course, must be suspected in these diseases, and it is advisable, if possible, to examine the blood in all cases of acute delirium by Widal's method. In *acute mania* fever is rare, emaciation is not so rapid, and the mental symptoms are more purely psychical. In *general paralysis*, toward the end maniacal attacks may develop, but the history of the previous existence of the disease, the presence of the Argyll-Robinson pupil, and the absence of fever lead one to suspect the true diagnosis. Finally, in *delirium tremens* the fine tremor of the hands and tongue, and, if possible to obtain it, a history of recent debauch, should clear up the diagnosis. The course of the disease is variable; it may vary from three or four days to as many weeks. Those cases are most rapid in which excitation is most profound.

The **prognosis** is most unfavorable, and is more so for men (according to Krafft-Ebing) than for women. Those cases that were previously debilitated, either as a result of chronic alcoholism, or chronic exhaustive diseases, or childbirth, are the most serious. Those that develop suddenly, and from the beginning are very severe, are also nearly always fatal; if there are no lucid intervals, or if those that occur are short and imperfect, the prognosis is graver; and the same is true of those who suffer from obstinate insomnia.

The **treatment** is of course unsatisfactory. In spite of progressive exhaustion, bloodletting is recommended, and calomel should also be administered in the earlier stages of the disease. At the same time the temperature should be combated by cool baths, and an ice-bag should be applied to the head. Sleep should be obtained by the use of chloral, bromids, and the more modern hypnotics, which are to be preferred to morphin. Hyoscin seems to be particularly indicated. In the later stages of the disease stimulants should be administered freely. Excellent results have been obtained (Solivetti) by the hypodermic administration of Bonjean's ergotin. Nutrition must be maintained by forced feeding with milk, eggs, broths, etc.

IV. DISEASES OF UNKNOWN PATHOLOGY.

EPILEPSY.

Definition.—A condition characterized by attacks of unconsciousness, with or without convulsions. We are scarcely justified in speaking of epilepsy as a disease. It seems, in reality, to be a symptom, though in many cases (the so-called idiopathic cases) we do not know the underlying cause. The type of cases in which the unconscious period is very brief (momentary), with no convulsion following or at most but a slight rigidity, is termed *petit mal.* The more pronounced type, with prolonged unconsciousness and severe general convulsions, constitutes *grand mal.* That form first described by Hughlings Jackson in which the convulsion is localized, and in which unconsciousness may or may not occur, is called *Jacksonian*, *focal*, or *cortical epilepsy.*

Pathology.—Gray regards epilepsy as a symptom, and if this theory be correct, the inevitable question must be, "Of what?" In certain cases this can be answered (in the organic cases), since the lesion is demonstrable; but in others (functional or idiopathic) there is no demonstrable lesion. Among the causes of the former are brain-tumors, meningitis, traumatism inflicted either at birth or subsequently, atrophy and sclerosis, vascular disturbances, syphilis, and toxemia, both autogenous and exogenous. Brown-Séquard pointed out in 1857 that epilepsy may sometimes have its cause in the spinal cord. Peripheral lesions too may give rise to it. Little can be said about the idiopathic variety. Chaslin has endeavored to show that in this form a constant lesion exists—viz. a diffuse sclerosis of the gray matter, a neurogliar overgrowth—but his views have not been corroborated. After all, we can only enumerate causes; we do not know in any case how these act, and we do not know the ultimate pathology. Many writers apply the name "epilepsy" only to the idiopathic form, while others include all apyretic affections characterized by the occurrence of fits, whether of centric or peripheral origin. Brown-Séquard believes that the distinction between the various kinds of convulsions is artificial, and that the correct classification should be based on the knowledge of the cause.

Etiology.—The causes are (1) *predisposing*, (2) *exciting* or *determining*. Among the former, which refer particularly to the idiopathic form, are—

(*a*) *Age.*—The following tables show the early onset in a large majority of cases analyzed by Gowers, Hesse, and Osler:

Age at onset.	Proportion affected. Observers.	
	Gowers.	Hesse.
Before 10	422	393
From 11 to 20	665	364
" 21 " 30	224	111
" 31 " 40	87	59
" 41 " 50	31	51
" 51 " 60	16	13
" 61 " 70	4	4
71	1	0
Total,	1450	995

Age at onset (Osler).	Number.	Age at onset (Osler).	Number.
1	74	9	17
2	62	10	27
3	51	11	17
4	24	12	18
5	17	13	15
6	18	14	21
7	19	15	34
8	23	Total,	437

(*b*) *Sex.*—In Gowers' cases 54.6 per cent. were males, 45.4 per cent. females. Under twenty-five years of age males are slightly in the majority; above twenty-five, the reverse is true.

(*c*) *Heredity.*—Family neuroses are common, but it is decidedly more the exception than the rule to find either parent epileptic. Féré has given us the lineage of 594 epileptics: 70 had epilepsy, 166 were insane, 88 paralyzed, 21 suffered from general paralysis, 72 from hysteria, 73 from senile dementia, 33 from puerperal eclampsia, 61 from chorea. Among the direct antecedents of these 594 epileptics, 1024 cases of nervous disorder had occurred.

(*d*) *Alcohol.*—The causal relationship between an abuse of alcohol by the parents and epilepsy seems rather pronounced. Féré says that of 594 epileptics examined by him, 258 had parents who were hard drinkers. Echeverria refers to 572, 257 of which he believed could be traced directly to the abuse of alcohol.

(*e*) *Syphilis* does not predispose. When it gives rise to changes in the brain and cord, which in turn cause epilepsy, it is in reality a determining cause.

(*f*) *Eye-strain* is no longer regarded seriously as a predisposing influence.

The **exciting or determining causes** are traumatisms, various morbid conditions of the membranes of the brain or of the brain proper (*e. g.* after hemiplegia), of the spinal cord or membranes, or peripheral irritation (dentition, worms, a cicatrix, an adherent prepuce, etc.). Not a few cases are dependent upon toxic substances in the blood, as in uremia and lead-poisoning. Excessive indulgence in alcohol or over-eating often precipitates the attack. Great emotion, nervous shock (fright), and masturbation are also said to be able to bring it about, though these are rather doubtful causes. There are cases of bradycardia in which epileptiform attacks occur.

Symptoms.—**Petit Mal.**—In this condition the majority of cases belong to the following type: The attack begins suddenly; perhaps while talking to the patient his expression suddenly becomes blank, the face pales, the pupils dilate, and he is evidently not conscious. In a moment or two he gathers his scattered senses, picks up the thread of the conversation, and continues to unwind it. Very often he is not cognizant of any lapse of time or has but a vague idea. If carefully watched for, fine clonic movements may be observed in many cases, it may be of the facial muscles or of the hands. Convulsions never occur, the dominant feature being the unconsciousness. On regaining consciousness the patient may act strangely and appear dazed; it is seldom, however, that he falls in attacks of this kind. Occasionally a peculiar dreamy state takes the place of an ordinary attack, or the individual may be the victim of imperative ideas. Falret has described a condition (*epilepsie larvé*) known as masked epilepsy, in which maniacal outbursts or explosions of passion occur.

Grand Mal or Haut Mal.—In many cases some subjective symptom precedes the actual attack. In its most specialized form it is termed an *aura*, and includes any phenomenon, motor and sensory, that ushers in an attack. While the aura differs in different cases, it is almost invariably constant in the same case, so that one will have a subjective sensation of sound, another of light, either flashes or colors, etc. There are other signs that occasionally antedate an attack, and which may or may not precede each attack (headache, drowsiness, change of disposition, palpitation, perverted appetite, sexual or other, etc.). Many attacks begin precipitately with absolutely no previous warning. In such cases the patient may or may not utter a piercing sound (*epileptic cry*), falling at the same time, no matter where or in what position he may be. Hence the danger to which epileptics are always subjected. A peculiar onset occurs in the so-called "*procursive epilepsy*," in which the patient suddenly starts off and runs some distance before the paroxysm begins.

Paroxysmal Period.—In many cases, whether preceded by an aura or not, this stage is ushered in by a *spasm* that is tonic in character. The patient falls, perhaps because of the loss of consciousness, though in those cases in which he drops precipitately he is probably thrown by the violence of the spasm. The head is usually extended, the muscles of the larynx and trunk contracted, and hence the epileptic cry and the dyspnea, while the lower limbs are generally extended, the upper semiflexed, and the fingers tightly clenched. This period of rigidity lasts but a few seconds before *clonic convulsions* appear.

Intercurrent contractions vary in different cases from very mild movements to those so severe as to toss the individual about. The face, pale at first, becomes congested, and the jaw works in churning the saliva into a froth; this is blood-tinged when the tongue is bitten. The respiration is jerky, gasping, and there may be a loss of control of the bladder and bowels. In idiopathic cases this stage lasts from one to five or six minutes. The spasms gradually diminish, and without regaining consciousness the patient passes into a deep sleep, immediately preceded in some cases, however, by coma in which the breathing is stertorous. During the sleep, which lasts about an hour, the patient is completely relaxed. On waking he usually appears confused and complains of feeling tired. His limbs may ache for several days.

Occasionally attacks follow one another in quick succession, with no period of consciousness intervening (*status epilepticus*)—a very dangerous condition.

Post-epileptic phenomena are variable. The patient may become maniacal, homicidal, or may simply be mentally deficient for a few days, with perhaps some slight speech-disturbance. In the course of time every epileptic's brain-power deteriorates. Paralysis sometimes occurs, is usually transient, and may be unilateral or bilateral.

Nocturnal Epilepsy.—In this condition the attacks occur at night, and may be entirely unknown either to the patient or his friends. He complains from time to time of feeling tired on rising in the morning, his limbs and head ache, and he is generally duller than usual; he may even be confused. Such a history is suggestive, and the suspicion is strengthened if in addition he has urinated involuntarily or if blood-spots are found on his pillow.

Jacksonian epilepsy is characterized by spasm that is generally local in character; in fact, it is always so in the beginning, though occasionally it may spread and become general. Consciousness is preserved in the milder forms. Tingling or other subjective sensations may precede an attack. They are usually due to some irritation of the motor cortex (tumors, meningitis, softening, trauma, etc.). Subcortical lesions and certain toxemic conditions can also give rise to it.

Diagnosis.—When a definite history is obtainable the difficulty of the diagnosis is less, particularly if an aura occurs. The attack can be frequently diagnosed from other epileptoid conditions at the time by the explosive onset, the brief tonic and somewhat longer clonic spasm, profound unconsciousness followed by a deep sleep, and when these are present by an involuntary passage of urine, frothing at the mouth, and biting of the tongue.

Differential Diagnosis.—In *uremia* the state of the urine (catheterize if necessary), and often the odor, serve to differentiate it. It may be impossible to detect fraud, so perfectly is the disease simulated by those anxious to excite pity, judicial or otherwise, or by those whose accomplices rifle the pockets of sympathetic bystanders. Hysteria may also resemble it very closely. Gowers has tabulated the chief differences as follows:

	EPILEPSY.	HYSTEROID.
Apparent cause . . .	None.	Emotion.
Warning	Any, but especially unilateral or epigastric aura.	Palpitation, malaise, choking, bilateral foot-aura.
Onset	Always sudden.	Often gradual.
Scream	At onset.	During course.
Convulsion	Rigidity followed by "jerking;" rarely rigidity alone.	Rigidity or "struggling," throwing about of limbs or head, arching of back.
Biting	Tongue.	Lips, hands, or other people or things. Very rare.
Micturition	Frequent.	Never.
Defecation	Occasional.	Never.
Talking	Never.	Frequent.
Duration	A few minutes.	More than ten minutes, often much longer.
Restraint necessary . .	To prevent accident.	To control violence.
Termination	Spontaneous.	Spontaneous or induced (water, etc.).

Prognosis.—Inherited epilepsy very rarely is cured. Brown-Séquard says most emphatically that idiopathic cases are occasionally cured: I would rather suggest that perhaps they get well. Cases that are evidently symptomatic are curable if the cause can be removed. Death is seldom due directly to an attack. Fatal accidents may, however, be caused by an attack.

Treatment.—When an aura occurs, advantage may indirectly be taken of it to aid in aborting the attack—viz. by employing the galvanic method, or by constricting the limb in which the aura occurs, or by forcibly moving the head, putting snuff up the nose, ice to the spine, etc., according to the special indication. Every effort should be made to lessen the liability of danger to the patient—first from falling, and secondly, from the violence of the spasms. One may at times be justified in using ether or chloroform by inhalation to control the severity of the convulsions. After loosening the clothing and putting a cork or something between the teeth to prevent biting the tongue, nothing more can be done at the time, although in a few cases amyl nitrite has aborted or lessened the attacks. Between the attacks special care should be taken to put the system in good condition, and all sources of worry and irritation should be removed as far as possible. Particular attention should be given to the stomach and bowels. The food should be light and easily digestible.

As to *medicinal measures*, the bromids are of the greatest value. The sodium and potassium salts are most commonly employed, the former, as a rule, being better borne by the stomach. They may be given in milk or in one of the medicated waters. Strontium bromid has been used rather extensively of late, and has yielded excellent results. While idiosyncrasies are met with, it may generally be given in from 15- to 30-gr. doses (0.972–1.944) three or four times a day, and preferably after meals. Each case must be treated according to its special indications. Symptoms of bromism (acne, sore throat, drowsiness, and gastric disturbance) should be carefully guarded against. Should they develop, the dose of bromid must be reduced, and Fowler's solution administered for a few days. H. C. Wood recommends that the latter should be given continuously with the bromids, thereby preventing, or at all events lessening, the liability to bromism. Other remedies sometimes employed are nitroglycerin (hypodermically), cannabis indica, silver nitrate, zinc, borax, chloral, antipyrin, trional, and tetronal. Surgical measures occasionally yield good results, this being particularly true in focal epilepsy. In idiopathic epilepsy removal of the motor cortex has been tried in those cases in which an aura suggested a local origin—*e. g.* in a center for a particular group of muscles. The results have naturally been less encouraging than in Jacksonian epilepsy. It is a curious fact that almost any surgical operation will diminish or check the epileptic attacks for a time, and I have known as simple a procedure as venesection to afford complete relief in a severe case for several months. The results ascribed to various operations may be explained in large part by this.

MIGRAINE.

(*Hemicrania; Sick Headache.*)

Definition.—A neurosis characterized by severe attacks of headache, often paroxysmal and more or less periodic, with disturbances of vision and with or without nausea and vomiting.

Pathology.—This is profoundly obscure, since no lesion has ever been discovered. By some it is thought to be a vaso-motor disturbance. Arterio-sclerosis has been present in many cases, but this is also frequently met with where no sign of migraine has ever occurred. Very rarely the disease has been observed in some subjects to replace an attack of epilepsy or even to alternate with true epileptic attacks.

Etiology.—The condition is frequently hereditary, and in the large majority of the cases that I have seen it has been transmitted by or through the mother. Various other neuroses are common in families subject to this condition. Females are more frequently affected than males, and migraine seems to be associated with diseases peculiar to women, especially menstrual disorders. Among the exciting causes may be mentioned gastric disturbances, dental irritation, naso-pharyngeal diseases (adenoids, etc.), eye-strain, grief, emotion—in short, anything that tends to lower the physical or mental tone occurring in those hereditarily predisposed. Recently attention has been called to auto-intoxication (leukomainic poisoning) as a cause of certain cases.

Symptoms.—As a rule, the patient can prognosticate an attack.

In the cases of slow onset he may feel indisposed for some hours before, being languid, with general discomfort and perhaps nausea. In other cases various *subjective sensations* occur, lasting from a few minutes to several hours. Of these, disturbances of vision are most common, such as flashes of light, spectra, visions of animals or weird forms, or scotoma, etc. Auditory sensations are rare, as are those of the other special senses. After these phenomena have existed for some time *headache* supervenes, when, as a rule, they cease. The pain, at least in the beginning, is usually unilateral, as the name suggests, though later it may and often does involve the entire cranium, spreading from a single point of origin—over one eye, for instance. The affected region may be tender to the touch or it may be the seat of numbness or tingling. *Nausea* and *vomiting* commonly occur, with or without vertigo. A brief period of unconsciousness occurs in some cases, and spasmodic movements may also be observed occasionally. This fact is of particular interest, since it serves to support the view that migraine is in some way related to epilepsy, and, as has been stated, attacks of migraine and epilepsy may alternate. Unlike epilepsy, migraine does not tend to impair the mental faculties, no matter how long the patient has been afflicted. During an attack, however, he may have melancholia or be incapacitated mentally and physically for two or three days.

Course.—The disease generally begins in early life, and in nearly half of the cases before the fifteenth year, recurring with a certain degree of periodicity until old age, when it often passes away. It may cease in women at the menopause, and in men between the fortieth and fiftieth years.

The **prognosis** is good as far as life is concerned. This disease is incurable, though the condition of the patient may be alleviated.

Treatment.—The management of the disease may be considered under two heads: (1) treatment of the attack, and (2) the treatment between the attacks, which necessarily includes prevention. The patient should be put to bed in a slightly darkened room, and all sources of noise and confusion should be removed as far as possible. The attack may be so severe as to justify the use of morphin hypodermically. The coal-tar derivatives have met with most favor, however, as remedial measures, and preferably antipyrin and phenacetin, though their occasional depressing effect should be borne in mind. The following has given excellent results in my hands:

℞. Caffein. citrat., ʒss (2.0);
Phenacetin,
Sodii bicarb., *āā.* ʒj (4.0);
Pulv. aromat., gr. xij (0.777).
M. et ft. chartæ No. xij.
Sig. One every three hours.

Acetanilid may often be substituted for phenacetin with apparent advantage. In twenty-four hours this may be discontinued, and potassium bromid should be given in liq. ammon. acetatis in doses of gr. x to xv (0.648–0.972). Local applications of menthol, or fly-blisters may be employed, or even superficial points may be made with the actual cautery. Electricity is of little avail. The electric *percuteur* or hammer, however, has given good results. It consists of a hammer driven at great speed by electricity, thus enabling short sharp blows to be directed to the part.

Between the attacks the general health should be carefully looked after. The uric-acid diathesis is common in subjects of migraine. Haig staunchly advocates the use of salicylates in such cases in addition to the dietetic measures. Anemia should be treated by iron in some form, dialyzed or Blaud's pill, or, with those who prefer the organic preparations, ferratin or peptomangan may find favor. The bowels should be kept soluble by means of some saline (Hunyadi, etc.), or by the fluid extract of cascara sagrada. The extract of cannabis indica is employed by some over a long period of time, just as the bromids are in epilepsy. It is given in doses of gr. ¼ to ½ (0.0162–0.0324) two or three times a day, after meals. While exercise and fresh air are admirable adjuvants to any form of treatment, it must not be forgotten that fatigue invites an attack. Proper rest, care and regularity in dieting, and the avoidance of excitement are the chief points to be observed.

ACUTE CHOREA.

(*Sydenham's Chorea; St. Vitus's or St. Anthony's Dance.*)

Definition.—The type of chorea described by Sydenham is a more or less acute disease, in certain respects resembling an acute infectious process, and by some regarded as such. It has a special predilection for children, and is characterized by involuntary muscular contractions, by usually slight and rarely-marked mental change, by a great liability to endocarditis and a tendency to recurrence, particularly during the spring and fall.

Pathology.—No definite lesion can be ascribed to the disease, though, as is usual in such cases, a great variety of lesions have been described. Chief among these is the embolic theory, advanced by Kirkes and subscribed to by Bastian and Hughlings Jackson. Since emboli are not found in every case, however, they cannot be the sole cause. The infectious theory has also been mentioned above, and certain cases unquestionably seem to be due to micro-organismal activity. For instance, the same organism has been found in the brain, heart, and the blood, or in the vegetations on the valves. Many cases develop suddenly, following fright or some other nervous shock, though these can scarcely be included in either of the two classes just mentioned. Acute chorea may also be due to functional instability of the nerve-centers, or it may possibly result from the irritation of antitoxins.

Etiology.—(1) *Age.*—By far the greatest number of cases occur before the twentieth year; in fact, the condition is rare after that age, most cases occurring between the tenth and fifteenth years.

Sex.—Females are most frequently attacked, and probably in two-thirds of all cases.

Race.—Acute chorea is rarely met with except among the white races. Infectious diseases do not seem to have any predisposing influence.

The relationship, however, between chorea and rheumatism is difficult to ascertain. The milder form of this latter trouble can be readily overlooked in young children, but if we include, as Sturges does, all cases with a history of "growing pains," the relation is of course greater. English statistics show a higher percentage with a rheumatic history than those of any other country, probably because rheumatism is more common in that country than elsewhere. In the United States unmistakable rheumatic attacks occur in but a small proportion of choreic patients either before or during the actual attack of chorea. The latter disease may be, as I have repeatedly observed, followed by rheumatism later in life.

Endocarditis.—As has already been stated, some observers regard chorea as the result of a cerebral embolus due to fragments of fibrin being washed from the valves and carried into the circulation to the cerebral vessels. Endocarditis occurs in the vast majority of cases of chorea, but as an effect, not a cause.

Pregnancy.—Chorea occurring during pregnancy is apt to be severe. It is most prone to develop during the earlier months, and especially in primiparæ. It often assumes the maniacal type.

Heredity.—In about 10 per cent. of the cases a history of chorea can be obtained in other members of the family. Children of neurotic stock are more susceptible than those of a normal type, and in such individuals worry, fright, or mental shock from any cause, also the strain of puberty or of education, are very liable to induce an attack. I have several times seen chorea follow attacks of religious fervor in young girls.

The influence of *reflex irritation* is probably much overrated, whether intestinal, genital, or from ocular defects.

Symptoms.—The common or ordinary form is frequently met with, particularly at seasonal changes (H. C. Wood), and especially during the spring and fall. Primary attacks may occur at any time, but recurrent outbreaks are most prone to develop in the spring. There is a slight affection manifested by restlessness, disturbed rest at night, and by irregular and spasmodic *muscular movements*, that are most marked in, or entirely confined to, the upper extremities, the head, or the facial muscles; or the condition may be unilateral. More or less *muscular weakness* is present and the patient tires very readily. The child's *disposition* is changed, outbursts of temper being quite common on the slightest provocation. *Fever* is absent in this form unless complications exist, though when severe joint-troubles or endocarditis is present the temperature will rise. *Anemia* is often present, and with it headaches, irritable heart, and hemic murmurs, very rarely giving rise to symptoms. Indeed, in some instances not even a murmur can be heard, though *postmortem* records of cases with a history of chorea show that in most of them the valves are affected. The mitral valve is most commonly involved, the endocarditis usually being of the verrucose type. There are rarely any sensory symptoms, severe pain, at all events, being extremely rare. Little dependence can be placed on complaints of tingling or burning pain, unless it is voluntarily spoken of, for the mental make-up of choreic patients is such that they are apt to dwell upon slight ailments suggested to them through leading questions. The reflexes do not differ from those of normal children.

There is a more severe type of chorea in which the movements are so pronounced as to incapacitate the patient. The *psychic symptoms* are generally more marked also, and speech may be interfered with to such an extent as to render articulation unintelligible. Such a condition may occur primarily, but it is probably met with more often after one or more mild attacks. The gravest form of chorea is *chorea insaniens*, in which the movements are violent and constant. Speech is much affected, insomnia is marked, and fever and maniacal delirium develop, followed in some cases by a typhoid condition and death.

The **course** is from six to twelve weeks, though the most trifling cases may recover in a month or less. Others persist six months or even more. In about two-fifths of all cases there is more than one attack, while Gowers has reported one case in which there were nine recurrences. Dr. W. Egbert Robertson observed the case of a girl nine years of age in whom the first attack occurred at the age of six. She had four attacks in three years, each one being more pronounced than the former. In one of my own cases two attacks occurred annually—spring and autumn—for two years. A fatal issue is very rare in children, and absolute recovery is the rule. The maniacal form, which usually develops in adults, especially in pregnant women, as previously stated, is more often fatal, though recovery is also the rule in such cases.

Diagnosis.—As a rule, this is quite simple. The age of the patient, the mode of onset, and the character of the movements suffice to prevent mistakes, though the condition may be simulated more or less closely by *hereditary ataxia*. Here, however, the existence of other cases in the family, the nystagmus, the peculiar scanning speech, the swaying, almost reeling gait, and the tendency to contractures will usually be sufficient to differentiate. Yet all these symptoms may be slight, and the case may be under observation some time before its real nature is ascertained. The same is true of *cerebral sclerosis*. It is only by a careful study of such cases in their entirety that a differential diagnosis can be made. The movements may be practically the same in both conditions.

It may be well to call attention to the possibility of meeting with a case that has been treated by arsenic to the point of saturation, when the true nature of the trouble will be masked by the arsenic-poisoning and signs of peripheral neuritis. Such a case was presented by Stengel at the Philadelphia Pediatric Society not long ago.

Treatment.—This is largely hygienic—a fact that must be strongly dwelt upon in discussing with the parent the management of the patient. The avoidance of all forms of nerve-strain is of the utmost importance, and the amount of school-work and home-study should be carefully inquired into, and all excess absolutely prohibited. In the milder forms rest in bed is not imperative, but active exercise must be forbidden, since it invites cardiac troubles, the tendency to heart-involvement already being great in chorea. In the more severe forms rest in bed is a *sine quâ non*. In any case an abundance of rest is called for, and when insomnia is present it should be promptly handled. An important element of the treatment that is sometimes indispensable is the change of environment, associated with rest. As a rule, the patients do best in a warm climate and at the seashore. The bromids should first be tried internally, and a warm bath administered just before bedtime. In other cases morphin or chloral may be required, though it must not be forgotten that the latter is a cardiac depressant; chloralamid or sulfonal should therefore be given the preference. Trional has also given good results in my hands. The bowels must be regulated, and the diet should be light and wholesome, with an abundance of fruit and fresh vegetables. When anemia is present, it is to be met by the use of some one of the preparations of iron. Should reflex irritation be found to exist, it should be corrected at once. Of the therapeutics of the disease not much can be said. We have no specific, but the two most useful drugs are arsenic and cimicifuga, the former as Fowler's solution, and the latter as the fluid extract. Fowler's solution should be given in 3- to 5-drop doses three times a day for a few days, and then increased 1 drop *per diem* until the point of tolerance is reached or the physiologic action is manifest. The remedy is now to be continued, but in reduced dosage. The late Hiram Corson first warmly recommended cimicifuga in the treatment of chorea, and I have found the combined use of this drug and arsenic to be more prompt and efficient than the latter alone, particularly in protracted cases. Other drugs have been employed, but with meager results; among these are the zinc and silver salts and belladonna. Antipyrin has also been used a great deal of late, but with no more promise than is given by the drugs just enumerated.

HUNTINGDON'S CHOREA.

(*Chronic Progressive Chorea.*)

Definition.—An hereditary disease affecting many members of a family, developing in early adult life, and characterized by irregular muscular contractions, incoördination, and progressive dementia. The disease was first definitely described by Huntingdon in 1872, but other writers had already alluded to it.

Pathology.—The changes found consist of chronic pachy- and leptomeningitis, chronic hemorrhagic encephalitis, characterized by round-cell infiltration of the cortex, degeneration of the ganglion-cells, proliferation of the neuroglia, sclerosis of the blood-vessels with dilatation of the perivascular and lymph-spaces, and numerous hemorrhagic foci (Facklam). There are also atrophy of the brain, slight irregular changes in the spinal cord, and multiplication of the nuclei in the muscles.

Etiology.—The disease is strictly hereditary, and has been traced through five generations. The offspring of parents that escape are forever immune. It occasionally alternates with idiocy, epilepsy, and various degenerative conditions. It appears to be endemic in certain localities, and still exists upon the southern shore of Long Island, where Huntingdon first observed it.

Symptomatolgy.—The *choreiform movements* commence insidiously and only in the rarest cases become pronounced. They are usually susceptible to voluntary inhibition and disappear during sleep. They usually commence in one extremity and then gradually invade the other parts of the body. There is considerable *incoördination* of movement. The *mental symptoms* consist of progressive dementia, irritability often attaining maniacal violence, delusions of persecution, and rapid diminution of intelligence. In some cases the mental changes are very slight.

The **course** is steadily progressive, but the patient may live to an advanced age.

Diagnosis.—The only condition likely to cause confusion is senile chorea with dementia. In this the mental symptoms are usually slight and the motor symptoms more violent. The family character of the disease is also lacking.

Treatment is entirely symptomatic.

RHYTHMIC CHOREA.

(*Hysteric Chorea.*)

In this condition rhythmic choreic movements occur, and affect any of the special groups of muscles or the single muscles. It may be confined to the abdominal muscles (*salaam convulsions*), or, as in a case under my care at present, it may involve only the sterno-cleido-mastoid muscle.

The **diagnosis** is readily assured by the rhythmic character of the movements occurring in an hysteric temperament.

CHOREIFORM DISORDERS.

PARAMYOCLONUS MULTIPLEX.

Definition.—This is a disease of unknown pathology, first described by Friedreich, and, as its name implies, characterized by clonic contractions in various groups of muscles.

Its **etiology** is obscure. Heredity unquestionably plays an important part, nearly all the patients having among their ancestry cases of one or more forms of nervous disease. It usually develops in early adult life, and is probably more common in males. In one case that I observed it was associated with idiocy.

The **symptoms** of the disease consist of *clonic contractions*, occurring chiefly in the muscles of the extremities and the trunk and only occasionally involving the muscles of the face. These contractions are very sudden; so much so that they have been described as lightning-like. Voluntary movement diminishes them somewhat, emotional disturbance increases them considerably, while during sleep they disappear. The power of the muscles, their size and nutrition, remain unimpaired. The *electric reactions* are normal, but electric stimuli and any cutaneous irritation are apt to precipitate an attack. The *tendon-reflexes* are increased. Sometimes the patient gives vent to a peculiar *grunt*, which is probably due to involvement of the larynx and diaphragm. In some of the cases sensitive points have been found over the spinal column, and not a few have presented other stigmata of hysteria.

The **differential diagnosis** is rather difficult, as it is necessary to distinguish the disease from *chorea electrica* and the *maladie des tics* of De la Tourette; between it and the former there is really no essential difference. *Chorea electrica* occurs in children, and the spasms are perhaps less affected by voluntary movement and more apt to be rhythmic. In the *maladie des tics* the movements are more coördinated, and usually are a repetition of some reflex or voluntary action. Moreover, the patient is apt to emit more or less intelligible sounds. If there is any real difference from clonic convulsions occurring in hysteria, it is only in the presence of hysteric stigmata in the latter and the increase in the muscular contractions under observation. The disease usually begins insidiously.

In the non-hysteric form the **prognosis** is serious, very few of the cases ever showing permanent improvement.

The **treatment** consists of rest, hypodermic injections of morphin, and the application of electricity. The latter seems most effective when applied to the spinal column, a constant galvanic stream being employed and the anode being placed over the sensitive vertebræ. It is not unlikely, however, that these cases are of an hysteric nature.

CHOREA ELECTRICA.

Chorea electrica is the name applied to at least two, and probably three, varieties of spasmodic disease. The **first**, chronologically, is that of Bergeron. The disease occurs most frequently in children between the ages of seven and fourteen years.

Its *causes* seem to be heredity, anemia, and fright. In some cases dilatation of the stomach has been observed, and it is believed that it bears somewhat the same etiologic relation to this disease that it does to tetany.

Symptoms.—It usually commences rather suddenly, and is characterized by lightning-like *contractions* in the muscles of one extremity, although occasionally it may affect the whole body. These are somewhat rhythmic and painless, and do not affect the nutrition of the muscles. They are slightly increased if the patient attempts to inhibit them, but disappear entirely during sleep. Occasionally the respiratory muscles are involved, causing peculiar sounds. Ordinarily the mind of the patient is unaffected; in some cases, however, there is slight melancholia or anxiety during the attack.

The *diagnosis* consists in differentiating the condition from paramyoclonus multiplex and the maladie des tics.

The *prognosis* is extremely favorable.

The *treatment* consists in the administration of arsenic and the correction of the anemia and the gastro-intestinal disorders if any exist.

A **second** form, very similar to, if not identical with, this has been described by Henoch. It occurs at the same age, and is characterized by the same symptoms, with the difference that the contractions are usually localized in the muscles of the back and shoulder, and are almost identical with those produced by a moderate induced current. Usually one side of the body is more affected than the other.

The *etiology* appears to be that of chorea, and cases have occurred after acute articular rheumatism and fright.

The *prognosis* is somewhat more severe than in the form described by Bergeron.

Treatment.—Arsenic is without effect, but occasionally the bromids are useful. Henoch himself, however, prefers a prolonged treatment with the galvanic current, which he has seen produce cure.

A **third** disease, closely allied by its symptoms with the two preceding, but probably of very different etiology, is the *chorea electrica of Dubini*, a disease endemic in Northern Italy. It occurs at all ages, affects both sexes, and appears to be of an infectious nature. Occasionally congestion of the meninges has been found; in other cases there are inflammatory lesions in other parts of the body, and particularly in the lungs.

Symptoms.—The disease commences with severe pains in the head, the neck, and the lumbar region. After a brief interval contractions occur in the muscles, usually appearing first in the upper extremities, but rapidly becoming general. They are almost continuous, and are separated by approximately equal intervals, so that they are distinctly rhythmic in character. From time to time there are attacks of general convulsions, that may occur as often as four times per day, and are usually followed by paresis of the limbs. There is slight hyperesthesia of the skin, and usually more or less fever.

The *prognosis* is extremely unfavorable, death occurring in 90 per cent. of the cases. The duration of the disease varies from two or three days to four or five months, death usually occurring from heart-failure while the patient is comatose.

No satisfactory *treatment* has been suggested.

FIBRILLARY CHOREA (*Morvan*).

This is an affection characterized by fibrillary contractions, appearing first in the muscles of the buttocks and in the posterior portion of the thigh; it may subsequently extend to the muscles of the body, and even to those of the upper extremities, but never affects the muscles of the neck and the face. It occurs between the ages of sixteen and twenty-two years, and affects chiefly males. Occasionally it follows excessive fatigue. According to Morvan, the lesion is situated in the anterior cornua, commencing usually in that governing the sciatic nerve, and extending upward for variable distances.

The *symptoms* consist of irregular contractions of bands of fibers in the muscle, giving rise to slight irregular tremors and disappearing upon voluntary movement. Occasionally there is a slightly increased secretion of sweat in the affected members, and the skin may become somewhat pinker than normal.

The *diagnosis* is easy, though the disease may occasionally be confused with the lightest forms of paramyoclonus multiplex.

The *prognosis* is favorable, though relapses sometimes occur.

The *treatment* consists in the administration of tonics.

ATHETOSIS.

Chorea affecting one side of the body may precede or follow an attack of hemiplegia. In the *former* case the choreic movements usually commence two or three days before the apoplectic attack ; they affect the side that is subsequently to be paralyzed, may be more or less severe, and are usually most pronounced in the arm. As soon as paralysis has occurred they disappear. *Post-hemiplegic chorea*, on the other hand, comes on some days or weeks after the attack, and is usually permanent.

Athetosis, a condition first described and named by Hammond, is closely allied to post-hemiplegic chorea. It may be either partial, affecting one side of the body and following a distinct focal lesion of the brain in some part of the motor tract, or else general, in which case it is called idiopathic, and is often found in cases in which a subsequent post-mortem examination fails to reveal any lesion.

Pathology.—The local lesions causing athetosis are particularly those of the *optic thalamus*. It is also the commonest sequel of cerebral infantile palsy. These cases usually follow a circumscribed encephalitis, but may also be due to thrombosis or embolism. In any event, the subsequent condition of the brain is usually that of porencephalia, and it may also follow congenital porencephalic conditions.

The *etiology* of the idiopathic or general form is not so well known. It sometimes develops in children born after prolonged and difficult labor, particularly when instruments have been used. It may be associated with microcephalia, idiocy, or paralyses.

The *symptoms* of the disease consist of peculiar *worm-like movements*, most marked in the fingers and toes, and then in the muscles of the arm and leg, but often also involving the face. The movements of the fingers are very characteristic, and seem to be produced by the combined action of the interosseous muscles and the extensors of the fingers; as a result, the latter are bent at the metacarpo-phalangeal joints, whilst all the phalangeal joints are extended, in old cases, as a result of constant pulling, usually somewhat over-extended. At the same time the fingers are in constant irregular rhythmic movement. The arm is swung to and fro, bent and straightened at the elbow, pronated and rotated. The movements of the toes are analogous to those of the fingers. In the muscles of the face contractions and relaxations occur, giving rise to peculiar grimaces and somewhat interfering with speech. In those cases in which athetosis develops after a lesion of the optic thalamus there is often associated hemianesthesia; in those following cerebral infantile palsy, sensibility is intact. The muscles show no trophic disturbances, and, indeed, may be slightly hypertrophied as a result of constant exercise. The idiopathic form is usually bilateral; the *gait* is curious and characteristic, the patients appearing to be constantly on the point of falling, although they seldom do fall, and maintain their equilibrium almost by a miracle; whilst the arms swing violently and the face exhibits a series of grimaces.

The *differential diagnosis* of the disease must be made from hemiplegic chorea, into which, according to Leube, it may sometimes pass, but the character of the movements in the two conditions is very distinct. It is sometimes more difficult to distinguish athetoid movements limited to the face from the facial tic or facial chorea. In such cases a history of some infantile cerebral injury will point to athetosis, and the character of the movements should also be considered.

The *prognosis* is hopeless as regards cure.

Treatment.—Improvement may sometimes be obtained in the idiopathic cases by the administration of arsenic or the bromids. Hammond has employed nerve-stretching with asserted good results.

HABIT SPASM (*Gowers*).

This is not regarded as a true form of chorea, though the affection is nevertheless characterized by localized contractile movements. We owe to Dr. S. Weir Mitchell the first accurate description of "habit-spasm." It occurs with great frequency in girls from seven to fourteen years of age. General ill-health (a neuropathic state) seems to precede the development of the disease in most cases, though by no means in all. Among specific *causes* may be mentioned overwork at school, fright, eye-defects (refraction-errors—De Schweinitz), nasal obstructions, and the occurrence of the disease in the parents or other older members of a family.

Symptoms.—In its commonest form there may be seen *spasmodic movements* of certain muscles of the face (grimaces), particularly vigorous winking or twitching of one side of the mouth or of the cervical muscles, associated frequently with a quick toss of the head to one or the other side, and sometimes with a peculiar sniff.

Less frequently the choreic movements manifest themselves in a shrug of the shoulder or a jerky, sobbing, irregular respiration, occasionally accompanied by a laryngeal sound—a cough or bark. Some authors mention strange tricks that are practised by these children, and Osler[1] speaks of a boy at his clinic who was in the habit every few moments of putting the middle finger into the mouth, biting it, and at the same time pressing his nose with the fore finger.

The **prognosis** in the immense majority of cases is eminently favorable, recovery ensuing within three or four months; but in a small contingent the condition lasts throughout life, usually as a localized muscular spasm (grimace), despite treatment.

The **treatment** embraces two important steps: the removal of all discoverable causes, for which a most careful search must be instituted; and the betterment of the general physical and psychic condition.

In subjects whose health is impaired, efforts to restore the normal vigor of the constitution by fresh-air exercise, cold baths with friction of the body-surface, abundance of suitable food, massage, and the administration of hematinics, particularly iron, are to be assiduously carried forward. Arsenic has been advised, and in one of my own cases proved curative; it is to be employed in augmenting doses in the same manner as in acute chorea (*vide* p. 1151).

GENERAL TIC.

(*Maladie des Tics convulsif; Maladie de Gilles de la Tourette.*)

Definition.—A disease apparently psychic in nature and characterized by coördinated spasmodic movements, explosive sounds or words, and imperative ideas, without intellectual disturbance.

The **pathology** of the disease is unknown. It occurs in those suffering from neuropathic heredity, and usually indirectly. It most frequently commences in childhood—that is, before puberty—and affects either sex. The exciting cause is usually some emotional disturbance.

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[1] *Text-Book of Medicine*, p. 996.

Symptoms.—The disease generally commences in the *orbicularis palpebrarum*, the first movement being an uncontrollable winking. This, as a rule, is rapidly associated with movements of the muscles of the face, causing the patient to exhibit various *grimaces*; finally, other muscles of the body may be involved, and the patient is compelled to repeat many times some apparently purposeful and coördinated movement, as the brushing away of insects or the stroking of the beard. From time to time he emits sounds that may be either inarticulate cries or imitation of some animal, as the crowing of the cock or the barking of the dog, or the repetition of some obscene word (*coprolalia*). These movements are partially under the control of the will, and are diminished by occupation, but increased by emotion. At other times the patient is compelled to imitate sounds that he has just heard, no matter how unusual or unexpected (*echolalia*). A more curious symptom is the imitation of movements that he has observed (*echokinesis*), which may lead to most absurd or painful results. I heard of one man who threw himself violently to the ground when he saw another man fall; and of another having killed his baby, which he held in his arms, by throwing it violently away from him in imitation of a man handling fagots. Still another psychic symptom is the occasional development of imperative ideas. These usually take the form of a desire to recall some unimportant word or syllable (*onomatomania*) or the performance of difficult problems in mental arithmetic (arithmomania). One of my patients, a boy of fourteen years, before undertaking a definite act would repeat the words "ten, ten, ten," three times, followed by a rapid count of figures from one to ten. If riding in a public conveyance, he would do the same, endeavoring to finish before reaching a definite place, as a street-corner, or before hearing the sound of the voice or whistle of the conductor if in a trolley-car. A failure to accomplish the task was cause for intense mental worry. These patients are usually affected at the same time with a certain degree of melancholia or anxiety that interferes to some extent with their normal life. The disease is, as a rule, very obstinate, and ordinarily continues throughout life.

The **differential diagnosis** is usually rather difficult. From *chorea* it may be distinguished by the coördinated character of the movements and by the presence of coprolalia, also by the fact that the movements can usually be controlled partially by the will; from hysteria by the absence of stigmata and the presence of coprolalia; from paramyoclonus multiplex it may be known by the coördinated character of the movements and the absence of increased reflexes.

The **prognosis** is unfavorable for cure; death, however, almost never occurs as the result of the disease.

The **treatment** is symptomatic, and consists in putting the patient in the most favorable physical condition possible; also hydrotherapy, change of climate, tonics, and the correction of any atonic condition are all useful measures. Potassium bromid to a certain extent controls the paroxysms when they become very severe.

SALTATORIC SPASM.

(*Jumpers; Latah; Palmus.*)

Definition.—This is a term applied to a peculiar clonic contraction occurring in the lower legs of a patient on attempting to stand upright. The disease was first described by Bamberger. It appears to occur more frequently in men than in women, and usually in individuals who have suffered from other functional diseases. Occasionally it appears in those who exhibit hysteric phenomena. The condition may develop after severe exertion, and sometimes appears during convalescence from an acute disease. In one of my own cases the condition suddenly arose in the course of habit-chorea. Saltatoric spasm is not a clinical variety of true chorea.

Symptoms.—When the patient attempts to stand violent clonic convulsions take place in the muscles of the legs, particularly of the calves. These may cause the patient simply to rise on his toes, or they may be so severe as to cause him to spring from the ground, in which case he usually falls. As soon as he lies down the spasms disappear, but they may be produced in patients lying in bed by pressing against the feet.

The **prognosis** is generally favorable. The attacks usually last for a period of from two days (Gowers) to a few weeks, but a few cases have been recorded that persisted throughout life. Gowers recommends diaphoretic treatment. Antispasmodics may also be employed, and in those cases with hysteric stigmata suggestion is useful.

CHOREA MAJOR.

(*Pandemic Chorea.*)

This is a form of hysteria (*hysteric chorea*). As early as the Middle Ages it prevailed as a pandemic affection, and was definitely traceable to religious ardor, excitement, or feeling. Writers upon the subject frequently instance an outbreak that occurred among the pioneer settlers in Kentucky. The convulsive movements are general, violent, and continuous; they are aggravated by attempts at voluntary control.

The **diagnosis** rests chiefly upon the causal religious excitement, the character of the convulsions, and the associated hysteric manifestations.

The **treatment** is wholly mental and moral, with the occasional use of drugs to produce quiet and sleep.

PARALYSIS AGITANS.

(*Shaking Palsy; Parkinson's Disease.*)

Definition.—A chronic disease characterized by a tremor; by the peculiar character of the speech and gait, and by a progressive, but very seldom complete, loss of power.

Pathology.—Lesions that are probably only senile in type have been frequently described. There are peri- and endarteritis, irregular degenerations in the posterior columns, and numerous amyloid bodies. Other observers have noted changes in the motor cells of the cerebral cortex.

Etiology.—Paralysis agitans is a disease of adult life, developing in the large majority of cases between the fortieth and forty-fifth years; it is met with more often in men than in women. No definite etiologic factor is known, though, as with most, if not all, nervous diseases, it is predisposed to by mental strain, worry, or trouble of any kind.

Symptoms.—The first evidence of the disease is the *tremor*, slight at first, and in the extremities, the hand usually being the first to betray it. The movement is very characteristic, the thumb and fore finger being approximated as in the act of making a pill. At the same time the hand is semi-rotated and the forearm trembles more or less as a whole. The upper arm is either but slightly or not at all affected. The legs are also but slightly implicated. The tremor is most noticeable when the patient is sitting with one leg crossed over the other, the foot then being sure to be in more or less constant motion. When the head is involved (rather the exception than the rule) the motion is a nodding one. The tremors cease when the patient sleeps, but are continuous during waking hours, though it is not rare to meet with cases in which, during purposeful acts necessitating the use of the affected parts, the tremors diminish or even cease temporarily, to return as soon as the voluntary motion is completed. The latter movements, it will be noticed, are awkward, and as the disease advances they become more and more stiff. This *rigidity*, with its consequent impairment of activity, is another cardinal feature of the disease. The patient's movements are slow and apparently measured. There is some impairment of power also, but it is slight, and may be rather from disease than from a direct nerve- or muscle-involvement.

Two of the most striking symptoms of the disorder are the *gait* and *attitude* of the patient. He walks with head and body bent forward, eyes directed toward the ground a short distance ahead, and takes short, mincing, and somewhat hurried steps (*festination*), giving one the impression that he is about to fall, which he would do but for each successive step, which, as it were, gives him a fresh center of gravity. His station is equally striking. The head and back are bent forward, the feet are kept some little distance apart, and one in front of the other, while the arms are slightly flexed and pendulous. From time to time the patient will make a slight forward movement (*propulsion*), or else, if walking, bend or fall backward (*retropulsion*). The facial muscles are set, the eyebrows arched, and the whole expression is "mask-like." The general slow character of all movements, except walking, which is necessarily quicker, is imparted to the speech, though after a sentence is begun the balance may be rendered normally or even hurriedly. The voice may be high-pitched. There are no trophic or sensory symptoms, and the reflexes are normal. Apart from the diffidence, amounting in some cases to a positive dislike for meeting people, and the melancholia occasionally induced by brooding over the affliction, there are no mental changes.

Course.—The disease is almost always of slow onset and of insidious progress. Very rarely the earlier symptoms may develop somewhat rapidly, but in every case their further progress is slow. Disappearance of tremor has been observed in the side affected by a subsequent hemiplegia. The course may be interrupted from time to time; even seeming improvement may take place, but it is not maintained. The disease lasts for years, and the patient usually dies of some intercurrent disease.

The **diagnosis** is not at all difficult when the tremor, attitude, gait, cases of true tetany or are hysteric in nature. There is ce that it is due to an intoxication occurring in the course rbid condition. Tetany is frequently associated with it also occurs in connection with gastro-intestinal dis- ilatation of the stomach, diarrhea, and intestinal para- egnancy and lactation, and it is associated with the myx- evelops after the removal of the thyroid gland. Exposure to ten preceded the disease. Occupation seems to exert a remark- nfluence upon the predisposition to it, the great majority of those ected being shoemakers or tailors. In childhood males are far more requently attacked than females, but in adult life this proportion is reversed. Heredity may have some influence, since tetany often occurs in several members of the same family. It is much more common in the winter months, and, curiously enough, it appears to be endemic in certain ocalities, particularly Leipsic and Vienna.

The **symptoms** fall naturally into two groups: (1) Those of the paroxysm, and (2) Those of the interval. (1) The **first symptoms** of an attack usually consist of peculiar sensory disturbances in the limbs, either tingling, formication, pain, or numbness, and these may precede the attack for some hours or even days. Stiffness of the muscles usually begins in the fingers. There may be slight clonic movements at first, but this is not frequent. The limbs are symmetrically involved. The *spasm* commences first in the hand, the fingers being straightened and flexed upon the hand, and bunched so that the hand has been likened to that of the scrivener or obstetrician. The spasm then extends to the muscles of the forearm and arm, and usually also to the feet and lower limbs. If the cramp is slight, the pain may be insignificant, but ordinarily it is severe, and is increased by attempts to overcome the contractures. The muscles are hard, painful upon pressure, and occasionally fibrillary twitchings may be observed. There is sometimes a slight edema, and often sweating of the limbs. The paroxysms may last for several minutes or for several hours or even days, and may even persist during sleep. If, however, the period is very long, remissions are usually observed. Sometimes a series of paroxysms may occur with considerable regularity. Attacks are more likely to occur at night, and they may also be brought on by prolonged and severe muscular effort, or by emotional shocks. Besides the paresthesiæ in the affected extremities, the patient may suffer from severe headache or pain in the back and neck.

(2) The **symptoms of the interval** are—*Trousseau's sign*—*i. e.* the possibility of causing an attack by prolonged pressure upon the main nerve-trunks or vessels of the limbs. Fränkel-Hochwart has shown that pressure upon the nerves is essential; pressure upon the vessels acting secondarily if at all. *Chvostek's sign* is a peculiar excitability of the muscles of the face, so that spasms are produced if the trunks of the facial nerve are lightly percussed by a hammer. This occurs in other conditions, particularly the cachexiæ, but in most cases of tetany the spasm occurs if the skin of the face is lightly stroked; and this reaction appears to be pathognomonic. *Erb's sign* consists of a greatly increased electric excitability of the muscles, and, occasionally, of an alteration of the electric reaction, so that AClC may be greater than KClC. Moreover, AOTe is often obtained, and, in at least 2 cases, KOTe has also been noted. The

and rigidity have developed. During the earlier stages it may be confounded with *multiple sclerosis*, though this condition develops earlier, and the volitional character of the tremor, the nystagmus, and the scanning speech should serve to differentiate it.

Treatment.—The medical management of the disease is absolutely without avail. No drug has been found to exercise the slightest influence on its course. Graduated exercise, however, tepid baths, and massage should be employed to keep up the tone of the muscles.

OTHER FORMS OF TREMOR.

1. **Hereditary tremor** has been described by C. L. Dana, who has also reported interesting cases. It may commence in infancy and continue till old age, unaccompanied by detectable lesions.

2. **Simple tremor**, lasting a longer or shorter period (oftener it is comparatively brief), is a rare condition and without serious possibilities. Its etiology is unknown, though it is sometimes aggravated by nervous shocks and other debilitating conditions.

3. **Senile Tremor.**—This is common in old persons, and rarely appears before three-score-and-ten years. It is excited by muscular motions, is always fine, and affects chiefly the hands and arms; more rarely the neck is also involved, and the head may then be seen to tremble.

4. **Toxic Tremor.**—This results from the action of alcohol, lead, mercury, tobacco, and other poisonous substances. I recently saw a case that followed the prolonged use of arsenic for an anemic condition.

5. **Smoker's tremor** is a recognized variety. The tremulousness produced by alcohol, lead, and opium will be considered in the discussion of poisoning from these substances (*vide* The Intoxications).

6. **Hysteric tremor** will receive more elaborate mention elsewhere (*vide* Hysteria, p. 1168).

TETANY.

Definition.—A disease of unknown cause, characterized by paroxysms of tonic cramp that usually affect the flexor muscles of the extremities, by sensory disturbances, and by a peculiar alteration of the electric reaction of the muscles.

Pathology.—Distinct morbid lesions of the nervous system have not been found in all cases. Slight vascular changes in the brain and cord and vacuolization of the ganglion-cells have been described, but these are not peculiar to this disease. It has been supposed that changes ought to be found in the motor nerves, but the most careful observers have failed to detect them.

Etiology.—Tetany may occur in epidemics, and has, therefore, been supposed to be infectious. There is some doubt, however, whether these epidemics are cases of true tetany or are hysteric in nature. There is also some evidence that it is due to an intoxication occurring in the course of some other morbid condition. Tetany is frequently associated with infectious diseases; it also occurs in connection with gastro-intestinal disorders, especially dilatation of the stomach, diarrhea, and intestinal parasites, during pregnancy and lactation, and it is associated with the myxedema that develops after the removal of the thyroid gland. Exposure to cold has often preceded the disease. Occupation seems to exert a remarkable influence upon the predisposition to it, the great majority of those affected being shoemakers or tailors. In childhood males are far more frequently attacked than females, but in adult life this proportion is reversed. Heredity may have some influence, since tetany often occurs in several members of the same family. It is much more common in the winter months, and, curiously enough, it appears to be endemic in certain localities, particularly Leipsic and Vienna.

The **symptoms** fall naturally into two groups: (1) Those of the paroxysm, and (2) Those of the interval. (1) The **first symptoms** of an attack usually consist of peculiar sensory disturbances in the limbs, either tingling, formication, pain, or numbness, and these may precede the attack for some hours or even days. Stiffness of the muscles usually begins in the fingers. There may be slight clonic movements at first, but this is not frequent. The limbs are symmetrically involved. The *spasm* commences first in the hand, the fingers being straightened and flexed upon the hand, and bunched so that the hand has been likened to that of the scrivener or obstetrician. The spasm then extends to the muscles of the forearm and arm, and usually also to the feet and lower limbs. If the cramp is slight, the pain may be insignificant, but ordinarily it is severe, and is increased by attempts to overcome the contractures. The muscles are hard, painful upon pressure, and occasionally fibrillary twitchings may be observed. There is sometimes a slight edema, and often sweating of the limbs. The paroxysms may last for several minutes or for several hours or even days, and may even persist during sleep. If, however, the period is very long, remissions are usually observed. Sometimes a series of paroxysms may occur with considerable regularity. Attacks are more likely to occur at night, and they may also be brought on by prolonged and severe muscular effort, or by emotional shocks. Besides the paresthesiæ in the affected extremities, the patient may suffer from severe headache or pain in the back and neck.

(2) The **symptoms of the interval** are—*Trousseau's sign*—*i. e.* the possibility of causing an attack by prolonged pressure upon the main nerve-trunks or vessels of the limbs. Fränkel-Hochwart has shown that pressure upon the nerves is essential; pressure upon the vessels acting secondarily if at all. *Chvostek's sign* is a peculiar excitability of the muscles of the face, so that spasms are produced if the trunks of the facial nerve are lightly percussed by a hammer. This occurs in other conditions, particularly the cachexiæ, but in most cases of tetany the spasm occurs if the skin of the face is lightly stroked; and this reaction appears to be pathognomonic. *Erb's sign* consists of a greatly increased electric excitability of the muscles, and, occasionally, of an alteration of the electric reaction, so that AClC may be greater than KClC. Moreover, AOTe is often obtained, and, in at least 2 cases, KOTe has also been noted. The

last two reactions occur in no other condition. ***Hoffmann's sign*** consists of an increased reaction of the sensory nerves to electric stimuli. The facies of the patient is peculiar and characteristic. The face is slightly swollen, dusky, and expressionless, but if carefully examined usually no edema can be detected. Often this swelling is also found in the hands and feet, and may be associated with distinct enlargement of the veins. Even during the interval the feet when at rest have a tendency, particularly in children, to assume a slightly inverted and extended position.

The *sensory disturbances* are of considerable importance. Often in the affected limbs there may be slight diminution in sensibility. In addition to this, there are changes that can only be described as true hysteric stigmata—*i. e.* hemianesthesia, or symmetric areas of insensibility to pain or touch. Often there are points of special sensitiveness on the spinal column, and in some cases pressure upon these will precipitate an attack. Distinct *trophic changes* have also been observed, consisting of herpetic eruptions along the course of the nerve-trunks, falling out of the hair, and sometimes even painless ulceration. The contractures are not invariably limited to the extremities. Sometimes the muscles of the neck, back, and larynx are involved; sometimes also the diaphragm, and occasionally the compressor urethræ. Involvement of the larynx gives rise to stridulous respiration; involvement of the diaphragm to severe dyspnea; when the urethra is compressed there is retention of the urine. *Fever* occurs in about one-half the cases; it is slight and generally limited to the paroxysm. The urine usually contains a large amount of phosphates, and less frequently indican is present in excess.

The **differential diagnosis** is very easy, as a rule, if the patient is seen during a paroxysm. The severer forms may, however, be confounded with *tetanus* or *meningitis*. Tetany can be distinguished from the former by the fact that the spasm begins at the periphery and rarely affects the muscles of the jaw. From the latter it may be diagnosed by the absence of coma and the slighter degree of fever. The diagnosis from certain forms of *ergotism* is more difficult, and can often be made only by careful attention to the etiology. The *hysteric forms* cannot always be differentiated, for, as has been stated above, hysteric stigmata occur in true tetany; but an epidemic occurring among young women should give rise to a suspicion of the true nature of the malady.

Prognosis.—The duration varies from a few days to many months, the most obstinate forms being those due to thyroidectomy and chronic diarrhea. The disease cannot be said to have disappeared until the characteristic symptoms of the interval (Trousseau's, Erb's, and Chvostek's signs) can no longer be elicited. The prognosis is usually favorable, nearly all cases tending to spontaneous cure. Death, however, may occur from chronic diarrhea, from respiratory failure when the diaphragm is involved, and from cachexia strumipriva.

The **treatment** is purely symptomatic. The patient should be placed in the most favorable hygienic conditions and given plenty of nourishing food. During the spasm bromids or chloroform-inhalations seem to give the best results. The most important therapeutic measure is the correction of the underlying cause. Thus, in children rachitis is almost invariably associated with tetany, and the most efficient remedies are iron and cod-liver oil. Intestinal disorders should be treated according to the principles laid down in the discussion of these diseases. The form due to removal of the thyroid gland always disappears under a course of thyroid-medication, while that occurring during pregnancy usually persists until delivery.

INFANTILE CONVULSIONS.

(*Eclampsia Infantilis.*)

UNDER this term are grouped a number of conditions, with convulsive attacks as the common symptoms.

Pathology.—The pathologic changes may be divided into two groups: (1) those bearing an etiologic relation to the convulsive attacks, and (2) those that are merely consecutive. Among the former are meningeal bleeding, tumor, gliosis (either hypertrophic or atrophic), and hydrocephalus. Then there are general conditions that seem to predispose to this condition or, at any rate, are frequently associated with it, such as rachitis. The consecutive lesions are hemorrhages into the meninges or into the substance of the brain and the spinal cord, an increase in the amount of cerebro-spinal fluid, and congestion of the pia or the substance of the brain.

The **causes** are: 1. Organic brain lesions. 2. Neuropathic tendency, that is manifested later as hysteria or epilepsy. 3. Emotional disturbances, as fright. 4. Rickets, in about 30 per cent. of all cases. 5. Acute infectious disease, especially as an initial symptom of pneumonia, and more rarely of scarlet fever, small-pox, and pernicious malarial infection. 6. Inflammation of the serous membranes, as meningitis, where the relation is direct, or pleuritis or peritonitis. 7. Kidney disease, in which they are uremic. 8. Peripheral irritation; dentition has long been supposed to be a chief factor in their causation, but it is now believed that the chief cause is the presence of rickets. Intestinal parasites have also been found, particularly the ascaris lumbricoides, and the convulsions have ceased after their expulsion. 9. Debility, especially that resulting from gastro-intestinal disorders.

The **symptoms** of the attack vary according to its intensity. In the most severe form they resemble in all respects those of an epileptic seizure. At first the eyes deviate upward or to one or the other side, and the gaze becomes fixed and staring; next there are *twitchings of the muscles* of the face, sometimes slight and limited to one side, and sometimes general, often involving the muscles of mastication and giving rise to trismus or gnashing of the teeth. Next there are *tetanic contractions* of the extremities, the fingers being strongly flexed, the hands flexed upon the arms, and the feet in the position of pes equinus or sometimes in the dorsal flexion, and both legs and arms rigidly extended. Often the muscles of the trunk are involved, and there is either opisthotonos or respiratory cramp, with excessive hardness of the abdominal muscles. This rigid condition is interrupted at brief intervals by sudden twitchings, or occasionally the convulsion becomes clonic instead of tonic, and there are repeated extensions and contractions of the extremities, shaking of the head, and quivering of the whole body. As a result of the respiratory cramp, *cyanosis* rapidly develops and may reach an extreme degree. The forced respirations give rise to a foam that collects about the lips, and is often mixed with blood from the bitten tongue. Urine is often, and feces occasionally, passed involuntarily. In nearly all cases unconsciousness is complete. Many of the slight attacks are accompanied by a cry or by an attack of screaming. The tetanic state usually lasts for a minute or two; then there are a few clonic movements, relaxation becoming rapidly complete, and the spasm is ended by a few deep respirations. The child may return to consciousness, although it is usually drowsy or stupid, or it may pass into a deep sleep from which it cannot be aroused. Often in the latter condition attacks will recur at irregular intervals, and sometimes a single attack may continue for some time, although from time to time there are slight twitchings followed by partial relaxation (*status eclampticus*—Lewis). The attack may come on suddenly, or, as is more frequently the case, it may be preceded by a period of restlessness and irritability. A milder form of the spasm consists of sudden fixation of the eyes, slight twitching of the body, and a peculiar dusky pallor that passes away in a few moments. In other rare cases consciousness may persist, although the patient is aphasic. Laryngismus stridulus is an analogous condition (*vide* Diseases of the Larynx, p. 481).

The **diagnosis** of the condition is very easy. The recognition of the cause, however, is very important and often difficult. Every case should be first examined for rickets, and then the gums should be investigated; also the condition of the child's nutrition and the presence of symptoms of gastric or enteric irritations. If fever exists, it is important to discover its cause. The character of the convulsion is often of value in distinguishing between the idiopathic or reflex type and that due to organic brain-disease. Convulsions beginning immediately after birth, or an injury, either persisting or else disappearing gradually, are probably caused by meningeal hemorrhage. An attack of a Jacksonian type would, of course, indicate the presence of a focal lesion; and if this be a tumor, there will probably be bulging of the anterior fontanel, severe headache, and the ophthalmoscope will reveal a neuro-retinitis. If, after the attack, pareses or paralyses are present, a focal lesion is still more likely. Hydrocephalus is usually recognized with ease. Some cases exist, however, in which it is impossible to discover any adequate cause.

The **prognosis** varies according to the etiology. In cases with organic brain-disease it is unfavorable as regards cure. In those forms that precede epilepsy or functional nervous diseases the spasms usually disappear after the first dentition, and the patients appear to have recovered for a time. In those, however, in whom the symptoms are due to some peripheral irritation or to rachitis, the outlook is fair, although even these now and then develop into permanent epilepsy. The convulsions themselves are either often immediately fatal, or so exhausting to the patient that he succumbs readily to the disease that produced them. In these cases the prognosis depends upon the frequency and severity of the attacks, death usually terminating those in which the status eclampticus has been established. The prognosis for ultimate cure depends also in part upon the length of time that the condition has existed; if but for a short time before an arrest has been established, recurrence is much less likely. Gowers, however, says that even after a year's duration permanent cure may sometimes be obtained.

The **treatment** naturally falls into two parts—that of the attack and that of the interval. Unquestionably, the most efficacious antispasmodic that we possess for this condition is chloroform. A few drops may be put upon a handkerchief and held carefully over the nose and mouth of the little patient. A very small quantity usually suffices, and the effect is almost instantaneous. In addition to this, chloral and the bromids may be given by the rectum, and it is often useful to add to these one of the coal-tar antipyretics, particularly antipyrin. Morphin may be given hypodermically. Formerly hot mustard-baths were much in favor, but unless they do good at once they are not likely to be of any use. In a very obstinate case under my care they were absolutely valueless, and were replaced by momentary immersion in ice-cold baths and vigorous friction, which seemed to act very favorably. If any known source of irritation is present, as an overloaded stomach, it should be relieved at once, if possible, by the stomach-tube or an emetic. An enteritis may be temporarily benefited by an enema or by a moderate dose of calomel. The treatment during the interval depends upon the nature of the cause. If rachitis exists, it should be treated according to the principles laid down in my discussion of this disease. If dentition is suspected, the gums may be lanced, but this should only be done when they present distinct signs of irritation. Gastro-intestinal disorders of any kind should be relieved as soon as possible, and intestinal parasites must be expelled. In infectious diseases the convulsions usually disappear after the initial stages, and require no further attention. In organic brain-disease, providing it be not syphilitic in nature, very little can be done. Finally, in those cases in which no cause can be discovered bromids are the only resource, and should be given in sufficient doses: from gr. iij–v (0.194 to 0.324) per day to children of six months, and from gr. v to x (0.324 to 0.648) to those between six and sixteen months.

OCCUPATION-NEUROSES.

Definition.—Conditions in which the performance of certain habitual coördinated movements is prevented by the development of cramp, tremor, paralysis, or pain. The commonest form is *writers' cramp* (graphospasmus, mogigraphia, scriveners' palsy).

The **pathology** of this condition is unknown. It is probably purely functional, and the discovery of appreciable lesions is not to be expected, though nodular thickening of the peripheral nerves has been described in a few cases.

The **etiology** is various. Males are far more frequently affected than females, the condition usually occurring in early adult life, although children are not exempt. The condition always occurs in those whose occupation demands much writing, and Gowers lays great stress upon improper methods of holding the pen, particularly those in which most of the writing is done from the wrist; that is, with the muscles of the forearm and hand. As scrivener's palsy occurs sometimes in those that write properly, and as a similar condition is not uncommon in other occupations, it seems unlikely that this is the most important cause. A person with a neurotic temperament is far more apt to be affected by the disease than one with a normal nervous system; we, therefore, frequently find it associated with hysteria, neurasthenia, or great bashfulness, and not infrequently it is possible to elicit a neuropathic heredity in the family history. It is also met with in certain other nervous diseases (epilepsy, locomotor ataxia—in the early stage.) Often the patients admit that at the time the disease developed they were suffering from severe anxiety.

Symptoms.—**Motor.**—When the patient attempts to write there is usually a cramp of the flexor muscles of the forearm, so that the pen is held more or less rigidly, and it is almost impossible to control its motions. Less frequently there is a cramp of the extensor muscles, so that the fingers are spread and it is impossible to hold the pen at all. Sometimes there is a sudden twitching, and the pen may be thrown altogether out of the hand. The spasm is nearly always tonic in character, but often it is associated with a fine tremor, and at times there are clonic movements. In some cases, and particularly those occurring in patients showing hysteric stigmata, there is a coarse, irregular tremor, most marked when the patient is under observation. Paresis is frequently associated with the cramp, so that the arm soon becomes tired and it is almost impossible to write. This fatigue may in a few moments progress to almost complete paralysis of the arm, but, curiously enough, both fatigue and paralysis disappear as soon as some coördinated movement other than writing is undertaken.

Sensory.—Pain is very common, and is neuralgic or cramp-like in character, being referred either to the muscles, bones, or joints. In intensity it varies from a dull ache to the most excruciating burning, and may form the only symptom, the muscles performing their work perfectly. At times it is sharply localized to one particular joint, affecting either the metacarpal bones or the fingers. Quite often the patient complains of a tingling or burning sensation in the limb, or it may be numb and the hand feels, when writing, as if a heavy weight were attached to it. Often there is tenderness either of the muscles or the nerves, which may be localized in certain points. In very severe cases *vasomotor disturbances* occasionally occur. The disease ordinarily commences slowly. At first the subject notices that the handwriting is not quite as perfect as before, a stroke occasionally going astray; later distinct spasms appear, and these are finally associated with pain.

The **diagnosis** is usually easy. Care must, however, be taken not to call every disturbance of writing writer's cramp; thus in paralysis agitans, in slowly-developing hemiplegia, in multiple sclerosis, and in locomotor ataxia disturbances of writing frequently—in fact, almost invariably—occur. Moreover, those cases in which hysteria or neurasthenia seems to be at the bottom of the trouble should be carefully differentiated from those that are apparently idiopathic.

The **prognosis** is rather unfavorable, though complete cure is sometimes attained.

The **treatment** consists first in a total cessation of writing; if this is impossible, various mechanical devices may be employed to use another set of muscles or the old ones rather differently, such as a thick penholder or one constructed with supports for the fingers. Local treatment of the arm in the form of electricity should be advised; the anode of a constant galvanic current of medium intensity should be placed over the sensitive points on the nerves and over the bodies of the muscles. The wire brush employed, with the rapidly interrupted faradic current, to stroke the painful nerves and muscles, affords great relief. Massage, and particularly careful and systematic exercises, are also of great value. At the same time, the general condition of the patient must not be neglected. In cases associated with hysteria, hypnotism may be employed, and in those associated with neurasthenia a treatment appropriate to this condition. If possible the patient should learn to use a type-writer and abandon penmanship altogether. Operative treatment is more likely to do harm than good.

The other occupations in which this disease occurs are—telegraphy, playing on musical instruments, cigarette-rolling, milking, and sewing. Strümpell suggests that stuttering is an analogous affection of speech.

PERIODIC PARALYSIS.

(*Paroxysmal Familiar Paralysis—Goldflam.*)

Definition.—A disease characterized by paroxysmal attacks of complete paralysis, and alteration in the electrical reactions and structure of the muscles, occurring in many members of a family.

Pathology.—Excised fragments of muscle show rarefaction of the fibers and slight vacuolation, without multiplication of the nuclei or proliferation of the connective tissue.

Etiology.—The disease is purely hereditary. Both sexes are affected. The attacks appear to be more frequent in summer, and often seem to occur after overfilling the stomach.

Symptoms.—The attacks are preceded by *prodromes* in the form of vague discomfort or paresthesia. The patient then usually falls asleep and awakens completely paralyzed. Speech, deglutition, and the sphincters are unaffected. During the attack there is often transient *albuminuria*, with blood-cells in the urine. The *reflexes* are abolished. The *paralysis* lasts from twelve hours to three days, and then there is an outbreak of perspiration, with gradual recovery, the muscles of the head first regaining power. During the interval the muscles react slowly to faradism and galvanism, and are easily thrown into CaC tetanus. The muscular changes have been demonstrated in children of an affected family in whom the disease had not yet developed.

Diagnosis.—Usually easy. Goldflam was once puzzled by a case of acute ascending paralysis.

Prognosis.—The disease does not kill; but there appears to be no tendency to recovery.

Treatment.—This involves only caring for the children during the attack. Even persistent starvation does not decrease their frequency.

HYSTERIA.

Definition.—A condition of the general nervous system partaking of the natures of both a neurosis and a psychosis, and characterized by a vast multiplicity of clinical manifestations, all indicative of a loss of voluntary control over inhibitory and active nervous influence.

Pathology.—Hysteria is to be regarded as essentially a morbid entity, without, however, any tangible pathologic features. The most careful *postmortem* examinations of subjects who have while in life manifested pronounced hysteric symptoms have failed to reveal any organic nervous alterations, however slight. The occurrence of the affection in men as well as in women excludes the former theory of a uterine pathology, which, though an idle fancy, held sway for so many centuries and gave origin to the name by which the condition is generally recognized.

Etiology.—There are a large number of predisposing and exciting factors, all of which, however, may be grouped under a few dominant heads. Thus among the former must be mentioned, pre-eminently, *heredity*. The investigations of many neurologists and alienists of divers lands have gone far to demonstrate that at the foundation of the vast majority, if not of all, of the hysterias is to be discovered an inherited neurotic tendency or temperament. The family histories of these patients generally reveal a large number of consanguineous, neurotic, or hysteric individuals. It is apparently in close relationship with the various psychoses and major neuroses (epilepsy, chorea, tetany); and with the so-called rheumatic diathesis.

In the process of transmission one generation may entirely escape the pernicious influence, and successive generations may manifest strikingly different evidences of the disease, in one the neurotic and in another the psychic element predominating. A curious phenomenon that is worthy of mention is the apparent *contagiousness* of hysteria; moreover, the baleful influence one neurotic individual exerts over the unfortunates of this temperament explains the so-called "hysteric epidemics" that have swept over communities, and even over vast tracts of land or entire countries, at different periods of the world's history. Similar, though limited, outbreaks may still be seen in the nervous wards of hospitals or in religious and political conventions, and these depend largely upon the general prevalence of the neurotic disposition untempered by a virile will-power.

The hysteric temperament may be, and often is, fostered by improper and pernicious modes of life, especially by luxurious and sensuous living and by the habit of gratifying every desire of the will during early life. It is manifested at this early stage of the individual's existence by hypersensitiveness, brilliancy, undue enthusiasm, and a more or less erratic turn of mind.

Contrary to the prevailing opinion, hysteria is not limited to the female sex, although they are the chief sufferers from the more dramatic forms. Instances of a most rebellious nature not infrequently occur in the opposite sex.

Age.—The condition is generally encountered between the ages of fifteen and thirty years. After the latter age the frequency of the disease rapidly diminishes, and prior to the onset of puberty the cases recorded are also comparatively few in number, although exceptionally it is seen in infancy.

A very influential factor in the production of the disease is the *lack of proper mental development.* It stands to reason that those who are coarse and illiterate, and who have not been taught the lessons of self-control, and who are subject to the various and multiplex superstitions that are ever prevalent among the masses, will respond more quickly and more generally to the causes that tend to destroy mental equilibrium. Hence, hysteria or insanity shows its rankest development among those whose education and culture are defective. This is, however, by no means an inevitable law, for over-stimulation of the faculties may be just as deleterious as under-stimulation, and some of the brightest lights of the world have manifested at various periods of their lives decided hysteric symptoms.

Improper hygienic surroundings, tending as they do to enervation and physical depression, are influential predisposing factors in the evolution of hysteria. In addition to poor and insufficient food, lack of proper ventilation, overcrowding in foul habitations, and insufficient bathing, must be mentioned the enervating influence of hot and moist climates. It is generally conceded that more cases of hysteria occur in the warm than in the colder portions of the temperate zones, and that this proportion increases *pari passu* with the height of the temperature.

Finally, the causal influence of the chronic toxemias (alcoholism, morphinism, absinthism, saturnism and intoxications by other metals) is to be mentioned. In systemic poisoning the depraved condition of the physical reacts upon the mental organism, and sooner or later hysteric manifestations may be found to coexist with the original toxic phenomena.

The *exciting causes* of hysteria may be grouped as follows:

(1) Most commonly psycho-neurosis follows some profound emotion or mental or moral shock. Thus, in neurotic males it may be excited by excessive and protracted business-worry or excitement, or by active competition in certain lines of occupation, or by some heavy and unexpected monetary reversal. In females it is not uncommon as a sequel to the establishment of puberty and the menstrual function, or to the physiologic arrest of menstruation at the period of the climacteric. Especially is it prone to develop in young and illegitimately pregnant women, or during the first pregnancy in newly-married women of a neurotic temperament. Great religious excitement during the progress of a revival-wave and profound political upheavals have been most potent in establishing the disease in numerous instances; and other profound mental impressions, of fear, grief, or great and unexpected joy, have assumed the exciting rôle.

(2) Extreme physical prostration, the result of some very acute or much protracted chronic disease, may exert an etiologic effect. Thus, some of the most marked and intractable forms of the disease have resulted from the specific fevers (typhoid, typhus, and the other exanthemata), while it is not rare in a varying degree in the final stages of tuberculosis, chronic nephritis, and other grave constitutional affections of long standing.

(3) The so-called "traumatic hysteria" has come to occupy a prom-

inent place in the etiologic category of the disease. Especially do we find the incurable varieties of hysteria resulting from a slight or it may be a more severe traumatism. It must be remembered that a considerable period of time may intervene between the date of the injury and the appearance of the initial hysteric symptoms, so that in all cases it becomes of the utmost importance to make a careful study of the patient's history for signs of traumatism, however remote. It has also been noted that a repeated minor traumatism may finally result in some hysteric manifestations. So well recognized has this fact become that it is not unusual to discover an hysteric element in certain occupational ailments. Thus the irritable neurasthenic condition of the centro-spinal axis so common among the employees of railroads, and known as the "irritable hysteric" or "railroad spine," is now acknowledged as the type of this traumatic hysteria. In fact, any slight injury or a blow upon the head or a trivial injury of another portion of the body, especially if it be brooded upon, may ultimately cause hystero-epilepsy.

(4) Finally, in a limited number of cases sexual excesses and masturbation are the influential factors in the production of hysteria. The sexual origin of the disease, which was formerly the chief etiologic theory, has now come to occupy only a minor causative rôle, but the tendency to abolish it entirely as a cause of the disease is as much an error in the opposite direction. These sexual cases, though few in number, do exist, and are especially to be found among the class of so-called sexual perverts.

Clinical History.—Clinically, hysteria presents three well-marked stages, known respectively as the *prodromal*, the *convulsive*, and the *latent*. The latter is also designated as the *interconvulsive stage* or the *period of the stigmata*, and during this period the number of the symptoms and their complexity almost baffle attempts at classification; they can, however, best be portrayed by presenting them under the heads of the various systems (*vide infra*).

(1) **The Prodromal Stage.**—The prodromes are invariably present, and at times they are more marked than at others. They are evident alike to both patient and physician, and are largely psychic in nature. There is noted a marked mental depression associated with introspection, and, it may be, with a form of mild mania or of melancholia. A condition of aprosexia develops, and the patient becomes irritable, restless, and discontented. The mental derangement may manifest itself in the form of delusions or nightmare, and there is a characteristic neglect of the toilet and attire. Naturally, there result disturbances of the gastro-intestinal tract—viz. anorexia, nausea, vomiting, constipation, and perversions of taste. The patient is pale, and the expression assumes an anxious or, it may be, vacant cast. These phenomena persist for several days, and are followed by emotional disturbances—spasms of hysteric laughing and crying—that immediately precede the *aura*, which is as marked a feature in hysteria as in epilepsy. It may assume one of a number of forms, but more commonly it has an ovarian, a cervical, a cerebral, or a surface or cutaneous origin (unilateral). Very frequently the convulsion is preceded by a condition of extreme sensitiveness and pain in one or both ovarian regions, so that the lightest touch at a point on the abdominal surface one inch above Poupart's ligament, and midway

between the pubis and the anterior superior iliac spine, will elicit exquisite tenderness. This is so constant and characteristic that many patients can invariably predict the onset of the convulsion. Not infrequently the aura begins in the neck, the patient experiencing a sensation as of a ball lodging in the throat (*globus hystericus*): this is due to a spasmodic contraction of the muscles of the pharynx and esophagus, and is accompanied by tachycardia and a sense of suffocation. If the aura originate above the scalp, it is characterized by the sudden appearance, generally in the top of the head, of a severe neuralgic pain, as if produced by the entrance of a nail (*clavus hystericus*); this is frequently associated with vertigo and tinnitus aurium. The aura, finally, may appear in the form of spots of cutaneous tenderness, mainly localized upon the trunk, to which areas has been given the name of *hysterogenous zones*.

(2) **The Hysteric Convulsion.**—Closely following upon the footsteps of these prodromes, and immediately following the aura, the hysteric convulsion may appear. Most commonly this is epileptoid in nature; rarely it assumes a less common type. Hence it becomes necessary to describe several of the forms of the convulsions—viz. (*a*) the *epileptoid* (hystero-epilepsy); (*b*) the *gymnastic* (clownism); (*c*) the *emotional cataleptic*, or *dramatic*; and (*d*) the *delirious*. All of these forms may be present in the same attack, the one passing quickly into the other, or, as in the abortive cases, one or the other form will predominate. Briefly described, the characteristic features are as follows:

(*a*) *Epileptoid* (*Hystero-epilepsy*).—Immediately upon the appearance of the aura the patient commonly emits a shriek and falls upon the floor or in some convenient place, taking special care to do herself no injury: this is in strong contradistinction to the true epileptic spasm. The head and limbs are thrown about by more or less violent clonic muscular spasms, and at times a condition of opisthotonos or other trunkal contortion (emprosthotonos, pleurosthotonos) may be noticed; these muscular movements, however, are more or less volitional, and are not the aimless movements of the true epileptic. The patient may or may not foam at the mouth. There is a constant twitching of the eyelids and the eyes are rolled about, but apparently retain a more or less observant expression. Consciousness, as a rule, is not fully lost. The facial muscles are distorted, rapid changes of expression being noted (*hysteric trismus*), and respiration is somewhat impeded. As the convulsion passes off the movements gradually subside, and the patient generally sinks into a state of quiescence or, it may be, into a light sleep. This may be followed by complete temporary recovery, or the epileptoid may pass into one of the other forms of the convulsive seizure. The duration of the spasm as described is usually longer than that of a true epileptic seizure.

(*b*) *The Gymnastic Form* (*Clownism*).—This stage is characterized by violent and grotesque muscular movements. Here are to be grouped all of the more curious manifestations of the disease recorded in the history of medicine. The most difficult feats of the contortionist are performed with apparent ease; the patient may suddenly begin to dance or jump at a most astonishing rate, persisting in the movements until she drops from pure physical exhaustion. The so-called religious ceremonies of the Shakers of Lebanon, Pennsylvania, and of the Jumpers of the Middle

Ages, are manifestations of this form of hysteria. In children the attack may appear as the so-called *beast-mimicry*, in which the movements or sounds of the lower animals may be simulated; such is also the explanation of the condition known as *spurious hydrophobia*. *Eclampsia nutans*, the "nodding spasm" or "salaam convulsion" of young children, is likewise a sub-variety of this form of the disease. Consciousness is never lost during this period.

(*c*) *The Emotional Cataleptic, or Dramatic Form.*—In this form the patient seems to suffer from delusions or hallucinations that are apparently the outcome of the preceding condition. The emotion that is most developed in the patient's moral constitution now dominates his spasmodic actions. As Lloyd aptly expresses it: "The third period of the hysteric convulsion is one of dramatic representation of emotional images, and these are of countless varieties, according to time and person." All of the manifestations of the cataleptic state are present. Sensation is largely abolished, consciousness is retained, and the patient is usually able to recall events that have transpired during the period. Especially common now is the assumption of dramatic and passionate attitudes, which, as described by Richer, include "the attitude of the cross, of defence, of menace, of appeal, of lubricity, of ecstasy, of dread of animals (as rats), of scorn," and the like. The body of the patient retains, at times for indefinite periods, whatever position is first assumed (*hysteric catalepsy*). In some cases the patient falls into a condition of apparent sleep or narcolepsy (*hysteric sleep, hysteric somnolence, hysteric trance*) of varying degrees of intensity; this may persist for any period of time, from a hour or two up to weeks, months, or even years. In these extreme cases, while the patient at first appears to be in a normal sleep, sooner or later the body assumes a corpse-like appearance, with pale, waxy skin, almost imperceptible respiration and cardiac action, and a subnormal temperature.

(*d*) *The Stage of Delirium.*—The final stage of the hysteric convulsion is but a continuation of the preceding period, with, however, a cessation of the muscular movement to a great extent. The tendency now is to delirium of a mild type, tinged with more or less melancholia. Consciousness is maintained throughout this stage, and there now appear some curious motor phenomena that may persist for days or weeks. These may consist in the abolishment of muscular power in various portions of the body. Very often associated with these motor phenomena is noted a condition of mutism that lasts for indefinite periods of time.

Hysteric paralyses occur, and may simulate any form of the organic paralyses (monoplegia, hemiplegia, paraplegia). In many cases the patient is left with a more or less permanent spasm of a single set of muscles or of associated sets. These so-called *hysteric contractions* may affect any portion of the body. One arm may be bent at the elbow or one leg at the knee; in the former case the fingers are rigidly contracted and embrace the thumb, which is crossed upon the palm, while in the latter the toes are strongly flexed upon the plantar surface and the foot is inverted The ankle- and knee-jerk persist. In other cases a curious spastic ga is produced that closely simulates that of spinal sclerosis. The muscl of the hips, shoulder, back, and neck (*hysteric torticollis*) may share in t process. In women the muscles of the diaphragm and abdominal w may be involved (*hysteric pseudo-cyesis*). *Hysteric rotatory spasm,* *teric athetosis*, and *hysteric tremor* are all dependent upon a spasmodic action of the muscles affected. The convulsive seizure generally is of *short duration*, lasting but fifteen to thirty minutes. Occasionally, however, there is developed a prolonged convulsive status, during which time the patient continually falls from one convulsion into another, until one hundred or more may be recorded and the excess of nervous power is exhausted.

(3) **The Latent or Interconvulsive Stage, or Period of the Stigmata.**—After the convulsive attack the patient enters upon a more or less prolonged interval of comparative quiet; this is characterized, however, by numberless and varied phenomena—the *hysteric stigmata*. The whole course of the affection may be comprised in this period, convulsions being absent. As I have already stated, these can best be described under the heads of the various systems:

(*a*) **The Nervous System.**—This presents the most characteristic hysteric stigmata. They are generally grouped into the three classes of *motor*, *sensory*, and *psychic*.

The *motor symptoms* have already been referred to in part in the description of the hysteric convulsion. They embrace every variety of muscular pathology, from obdurate paralysis to and including tremor, incoördination, and tonic spasm or contraction. The hysteric paralyses, as stated, may be absolute or partial, and either general or limited to groups or to individual muscles, and may simulate any variety of true paralysis of organic origin. There is usually noted an exaggeration of the reflexes of the affected side, muscular wasting may or may not be present, and it is not at all uncommon to find associated contractures and sensory phenomena. The paralyzed limb or limbs show evidences of circulatory disturbances, as edema and bluish discoloration. In the paraplegic cases it is unusual for trophic disturbances (bed-sores) to appear; some atrophy of the affected muscles may, however, be noted. Hysteric tremors are not infrequent, and are usually well marked and persistent. They are generally associated with contractures and other hysteric stigmata.

Hysteric incoördination (*hysteric ataxia*) has also been termed astasia-abasia; it is one of the rarest of the motor phenomena of hysteria. The name implies an inability to stand or walk, although muscular power in the legs and trunk is retained (*vide* Astasia-abasia, p. 1185). *Hysteric contractures* may occur as distinct phenomena or may be associated with some form of hysteric paralysis. Usually the contractures occur with startling abruptness, and are most intense and persistent, resisting even the effects of sleep and the anesthetics. There may be associated sensory phenomena. The toes and the fingers are most frequently the seat of contracture, but the muscles of the face and neck may likewise share in the affection.

Sensory Symptoms.—The anesthetic, hysteric, and paresthetic varieties are noted. Patients showing hysteric anesthesia are able to run pins into themselves without showing the slightest degree of suffering. The anesthesia may be general or it may involve but half of the body or scattered areas of the cutaneous surface. *Segmental anesthesia* is the term applied to that condition in which a limb or a portion of a limb is involved. Not only is the skin affected, but often the deeper tissues as well, and there is generally some vasomotor involvement, as is shown by the fact that punctures by a needle are not followed by bleeding. There is often associated an anesthesia of one or more of the special senses (*hysteric amaurosis* or *blindness*, *hysteric deafness*, and *hysteric anosmia*). The conjunctivæ very frequently escape. The anesthesia is severe, as a rule, immediately after an hysteric convulsion, but it may be entirely absent throughout a given case of hysteria. There is often contraction of the field of vision or inversion of the color-fields, the red being more extensive than the blue.

Hysteric hyperesthesia is also a frequent clinical manifestation, and is generally confined to limited areas, as the ovarian, mammary, or spinal regions, or to one of the larger joints (*hysteric joint*), simulating organic disease of the part. These conditions can be recognized by etherizing the patient, when perfect mobility of the affected joint is noted. When one of the mammæ is involved, the organ becomes exceedingly painful to the touch and slightly edematous (*hysteric breast*). *Hysteric paresthesiæ* include the common varieties of formication, dead fingers, and the like.

Psychic Symptoms.—These have already been mentioned among the prodromal symptoms—violent and capricious changes of temper, mental depression and unrest, melancholia, and a notable lack of volitional power whereby the patient becomes especially open to the suggestions of the hypnotist. Such patients may develop into that strange condition known as "double consciousness."

(*b*) **The Digestive System.**—Among the usual clinical manifestations of this group may be mentioned *anorexia* (which may be complete), a strange and persistent perversion of taste, occasional uncontrollable vomiting without nausea (*hysteric vomiting*, *anorexia nervosa*), marked dyspepsia, and at times extreme emaciation with dryness and a parchment-like feel of the skin. Excessive flatulence and the peristaltic unrest of Küssmaul may be marked symptoms, as may also either diarrhea or constipation. *Hysteric hematemesis* is the result of swallowing blood; this is usually drawn from the gums or tonsils, or it may be taken secretly by the patient from other external sources.

(*c*) **The Respiratory System.**—Difficulty of respiration (*hysteric dyspnea*) is not uncommon, and is characterized by an extreme rapidity and shallowness of the respiratory movements. These are much out of proportion to the heart-beats, and are unassociated with cyanosis. In other cases the disturbance assumes the form of uncontrollable yawning, sneezing, or hiccoughing, due probably to a spasmodic action of the involuntary muscles of the bronchial tubes and diaphragm. *Hysteric cough* is a troublesome, and very often a stubborn symptom, occurring especially in young females. It is dry and barking, and, as a rule, unaccompanied by expectoration. At times it may be followed by *hysteric hemoptysis*, in which there is an escape of light-red fluid from the pharyngeal mucosa. *Hysteric aphonia* is also frequently noted; in this condition the patient speaks in a scarcely audible whisper. In such cases restoration of the voice is as of sudden occurrence as is its loss. In one of my own cases aphonia manifested almost true intermittence for a period of five years, while during the last two years or over it has stubbornly persisted even without remission.

(*d*) **The Vascular System.**—*Hysteric tachycardia* is often noted, and much less frequently *hysteric bradycardia* appears. A variety of *pseudo-angina* is not of rare occurrence (*vide* Angina Pectoris, p. 675). Very frequently the patient exhibits a localized flushing of the skin (*hysteric erythema*), and especially of the face and neck, or, as has already been noted, there may be an apparent bloodlessness of a part. Profuse general or localized sweating is not uncommon, and may at times be bloody.

Hysteric fever may be mentioned here as a rare manifestation, the bodily temperature usually being normal in hysteria. The elevation of temperature may be moderate or there may be an extreme hyperpyrexia (110°–120° F.—43.3°–48.8° C.), without grave results. If this be associated with localized neuralgia, it becomes a difficult matter to diagnose between the neurotic condition and organic disease of the apparently affected part.

(*e*) **The Urinary System.**—An excessive flow of urine (*hysteric polyuria*) is of very common occurrence, while the opposite condition (*anuria*) is much rarer.

Diagnosis.—The diagnosis of hysteria depends entirely upon the discovery and recognition of the hysteric stigmata; for one or more of them is always present. Of these the most frequent are areas of anesthesia, concentric narrowing of the visual field and inversion of the color fields, and hysterical aphonia, although any of the others that have been described may occur. If, in addition, hysterical crises are present or have been observed, the diagnosis becomes certain. A valuable feature is the inability to explain the symptoms by reference to the anatomy of the nervous system. It must not be forgotten that hysteria and organic disease may coexist, although this is relatively rare.

Differential Diagnosis.—Very important is it to distinguish between hysteric and true paralyses, and between hysteric and organic abdominal tumors. In the following tables the most striking points of difference between these conditions have been set down:

HYSTERIC PALSIES.	ORGANIC PALSIES.
Occur without a previous history of organic disease, but with a neurotic history. Traumatism may be the cause.	Are always secondary to organic disease of the neuromuscular system.
Are accompanied by other hysteric stigmata or perversions of sensation.	Hysteric stigmata are absent.
Are not accompanied invariably by wasting of the muscles involved.	Are always accompanied, sooner or later, by muscle-wasting.
Reactions of degeneration are absent.	Reactions of degeneration are present.
The paralysis is apt to be more or less transient and shifting.	The paralysis is permanent and marked.
The power of motion returns before sensation.	Sensation first reappears.
In hysteric hemiplegia the facial muscles are not involved.	The facial muscles of the same or opposite side are always involved in true hemiplegia.
Anesthesia generally causes relaxation of hysteric contractions.	Organic paralytic contractions are not affected by anesthesia.

Hysteric Abdominal Tumors (Pseudocyesis).	Organic Abdominal Tumors.
Almost invariably occur in neurotic women near the menopause.	Occur irrespective of sex.
The percussion-note is invariably tympanitic.	The percussion-note over the swelling is dull, or a dull tympany.
Anesthesia causes a disappearance of the tumor.	Anesthesia has no effect upon the tumor.
Is variable as to size and tonicity.	Slowly but steadily progresses in size.
Is accompanied by tympany and flatulence.	The bowels are not always distended by gas.

The differential diagnosis between hysteria and true neurasthenia, and epilepsy, will be found in the discussion of the latter two affections.

Prognosis.—As regards death, the prognosis in hysteria is good; true hysteric patients never die of the disease, nor does the hysteric spasm ever result fatally. As to an ultimate cure, however, the prognosis is very doubtful. If the disease occur early in life and if there is a marked congenital neurotic tendency manifested in the patient, there is almost no hope of effecting a permanent cure. In the acquired cases, under proper moral and hygienic control great benefit may be effected or even an absolute cure recorded.

Treatment.—**Of the Temperament.**—Accurately speaking, the treatment of hysteria should be begun before birth. Neurotic women bearing children should be subjected to a course of rest-cure and mental and moral suasion, and the condition of their nervous systems should receive the careful attention of the attending physician. Neurotic children require the greatest care during the developmental period. A strong physique must be secured by proper attention to out-of-door exercise, and, for the time being, even at the expense of mental culture. Such children should not be subjected to the "cramming" process so common in our modern courses of education, but should be trained, if possible, at home, where the element of competition may be eliminated. Systematic hours of study and of recreation (with absolute rest from study during the summer months), and opportunities of travel and change of air and scene, will work wonders in these hyperesthetic little individuals. Especially at the time of puberty is the greatest of care required in order to avoid an additional strain upon the already seriously taxed nervous system. In addition to the foregoing a strict watch must be kept over the moral nature of the child. The satisfaction of every whim and the lack of moral suasion are the surest ways to develop the hysteric temperament. When possible the child should be taken away from the enervating influences of city life. The diet should be plain, but nutritious, and all over-indulgence is to be absolutely prohibited. Frequent bathing and friction of the skin are very beneficial, as well as careful regulation of the emunctories generally.

The Hysteric Convulsion.—As hysteric patients almost never injure themselves during a paroxysm, protective measures are not necessary. Indeed, the attack is usually prolonged by attention and observation. Extreme measures to cut short an attack are only justifiable if the friends and relatives become unduly anxious. Cold plunge-bathing, dashing cold water into the face, or the hypodermic injection of apormorphin, thereby producing a profound mental shock, may have a beneficial effect. Pressure over the ovary or upon one of the large vessels (as the carotid) will sometimes promptly induce a termination of the attack.

Internal Treatment.—In this period of the disease it is probable that most can be done to improve the condition of the patient. In addition to the general laws of mental and physical regimen already advanced, the patient should be taught, as far as is possible, the undignified condition into which she is sinking, and advised and encouraged to exert powerful efforts to control her nervous organism. To this end she should also be given full doses of the nerve-sedatives and antispasmodics (valerian, asafetida, sumbul, musk, and camphor), together with the general tonics (iron, arsenic, strychnin). I have repeatedly found the rest-cure of Weir Mitchell especially beneficial at this time; it is fully described under Neurasthenia (*vide* p. 1181).

Hypnotism has commanded considerable attention during this stage of the disease, and it is claimed that under the suggestion of the hypnotist an absolute cure very frequently follows. This is not altogether true, however, for while many patients are undoubtedly benefited by this procedure, the good result must be attributed not alone to the suggestion of the operator, but also to the profound mental effect produced upon the patient by the mysterious process.

In the treatment of the organic manifestations, which, it must be remembered, are dependent entirely upon the general nervous condition, the physician is called upon to exercise the greatest amount of tact. As far as is possible the mind of the patient must be directed away from the affected part. The *irritable bladder* must be treated by internal remedies, as boric or benzoic acid, salol, or the compound infusion of buchu, and not by local irrigation and catheterization.

Hysteric vomiting may not require any special medication. Occasionally, however, it may be relieved by rectal alimentation. Cocain hydrochlorate in the form of a 10 per cent. solution (dose internally), and the application of mild counter-irritation or of a small fly-blister over the epigastrium will be useful. Cannabis indica, acetanilid, phenacetin, and antipyrin, in small doses and only when absolutely needful, will relieve *hysteric neuralgias*, especially the cephalalgia. For the pseudo-angina pectoris, digitalis, strophanthus, caffein, amyl nitrite, or nitroglycerin, or a combination of these drugs in suitable doses, may be exhibited.

For the *pelvic hyperesthesia* of hysteric females local applications (tincture of iodin, croton oil, or a small fly-blister) over the ovarian region may prove very beneficial.

Hysteric palsies, either general or local, and hysteric disturbances of the special senses, must be treated on general principles. As far as is possible the patient's attention must be directed from the affected part or parts, and an occasional local blistering, the use of galvanism and massage, with daily friction, will be of service, especially when they are supplemented by an appropriate course of internal medication.

Electricity is a very valuable adjunct. The static current is most effective, and it may be applied in various forms. Perhaps the most useful of these is the spark, which should be drawn from the anesthetic area or the paralyzed limb, thus producing a profound mental effect.

NEURASTHENIA.

Definition.—Functional exhaustion and irritability of the nerve-centers. Neurasthenia is the expression of an abnormal sensitiveness (irritability) in response to stimuli, and of weakness of the nerve-centers presiding over the organic functions. Several varieties—cerebral, spinal, cardiac, and gastric—have been distinguished, owing to the fact that the predominating features may be manifested by single organs or systems of the body. That the disease is essentially generalized in all instances, however, I do not doubt. It is not a psychosis.

Pathology.—A variable degree of weakness of the sympathetic centers, permitting congestions on trivial provocation, is obvious, but there are no discoverable lesions (coarse) in the nerve-centers that are peculiar to the affection. C. Y. Hodge[1] has invited attention to certain changes in nerve-cells during the active exercise of their function, and something of pathologic importance has been added to our previous knowledge by his observations. There are many causes and associated affections that present a variety of morbid lesions, but they are purely incidental. It should be pointed out here that neurasthenia is often found in association with other functional nervous disorders—a fact that has not only caused mental confusion among certain authors, but has also led to the belief among others that as a distinct affection it does not exist.

Etiology.—The causes are divisible into—1, predisposing; and 2, exciting. Among the former (*a*) *heredity* heads the list. A clear history of nervousness or morbid irritability in one or both parents (oftener the father) is at times obtainable. Ancestors that were sufferers from gout, rheumatism, syphilis, tuberculosis, and chronic alcoholism, all diseases that exhaust vitality, may have transmitted to their offspring a strong neurasthenic disposition. The latter have inherited a small stock of nervous energy with which to begin life's unceasing struggle.

Of other predisposing factors may be mentioned in particular (*b*) improper training, mental and physical, and (*c*) the character of the mental pursuits, those entailing strains being especially deleterious. (*d*) *Age* and *sex* are not without appreciable effect, most cases occurring between the twentieth and fiftieth years, when the work and worry of life are maximal; they are more frequent in men than in women.

Exciting Causes.—According to my own observations, *traumatism* has an active potency, though it is probably not the most frequent cause. *Overwork*, at least in America, is responsible for a greater number of cases than any other single factor, and in estimating its effects the relativity of individual nerve-capital must be carefully considered. Associated causes are to be observed in unpleasurable emotional excitement, mental worriment, particularly if dependent upon love-affairs, and sexual excesses. *Abuse of the sexual organs*, excessive venery, masturbation, and the like are powerful in producing neurasthenia. Finally, as stated under Pathology, the condition may be induced by other functional and organic affections (symptomatic neurasthenia).

Symptoms.—The subjective symptoms are protean and varied, and are usually described with great detail, for the patients are, as a rule, exceedingly voluble. Among the more prominent and numerous features entering into the symptom-complex of neurasthenia are great *irritability*, *physical fatigue* without adequate reason, even to a feeling of utter exhaustion on rising in the morning, *disturbed sleep*, *headache*, with a sense of weight and constriction, *impairment of memory*, *anorexia*, and *constipation;* the patient is very irritable, dispirited, is fearful, and frequently sinks into a state of absolute dejection. Female sufferers—and less frequently males also—may manifest strong emotions, and in such cases the condition presents many points of resemblance to the milder forms of hysteria. The external appearances may be indicative of sound, vigorous health; oftener, however, the physiognomy is worn and anxious.

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[1] *Journal of Morphology*, vol. v. No. 11, p. 95.

The **motor phenomena** include, besides readily oncoming exhaustion of the muscular strength under exercise, a variable condition of the tendon-reactions. On the whole, however, they are increased. Muscular *tremors* (fine) are sometimes present, and particularly when neurasthenia is the result of trauma or fright (Dercum), and spasmodic contractions (usually brief) of small isolated groups of muscular fibers of the face, trunk, or extremities are observed.

The **sensory** disturbances are varied and sometimes striking. The patient makes constant complaint of feeling "tired" or "never rested," and indeed sometimes betakes himself to bed for this reason. A feeling of "lightness," giddiness, and even true vertigo, may occur and recur, and rarely the latter symptom is wellnigh continuous. The *headache* (previously mentioned) is often wholly dependent upon mental work, since it disappears with the cessation of the latter. Another form of *pain* is a dull aching that may be generalized, though more commonly it is confined to the small of the back and limbs. *Spinal tenderness,* when sought for, may often be elicited over certain circumscribed areas or mere points, and it may be combined with a deep-seated ache or an exacerbating pain ("spinal irritation"). Cutaneous *hyperesthesia* is common, but anesthesia is not found in uncomplicated neurasthenia. Numbness, either spontaneous or as the result of slight pressure, is a conspicuous feature for a variable period upon or near to the nerve-trunks, and linked with it there may be a generalized or localized feeling of coolness of the body-surface, or of pricking sensations (formications) and circumscribed subjective sensations of heat and burning.

The *psychic* symptoms grow out of the same fundamental conditions as do the *physical* symptoms—*i. e.* fatigue of the nerve-centers. As would be expected, then, the capacity for sustained mental work is generally lessened, and the power to concentrate or rivet the attention upon any subject as well. The patient is self-centered, sensitive to a degree, easily angered, and is morbidly suspicious. His emotional nature is unstable, and the mental depression (before mentioned) deepens until it approaches true hypochondria.

Insomnia, varying in form, is frequent, and disturbances of the organs of special sense are not wanting. The eye presents the most important fatigue-symptoms. *Vision* may be imperfect (blurred), and continuous close use of the eyes be impossible. There is a lack of power of accommodation and retinal hyperesthesia may supervene. The pupils may be unnaturally large. All forms of *tinnitus* constantly arise in neurasthenia, and may lend so vivid a coloring to the clinical picture that the real nature of the attack is liable to be overlooked. I have recently seen a case of the sort occurring in a clergyman (thirty-six years old), in whom the diagnosis of aural disease had previously been made. This symptom, like all others due to neurasthenia, may, however, be associated with genuine organic diseases of the ear (*otoneurasthenia*). Disturbances of taste sometimes appear, but they are of minor importance. *Vasomotor* disorders, such as hot flushes and profuse sweats, commonly arise in consequence of the diminished tone of the arteries; these form quite distressing fatigue-symptoms. Visible throbbing of the superficial vessels and of the abdominal aorta, and rarely also of the veins and the capillary pulse, occur in the affection (*vide* Aortic Regurgitation, p. 601). The *urinary phenomena* may excite particular attention owing to their prominence, and this remark applies especially to the frequent combination of neurasthenia and lithemia (*lithemic neurasthenia*). Oxaluria and transient glycosuria and albuminuria may also be present. The daily amount of urine is often small, and less frequently it is large. The sexual apparatus is weak and irritable, as shown by frequent seminal emissions (nocturnal) and incomplete erections, and, if the subject be married, by premature ejaculation. The fear of becoming impotent often renders the mental attitude of those really potent such as to excite the keenest compassion. The orgasm in the female and the emission in the male are followed by a sense of prostration and mental depression.

The somatic disturbances referable to the heart (palpitation, precordial pain) have been considered under Neuroses of the Heart, and the various gastro-intestinal features in the discussion of Neuroses of the Stomach. Reference has already been made to several clinical varieties based upon the predominance of special and localized groups of symptoms—*e. g.* when the reigning features are spinal the variety is termed *spinal neurasthenia*; when these are presented by the sexual apparatus, *sexual neurasthenia*; and so on; but I am in entire accord with Dercum when he avers that groups of symptoms cannot be considered as sufficient ground for the division of neurasthenia into separate forms.[1]

Diagnosis.—That cases of neurasthenia are misdiagnosed as other conditions, and the reverse, I feel convinced. An important matter at the outset is to avoid confounding the neurasthenic symptoms (secondary) of various local and general organic diseases with the primary form by a careful exclusion of the latter. From *hysteria* the diagnosis is as follows:

HYSTERIA.	NEURASTHENIA.
By nature a psycho-neurosis.	A neurosis; often with a pronounced psychical element.
Occurs in individuals presenting a marked hereditary taint.	Occurs as the result of nerve-tire, overwork, and the like in individuals not necessarily presenting hereditary taint.
The onset is frequently abrupt.	The onset is always gradual.
The clinical features are dependent upon an excess of nervous energy.	Is characterized by a notable lack or insufficiency of nerve-force.
Presents the characteristic stigmata, as paralysis and anesthesia in most cases.	These are absent.
Is sometimes accompanied by violent convulsive seizures.	Convulsive seizures never occur.
Neuralgic attacks are infrequent or altogether absent.	Neuralgic attacks are very common.
Insomnia is not marked.	Insomnia is very common.

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[1] *Nervous Diseases by American Authors*, p. 73.

Hysteria, it is to be remembered, may be a complication of neurasthenia, and this association must be distinguished from simple hysteria.

Prognosis.—Neurasthenia is a curable disease if appropriate treatment be commenced before secondary structural changes set in and render the use of the most approved measures of no avail. In long-standing cases deleterious habits (morphinism, chloralism, alcoholism) are sometimes developed and prevent the possibility of a cure. Hysteria (the complication) tends to delay, but does not preclude, recovery.

Treatment.—The first step should be, after locating the major cause or causes, to remove them, or, if this be impossible, to minimize their baneful influence so far as may be. For example, if the conditions have been induced by overwork of the brain, rest for the organ must be procured; if sexual excesses have been the obvious responsible factor, rest for the sexual apparatus is imperatively demanded. In the next place, the mental and moral environment must be conducive to contentment and to wholesome forms of exercise of the mind. In this way the exhausted stock of nervous energy can be often increased by the natural recuperative forces alone. Indeed, successful removal of the essential etiologic influences is in the milder forms followed by prompt recovery. In not a few instances the symptoms disappear as the result of a prolonged sojourn in a suitable climate or by travel for a considerable period with its ever-accompanying change of scene, though it is well in doing so to avoid the din and excitement of large cities. The compulsory rest and complete isolation, combined with the purity of atmosphere, afforded by a sea-voyage sometimes work admirable results. Unfortunately, many subjects suffering with neurasthenia, and particularly males, are either unable or unwilling to arrest the loss of nervous function by ceasing their excessive activities. In the majority of instances, for the reasons above stated, certain other measures—hygienic and medicinal—are to be advised.

Next to the importance of removing the exciting agents, stands rest. In severe and long-standing cases this should be made as nearly absolute as possible, while in the milder forms merely lengthening the hours for sleep or rest in bed, as first pointed out by Dr. S. Weir Mitchell, often suffices. The amount of rest must be accurately proportioned to the necessity of individual cases. To Dr. S. Weir Mitchell belongs the credit of having introduced the "rest-cure" in the management of this disease. This mode of treatment in very old, and in profound cases produces curative effects unobtainable in any other manner, though it does not give complete restoration to health, as a rule, and must be variously modified in individual instances. It embraces not only absolute rest, but also "passive exercise" and forced feeding. Both body and mind must receive rest; hence confinement often is not sufficient, and the patient must also be strictly isolated from friends and relatives, particularly if hysteria be associated.

The patient is to be put in charge of a properly selected nurse, who will afford agreeable entertainment by suitable conversation and reading under the instructions of the physician. In desperate cases the patient should not be allowed to feed himself, must not rise to void the urine or feces, nor even turn in bed without the help of the nurse.

The neurasthenic also demands a special *dietary*, that is to be made up at first of milk. This should be administered in small quantities at the beginning (℥iv or v—120.0 or 148.0, every two hours), and slowly and gradually increased until at the end of a week or ten days large quantities are taken (℥viij–x—236.0–300.0 every two hours). When whole milk cannot be readily digested skim-milk should be employed. To the milk, should the patient become decidedly hungry, may be added in the course of five or six days very light nutrients (plain boiled rice, a soft-boiled egg) and a little later meats (lamb-chop, steak). Constipation calls for the use of unbolted bread (graham), fresh and stewed fruits, and butter. This simple dietary is to be enriched until three large meals are taken daily—"such, for instance, as a breakfast of fruit, cracked wheat, one or two soft-boiled eggs, or a good-sized steak, well-served chops, bread and butter, and milk; a dinner of a good slice of roast beef, with vegetables and boiled rice (in place of potatoes). The supper should remain as a light meal of bread, butter, fruit, light pudding, and milk" (Dercum). Tea, coffee, and alcohol should be avoided.

Passive exercise, *massage*, and *electricity* form an essential part of the "rest-cure," though the former should not be commenced until the second or third day. At first it should be continued for a few minutes only, and consists of gentle rubbing or light strokes. As tolerance becomes established massage should be practised for a longer period (about an hour). Deeper rolling, kneading, and spiral manipulations are then allowable. The direction of the venous blood-current—toward the center of the body from the periphery—is to be borne in mind, and all massage-motions are to be made in the same direction. This measure is to be carried out by the nurse, who should be a well-trained masseuse and thoroughly acquainted with the details of her work. Electricity, like massage, compensates for the lack of exercise. The slowly-interrupted faradic current is to be selected, and the aim should always be to induce satisfactory contractions with the least amount of pain. The current should be applied to the individual muscles, one of the extremities being selected, and the poles applied over the motor points, passing from muscle to muscle until all have been faradized. The time of each sitting should not exceed half an hour. The entire body should also receive the faradic current (rapidly interrupted). A large sponge moistened with salt water is applied at the nape of the neck, and another to the soles of the feet, and the strongest current tolerable is thus used. This process should be continued from fifteen to twenty minutes, and, like the faradization of the single muscles, it is to be repeated at intervals of twenty-four hours.

The rest-cure in all of its details should be continued for a period ranging from four to eight weeks. The patient should leave his bed in the most gradual manner, and should sit up for a few minutes only at first, the time being gradually lengthened; soon exercise may be commenced in a like manner and be cautiously increased. During this period of convalescence it is my custom to omit the electric treatment, while the massage is continued at intervals of two or three days for some weeks. After the patient has reached the point of marked improvement, as evidenced by a large appetite, the disappearance of the most pronounced subjective symptoms, and especially by a substantial gain of weight (twenty to twenty-five pounds—11.3 kgms.), he should be advised to make a change of air to the country, or to the mountains or the seashore (if it be not the summer season).

Hydrotherapy is positively and rationally serviceable in the management of neurasthenia. The water may be employed in the form of the shower, spray, bath, or pack, and is most efficacious when quickly applied for a few moments and followed by vigorous towelling to reinforce the action of the cold. A portion of a garden-hose with a sprinkler is readily attached to the water-pipe in the bath-room and furnishes the readiest means of applying cold water. Extreme caution is necessary at the beginning of the application of cold to the surface, since there are neurasthenic subjects that not only fail to receive benefit, but are rendered worse thereby, in consequence of a highly sensitive organization.

Drugs are of minor importance in the treatment, and their routine use is to be condemned. Laxatives are often needful, and in my experience broken doses of calomel, followed by a saline, or the fluid extract of cascara, have proved most effective. For a further consideration of the treatment of the gastro-intestinal symptoms the reader is referred to Neuroses of the Stomach.

Strychnin is constantly being employed in the treatment of this affection, but its use should be limited to the more profound types. Full doses are required if we would expect good results. In cases in which anemia is marked arsenic may be employed with advantage. Phosphorus has also been recommended, but it tends to disturb the digestive organs and rarely gives striking general results.

ACROMEGALY.

(*Giantism.*)

Definition.—A disease first recognized and described by Marie, and characterized by a progressive and peculiar enlargement of the face and extremities.

Pathology.—Those cases that have been examined *postmortem* have shown, as the most constant change, an enlargement of the pituitary body, with a corresponding dilatation of the sella turcica, and a persistence of the thymus gland. Less frequently there is fibroid degeneration of the thyroid gland. A few cases, however, have been reported in which one or all of these organs were normal. The lips, tongue, and trachea are usually considerably enlarged, and the sexual organs may either be hypertrophied or atrophied, the latter condition being more common in the uterus and testicles. The bones of the extremities and face are thickened, apparently chiefly as a result of hyperplasia of the spongy portion, and Klebs has shown that the peripheral vessels, particularly those in the affected bones, are also larger. Occasionally there are hypertrophy of the heart and enlargement of the spleen and liver.

The **etiology** of acromegaly is unknown. Marie believes that it is a form of systematic dystrophy analogous to myxedema, and probably due to interference with the function of the pituitary body. Freund holds that it is a sort of inversion of growth associated with alteration in the sexual organs at puberty. Klebs believes that it is due to a neoplastic condition of the vascular tissues, associated with a functional persistence of the thymus gland. Various diseases have preceded the development of acromegaly, but none with sufficient regularity to indicate that the subsequent appearance of the latter condition was other than accidental. Both sexes are about equally affected, and the disease ordinarily commences in adolescence.

The earliest **symptom** is usually an increase in the thickness of the fingers and toes, so that rings, gloves, and shoes are too small and can no longer be worn. This *enlargement* is chiefly in thickness, although there is also a certain amount of increase in length. Both the soft and hard parts are affected. The nails are flattened, longitudinally ridged, and more friable (*spade-like hand*). The *face* becomes considerably enlarged; the supraorbital ridges project, giving rise to a rather simian aspect; the nose becomes broader and longer; the cheek-bones project; but the most positive characteristic is the enormous enlargement of the lower jaw, so that it becomes broader and prognathous, and the lower teeth can no longer be brought in apposition with the upper. The *spinal column* is ordinarily kyphotic, the change affecting the upper dorsal and cervical regions. Frequently there is also an associated scoliosis. The rest of the skeleton remains unaffected for a long time; finally, changes may be observed in the clavicles, sternum, ribs, pelvis, and particularly in the patellæ. The *skin* sometimes shows slight pigmentation; the hair is rough and may become thinner; the *muscles* occasionally exhibit increased electric excitability, and less frequently there is muscular atrophy with reactions of degeneration. The lips, tongue, and tonsils are usually enlarged, and the larynx is increased in dimensions, so that the *voice* becomes deep and rough; this is a very characteristic symptom in women. Ordinarily, an area of dulness can be detected in the upper part of the sternum that has been ascribed to the persistence of the thymus gland. The *tendon-reflexes* may either be normal, diminished, or abolished. They are never exaggerated. The *urine* is increased in amount, and glycosuria is often present. The secretion of *sweat* is also greatly increased. The subjective symptoms consist of severe intermittent or continuous *headache* and of a *diminution of the visual power*. There may be paresis of the third nerve, giving rise to external strabismus, and sometimes to *temporal hemianopsia* as a result of pressure upon the central part of the chiasm by an enlarged pituitary body. Sometimes late in the disease there are occasional momentary general tremors. The patients often present polyphagia and polydipsia. Neuro-retinitis and subsequent atrophy of the optic nerve may also occur. Sexual power is usually abolished. In women menstruation ceases early in the disease, and the breasts atrophy. The mental condition is affected, and there are usually great apathy and diffidénce (perhaps explicable by their changed appearance), loss of memory, and even, in some cases, imbecility.

Diagnosis.—In the later stages the appearance is characteristic, and acromegaly can then hardly be confounded with other diseases. The peculiar enlargement of the extremities, the oval, prognathous, and distorted face, the deep, rough voice, the more or less pronounced pigmentation of the skin, the wasting of the muscles, and the profound cachexia give a perfect clinical picture. In those cases in which the cachexia has become extreme there are from time to time peculiar tremors or spasms of the body.

Differential Diagnosis.—In the earlier stages the disease is most easily confounded with the *hypertrophic pulmonary osteo-arthropathy of Marie.* In this both hands and feet are greatly enlarged; but the fingers are club-shaped, the face is not involved, and there usually exists some chronic pulmonary complication. In a case that I observed there were bronchiectasis and bronchorrhea. From *osteitis deformans* it may be distinguished by the fact that in this condition chiefly the long bones of the limbs and the flat bones of the skull are hypertrophied and very painful. *Elephantiasis* may be distinguished by the fact that it attacks the lower limbs, does not involve the bones, and the skin presents a granular or a nodular appearance. From *arthritis deformans* acromegaly may be distinguished by the fact that the disease develops late in life and is associated with great deformity of the joints, the face ordinarily escaping. The following table (after Dercum) will serve to distinguish two diseases that are apt to be confounded with one another:

ACROMEGALY.	MYXEDEMA.
Occurs most commonly in early adult life.	A disease of mature life—forty to fifty years.
In males and females equally.	Five times as frequent in females as in males.
Enlargement of the bones characteristic.	No enlargement of the bones.
Marked prognathism of jaw and flattening of cheeks.	Face full-moon-shaped.
Skin brownish-yellow; hair coarse and unwieldy; nails short and striated.	Skin pale, waxy, shiny, and boggy; hair falls out; nails not affected.
Fingers symmetric and sausage-shaped.	Fingers clubbed at the end.
Administration of thyroid extract is of the smallest benefit.	Thyroid treatment of the greatest benefit.

The **prognosis** is hopeless for cure and doubtful for duration. The disease is progressive, although it remains stationary for a longer or shorter period. Retrogression never occurs. Ordinarily, the patients die of some intercurrent condition; although death may be due to the cachexia of acromegaly itself. Life, however, may last for twenty years after the appearance of the first symptoms.

Treatment of the condition itself has proved unavailing. Certain cases have been reported in which there was slight temporary improvement after the use of extract of pituitary body or of thyroid gland, but the results are contradictory. The cephalalgia can be more or less completely controlled by antipyrin or caffein. Phosphorus, mercury, the iodids, and arsenic have been wholly useless.

ASTASIA-ABASIA.

ASTASIA-ABASIA is rather a symptom than a disease *sui generis.* It consists of an interference with the power of walking, although the limbs show no trace of paralysis and are capable of performing perfectly other complicated movements. In this respect it is somewhat allied to the functional neuroses. In a case reported by Burr, complicated with severe anemia, changes were found in the posterior and lateral columns of the cord; ordinarily, however, no gross lesions can be discovered. The dis-

V. VASOMOTOR AND TROPHIC DISORDERS.

ANGIONEUROTIC EDEMA.

(*Acute Circumscribed Edema of the Skin; Intermittent Angioneurotic Edema.*)

Definition.—A disease characterized by the appearance of an edematous swelling of the skin or mucous membranes. In general it is not accompanied by constitutional symptoms.

The **pathology** of the disease is obscure. It is supposed to be due either to venous stasis or to some nervous influence upon the lymph-channels, causing them to exude liquid. No lesions have as yet been described.

Etiology.—Neuropathic heredity appears to have some influence upon the disease, but nervous manifestations in the patient himself are more important. Occasionally the condition follows infectious diseases or severe hemorrhage. The most important exciting causes are cold and emotional disturbances. The disease occurs most frequently in males, and almost exclusively in early adult life.

Symptoms.—The *edema* usually appears suddenly, is sharply circumscribed, and the skin of the affected area is slightly elevated and reddened, or else somewhat paler than the surrounding tissue. Ordinarily, subjective symptoms are absent; occasionally there are slight *paresthesia*. The edema may appear in any part of the body, but usually it is most common on the backs of the hands or legs and in the face, especially the eyelid. Occasionally it may appear upon the mucous membranes either of the lips, tongue, or glottis; in the latter situation it sometimes produces severe dyspnea, and at least in one case it has caused death. Its presence has also been suspected in the mucous membrane of the gastro-intestinal tract. Ordinarily the patient has no symptoms whatever of disease; occasionally, however, there are severe colic and sometimes vomiting. In one case hematuria was observed, and in another hemorrhage from the swollen gums; of course in the latter case the diagnosis was doubtful. The patient may exhibit a certain degree of anxiety during the attack. Ordinarily the swelling persists a few days, and then disappears, but relapses are exceedingly common, and may recur very frequently for many years.

The **differential diagnosis** has to be made from urticaria, to which it bears a great similarity. According to Osler, giant urticaria is the same disease.

The **prognosis** is of course favorable for life; for cure it is more doubtful, as the disease is sometimes exceedingly obstinate.

The **treatment** consists of rest, the use of tonics particularly directed to the nervous system, and the correction of any gastro-intestinal disorder. Quinin has occasionally proved very valuable. Hypnotism has also been suggested.

are suddenly liberated, with the formation of air-emboli; others have believed that the changes are due to a sudden disturbance of the gaseous metabolism. In cases in which death has occurred after a considerable interval the lesions of disseminated focal myelitis have been discovered.

The **etiology** of the disease is very clear, and certain predisposing factors are worthy of note. Divers are more apt to suffer if they have been working at extreme depths, particularly if the period of exposure to great pressure has been prolonged; even moderate pressure will sometimes produce symptoms if continued for a sufficient length of time, and short periods of rest do not prevent the development of the disease. Ordinarily, it can be said that unless the pressure exceeds two and one half or three atmospheres no danger may be apprehended.

The **symptoms** vary greatly in intensity. In the mildest form they consist of *neuralgic pains in the joints*, sometimes with slight articular swelling, *headache*, *giddiness*, and a little *tinnitus*. These pains may become more violent, particularly in the loins, and be followed by a gradual *loss of power* and by *anesthesia* in the limbs; these symptoms may disappear in a few hours or become more severe, with the development of *complete paralysis* and interference with the action of the sphincters. This paralysis usually assumes the form of *paraplegia; monoplegia* and *hemiplegia* also occur, and sometimes there are complete paralysis and anesthesia of all four extremities and of the trunk. In the most severe cases *cerebral symptoms* are also present, consisting of sudden loss of consciousness, profound coma, irregular respiratory action, and finally, after a short time, death from cardiac failure.

The **diagnosis** is very easy. It is possible, however, that an attack of *apoplexy* should occur in a man who has been under water, and the patient should always be examined for the presence of this or some other organic lesion.

The **prognosis** varies with the intensity of the symptoms. The lighter forms consist merely of joint-pains and slight dizziness that usually pass away in the course of a few hours. Paraplegias or hemiplegias, developing slowly and not assuming a severe form, are also transient in character. A more severe paraplegia is usually permanent, although some improvement may be expected. The apoplectic forms are almost invariably fatal in the course of a few hours.

The **treatment** consists, firstly, of prophylactic measures. In all places where caisson-work is carried on one or more locks should be provided in which the pressure can be gradually reduced until it is approximately that of the atmosphere. Divers should be instructed to come slowly to the surface. If the pressure exceeds three atmospheres, the maximum length of the working-period should not be more than one hour, and several hours should be permitted between the descents. A chamber should also be provided in which a man who exhibits symptoms of the disease can be once more subjected to a pressure greater than that of the atmosphere, as this usually causes an arrest of the process. When, however, the condition resembles that of acute myelitis, the treatment is purely symptomatic. It consists of rest, careful hygiene, and a stimulating diet. Potassium iodid may also be administered in the later stages, but its value is very doubtful. In some of the acute forms with more or less respiratory failure inhalations of oxygen have been recommended.

posed to be purely hysteric in nature. The most important exciting cause is exposure to cold, although attacks may also be brought on by severe emotional disturbances.

Symptoms.—The disease presents three grades of severity: first, anemia or local syncope; second, cyanosis or local asphyxia; and third, gangrene. *Local syncope* consists in a vasomotor spasm in one or more extremities, the fingers being most frequently affected, and rarely more than one at a time. They become white, almost waxy in appearance, cold, and hard to the touch, and they may be either dry or covered with a cold perspiration. The finger is perfectly numb, but severe neuralgic pains may be felt in the arm; if the skin be pricked with a pin, no blood flows. Ordinarily this syncope disappears gradually, the reaction being accompanied by tingling and formication in the affected digit, which ultimately returns to a normal condition. *Local asphyxia* is a further stage of this condition: in this the finger is blue and swollen, and there is a sense of discomfort that is apparently due to the stretching produced by the engorged veins. This cyanotic condition may also affect the ears, toes, and the tip of the nose, and, like the preceding stage, it may disappear without leaving any trace of its existence. Patients that have reached this stage seem to be more liable to a recurrence upon slight exposure than those who only present local syncope. The attacks are more likely to recur constantly in the same digit, and not to appear first in one and then in another. During the existence of this stage a not infrequent associated symptom is *hemoglobinuria;* this is especially apt to occur in children, and has led to the suspicion of malarial influence. In some cases, when hemoglobinuria is not found, the urine contains an excess of urates. If the attack lasts for several days, *trophic changes* take place in the finger-nail, giving rise to a transverse ridge, which persists until that portion of the nail has grown beyond the end of the finger. If local cyanosis, however, continues sufficiently long, *gangrenous* changes take place. These appear first as small black spots or vesicles filled with serum upon the end of the fingers or about the root of the nail; these gradually slough off, leaving a small ulcer that may slowly cicatrize. Often patients subject to recurrences of the disease show a number of cicatrices on the ends of the fingers, or if the ears are affected there may be slight shrivelling of their edges. The gangrene, however, may be more severe, in which case the distal phalanges of the affected fingers may become black or dark red, covered with blebs, and finally mummified. The line of demarkation then forms, and ultimately the gangrenous portion falls off, leaving an ulcerated stump that slowly cicatrizes. This form may not be limited exclusively to the hands and feet or ears, but symmetric patches sometimes appear in the skin of the breast. During the time that the gangrene is present the patients suffer from excruciating *pains* in the limbs that interfere with sleep, often causing transient melancholia, and seeming, more than the gangrene itself, to depress the general condition. Fever is rarely present; sugar is sometimes found in the urine, but not constantly.

The **diagnosis** is necessarily difficult when it is remembered that all clinicians do not agree that such a disease exists. According to Raynaud's definition, it is a neurosis characterized by enormous exaggeration of the excito-motor energy of the gray parts of the spinal cord that control the

HYDROPS ARTICULORUM INTERMITTENS.

Definition.—A condition characterized by periodic effusions into one or more of the large joints, and usually the knee.

The **pathology** is unknown, but it is suspected that it depends upon some nervous disturbances of the vessels or lymph-channels in the joints.

The **etiology** is also doubtful. The disease occurs in nervous individuals, and has been found associated with other nervous diseases, as exophthalmic goiter, or in patients suffering from other vascular diseases, as angina pectoris.

The **symptoms** consist of the sudden development of a swelling in the affected joint, which, however, does not present any symptoms of inflammation and is rarely painful. This swelling lasts from three to eight days and then disappears as suddenly as it came. At regular intervals of from one to four weeks it is repeated, and this repetition may continue for years.

The **diagnosis** must be made upon the symptoms and the periodicity of the condition.

The **prognosis** is doubtful, most cases being exceedingly obstinate.

Treatment.—Electro-therapy in various forms has been recommended. Among the drugs that have been suggested are salicylic acid, quinin, arsenic, and ergotin. None, however, has proved particularly valuable.

RAYNAUD'S DISEASE.

(*Symmetric Gangrene.*)

Definition.—A condition apparently of vasomotor nature, affecting symmetric parts of the body, and chiefly the tips of the extremities.

The **pathology** is by no means definitely made out. It is supposed that alterations must occur in the vasomotor centers of the medulla and cord, but none have as yet been found, partly because the disease is rarely fatal. In some cases peripheral neuritis has been observed and in others peripheral endarteritis. The gangrene is usually superficial, and resembles closely that caused by cold; rarely it causes an extended loss of substance.

The **etiology** of the condition is obscure and complex, largely, no doubt, because a number of different conditions have been confounded under this designation. The disease occurs in children and in neurotic women, less often in men. A neuropathic heredity seems to predispose to it, and occasionally it exists in connection with other nervous diseases, as epilepsy, migrain, hysteria, and mental disorders. The occurrence of paroxysmal hemoglobinuria has led to the suspicion that malaria is an etiologic factor. I am not aware, however, that plasmodia have been found in any case, and the asserted good results following the administration of quinin are insufficient to establish the contention. Syphilis and various other infectious diseases have also been mentioned as etiologic factors, and more recently a form has been described that is supposed to be purely hysteric in nature. The most important exciting cause is exposure to cold, although attacks may also be brought on by severe emotional disturbances.

Symptoms.—The disease presents three grades of severity: first, anemia or local syncope; second, cyanosis or local asphyxia; and third, gangrene. *Local syncope* consists in a vasomotor spasm in one or more extremities, the fingers being most frequently affected, and rarely more than one at a time. They become white, almost waxy in appearance, cold, and hard to the touch, and they may be either dry or covered with a cold perspiration. The finger is perfectly numb, but severe neuralgic pains may be felt in the arm; if the skin be pricked with a pin, no blood flows. Ordinarily this syncope disappears gradually, the reaction being accompanied by tingling and formication in the affected digit, which ultimately returns to a normal condition. *Local asphyxia* is a further stage of this condition: in this the finger is blue and swollen, and there is a sense of discomfort that is apparently due to the stretching produced by the engorged veins. This cyanotic condition may also affect the ears, toes, and the tip of the nose, and, like the preceding stage, it may disappear without leaving any trace of its existence. Patients that have reached this stage seem to be more liable to a recurrence upon slight exposure than those who only present local syncope. The attacks are more likely to recur constantly in the same digit, and not to appear first in one and then in another. During the existence of this stage a not infrequent associated symptom is *hemoglobinuria;* this is especially apt to occur in children, and has led to the suspicion of malarial influence. In some cases, when hemoglobinuria is not found, the urine contains an excess of urates. If the attack lasts for several days, *trophic changes* take place in the finger-nail, giving rise to a transverse ridge, which persists until that portion of the nail has grown beyond the end of the finger. If local cyanosis, however, continues sufficiently long, *gangrenous* changes take place. These appear first as small black spots or vesicles filled with serum upon the end of the fingers or about the root of the nail; these gradually slough off, leaving a small ulcer that may slowly cicatrize. Often patients subject to recurrences of the disease show a number of cicatrices on the ends of the fingers, or if the ears are affected there may be slight shrivelling of their edges. The gangrene, however, may be more severe, in which case the distal phalanges of the affected fingers may become black or dark red, covered with blebs, and finally mummified. The line of demarkation then forms, and ultimately the gangrenous portion falls off, leaving an ulcerated stump that slowly cicatrizes. This form may not be limited exclusively to the hands and feet or ears, but symmetric patches sometimes appear in the skin of the breast. During the time that the gangrene is present the patients suffer from excruciating *pains* in the limbs that interfere with sleep, often causing transient melancholia, and seeming, more than the gangrene itself, to depress the general condition. Fever is rarely present; sugar is sometimes found in the urine, but not constantly.

The **diagnosis** is necessarily difficult when it is remembered that all clinicians do not agree that such a disease exists. According to Raynaud's definition, it is a neurosis characterized by enormous exaggeration of the excito-motor energy of the gray parts of the spinal cord that control the vasomotor innervation. If this be accepted, then all cases in which lesions of the nerves and arteries have been found are not properly of the same nature; and there are a number of other conditions that may produce gangrene, often somewhat symmetric in type and perhaps due to vasomotor spasms (particularly *syringomyelia* and *leprosy*). The so-called "dead finger" is a common symptom, and its occurrence is by no means sufficient justification for a diagnosis of Raynaud's disease. In fact, the typical forms of this condition should advance to slight superficial and symmetrically placed patches of gangrene. Gangrene may also occur in *diabetes*, and hence the urine should always be examined to exclude this condition. *Hysteric gangrene* is rarely symmetric, and the patients present various hysteric stigmata.

The **prognosis** is very favorable. Only in marasmic children do the attacks ever lead to death. Ordinarily they become in time less frequent and ultimately disappear, but in a few cases the tendency to recurrence is obstinate.

The **treatment** consists of improvement in the general condition during the intervals. During the attack the most effective measures are a mild massage, the use of local lukewarm baths, and electricity very cautiously applied, either in a constant descending stream to the spinal column or by the application of the anode to the spine and of the cathode to a vessel containing water into which the hand has been plunged. Amyl nitrite, which might be expected to relax the vasomotor spasm, fails to have any effect; on the other hand, pilocarpin has been employed with good results. If the pains are very severe, they must be combated by morphin—although gangrene may occur at the site of the injection—administered hypodermically, if necessary. Sleep should be obtained by means of narcotics. The gangrenous parts should always be carefully protected by a local dressing.

PROGRESSIVE HEMIATROPHY OF THE FACE.

(*Progressive Facial Atrophy.*)

Definition.—A rare disease, characterized, as its name would indicate, by a progressive atrophy of one-half of the face, stopping sharply at the middle line, and in the severer forms involving the skin, muscles, and bones.

The **pathology** of the condition is unknown. Rarely symptoms indicating inflammation of the cervical sympathetic, such as dilatation of the pupil or flushing, have been present, and symptoms indicating inflammation of the trigeminus have been equally infrequent. Mendel, however, has reported a case in which he found chronic interstitial neuritis of the branches of the trifacial, and other cases have been reported in which the Gasserian ganglion was diseased. Microscopic examination has shown a disappearance of the subcutaneous fatty tissue and a general atrophy of the elements of the skin itself, often associated with the presence of an abnormal quantity of pigment. As a rule, the vessels are relatively enlarged.

The **etiology** is unknown. The condition usually commences early in life and shows no predilection for either sex. An hereditary tendency does not appear to exist, but the disease occurs frequently as a complication of, or rather in connection with, other neurotic conditions. Of these the most frequent are neuralgia, migrain, epilepsy, and mental disorders; less frequently, tic convulsif and chorea, particularly if the latter affects the muscles of the jaw and tongue. Occasionally it has been recorded as occurring in patients suffering from locomotor ataxia or multiple sclerosis. It does not appear, however, that progressive facial atrophy has any anatomic connection with these conditions. In a few cases the disease has been preceded by an injury to the skull or face, and in others it has followed an acute infectious disease. Ordinarily it occurs in early life—*i. e.* between the tenth and fifteenth years—and in these cases it usually progresses to the most severe type.

The earliest **symptom** is a flattening of the skin on the affected side, constituting the lightest form of the disease, which may remain stationary at this point; if, however, it progresses, the muscles and bones also become involved, so that the affected half of the face is distinctly smaller than the healthy side. The objective changes that take place in the skin are the development of *white spots* in which the pigment has disappeared. and which have the appearance almost of scar-tissue, or, what is more commonly the case, of an increase in *pigmentation* with a formation of yellowish or brownish blotches, the skin being depressed in these areas, which usually lie along the course of the nerve-trunks, especially the infraorbital. The *hair* becomes thinner, dryer, and often falls out. The secretion of the sebaceous glands is diminished and the skin dryer. Rarer phenomena are the *disturbance of blushing*, so that the affected side of the face remains unchanged in color when, as a result of some emotional disturbance, the other is distinctly reddened. Disturbances of sensation are not common. In some cases electric and tactile sensibility have been diminished; in others the patients have complained of slight paresthesiæ. The special senses remain unaffected, and even when the atrophy extends to the tongue, taste remains perfect on the affected side. In one case there were a slight disturbance of hearing and occasional tinnitus.

The **diagnosis** of the condition is easy both when it is suspected and when it is far advanced. The only condition with which it could be confounded is congenital facial asymmetry. In facial hemiatrophy, however, the skin is shrunken and wrinkled, and the hair is dryer and thinner, contrasting markedly with the healthy side, and there is usually a history of commencement some years after birth. In congenital asymmetry the difference between the two sides is slight, and the skin over the smaller side is normal in every respect. Commonly in this condition we also find differences in the development of the extremities. In a case that I recently observed with marked facial asymmetry, the left side being smaller, the hand and foot on the same side were distinctly smaller than the corresponding members.

The **prognosis** is unfavorable as regards cure. The disease itself is not in the least dangerous, and cases have been recorded that have been under observation for thirty years or more.

Treatment is unsatisfactory. The prolonged use of electricity has been said to arrest the process, and sometimes this arrest occurs spontaneously; it is not certain that the treatment is of any use.

An allied condition is **hemihypertrophy of the face.** This is an exceedingly rare condition, and is apparently always congenital. It involves chiefly the soft parts, the ear, skin, tongue, and tonsils being all enlarged. There is an increased secretion from the sebaceous glands, which may appear as small elevations upon the skin. Usually, as in congenital asymmetry, there is enlargement of the extremities on the same side. The only case that has come to autopsy presented no lesions.

Treatment is of course unavailing.

SCLERODERMA DIFFUSUM.

Definition.—A peculiar hardening of the skin, with areas of pigmentation and depigmentation, associated in the more advanced stages with trophic lesions, muscular atrophies, and affections of the bones.

Pathology.—The affected skin is characterized by an increase of the connective tissue and of the elastic fibers, and by a narrowing of the vessels as a result of perivascular infiltration.

The **etiology** is not clear. Some of the cases are associated with joint-affections that resemble those of chronic rheumatism; others follow exposure to a very low temperature. The presence of trophic lesions in the skin and the development of myopathies lead to the supposition that it is properly classed with the trophic neuroses. The disease usually occurs in middle life, although cases have been observed among children. Women are more frequently affected than men.

Symptoms.—Three stages are recognized: *First*, a rather dense edema. *Second*, a true sclerosis, in which the skin appears thicker, with an absence of the normal folds; it becomes firm and hard, so that it cannot be pinched between the fingers and lifted from the flesh. Moreover, there are always *pigmentary changes*, certain parts being darker than normal, while others become a dead white, appearing almost as if composed of alabaster. The disease, as a rule, attacks first the upper portion of the body—*i. e.* the face, neck, hands, and arms, or the surface of the thorax, and is most pronounced in those regions where the bones are subcutaneous. The *diminished elasticity* considerably interferes with the movements of the body. If the neck is affected, it is difficult to turn the head; if the skin over the joints is involved, their normal flexion and extension cannot be perfectly performed. The subjective sensations are those of tension, the patient complaining that the skin has become "too small" for him. If any forcible action is attempted, there is severe pain, accompanied by slight tears in the skin. The skin is paler and cooler than normal, and the slightest exposure to cold causes great discomfort and cyanosis. The secretion of sweat may be normal, but is usually diminished. Tactile sensibility is unimpaired. The *third stage* is that of atrophy; the skin becomes thin as paper; the other symptoms, however, remain as before, except that the secretion of sweat is abolished and

Treatment.—This should always be tonic, and employed during the intervals; massage, hot and cold douches, and the faradic current may be used upon the affected extremities. The pain may call for anodynes.

ACROPARESTHESIA.

(*Spastic Vasomotor Neurosis of the Extremities.*)

Definition.—A disease characterized by abnormal sensations in the hands, slight vasomotor disturbances, and slight stiffness of the fingers.

The **pathology** and **etiology** are not understood. Possibly the condition is due to some disturbance of the peripheral nervous system. It occasionally occurs after injury or as a result of prolonged exposure to cold, hence is common among laundresses. It is more frequent among women than men, and usually develops in middle life.

The **symptoms** consist in the more or less sudden development of *formication* and *tingling* or *numbness* in the fingers and finger-tips, usually bilateral, but sometimes occurring only on one side. Less frequently the toes are affected. These pains are more severe in the night and early morning, and worse in summer or after exposure to heat. The vasomotor disturbances are variable. Sometimes nothing can be observed, and sometimes the extremities are bluish and cold, sometimes pink and warm. Sensibility is rarely affected. In some cases, however, there is considerable hyperesthesia; in others moderate anesthesia. In a few cases there is stiffness of the hands. Slight *trophic disturbances* have been reported in a few cases. The attacks may last from a few minutes to several hours, and may recur frequently or only at considerable intervals. Usually during the attack the abnormal sensations are continuous, but occasionally they are intermittent in character. The condition known as *tender toes*, that occasionally occurs after an attack of typhoid fever, is probably a form of this disease. It is ascribed to the Brand treatment, but incorrectly.

The **diagnosis** is usually easy. Care should be taken, however, not to confuse these acroparesthesiæ with commencing locomotor ataxia, tetany, or hysteria. In Raynaud's disease cold increases the intensity of the symptoms.

The **prognosis** is, in general, favorable, the disease usually disappearing after some months; sometimes, however, the condition is obstinate.

The **treatment** is rather unsatisfactory. Laundresses should be advised to adopt some other vocation. Local stimulation with the faradic brush has sometimes been of value, and hydrotherapy may also be employed. At the same time, the patient should be given tonics, particularly if anemia is present. Alkaline washes are almost a specific for the tender toes. Saturated solutions of sodium bicarbonate should be employed.

it is one of the manifestations of leprosy or not. At any rate, it does not appear that typical lepra bacilli have been found.

Etiology.—The disease may occur in childhood or early adult life, and is most common in negroes. It occurs almost exclusively in tropical regions—*e. g.* Brazil and Syria.

The **symptoms** of the condition consist in the formation of a *furrow* at the base of the little toe of one of the feet. This grows deeper and deeper until spontaneous amputation has occurred. Rarely the other toes on the same foot become progressively involved. Certain *vasomotor disturbances* may be observed; the foot is usually swollen, bluish-red, and cold; sometimes the other foot may exhibit similar changes without the formation of furrows at the base of the toes. There is some *diminution of sensation* to touch, temperature, and electricity, and ordinarily the patient complains of vague pains in the limbs.

The **diagnosis** is to be made from leprosy, with which, indeed, it may be identical, and congenital amputation: the latter only occasions difficulty when the disease commences in early life.

The **prognosis** is favorable to life, but the disease is usually slowly progressive.

No effective **treatment** has been discovered, but the parts should be protected against injury, and the patients may be given tonics and anodynes as required.

ERYTHROMELALGIA (*Weir Mitchell*).

(*Paralytic Vaso-motor Neurosis of the Extremities.*)

Definition.—A disease characterized by paresthesia, redness of the skin, and by pain, usually in the toes and heels, associated with more or less severe general disturbances.

The **pathology** is unknown, but the disease appears to be due to some disturbance of the vasomotor centers or nerves.

Etiology.—The cause seems to be exposure to cold, but a nervous temperament or a previous attack of rheumatism appears to have some predisposing action. Men are more frequently affected, and the disease usually develops in early adult life.

Symptoms.—The earliest symptom, as a rule, is the occurrence of severe *pains in the feet.* Objectively, there are swelling and reddening of the skin, and the sensitiveness is so severe that the patient is unable to walk. The general symptoms consist of headache, dizziness, palpitation of the heart, or even fainting. The attacks occur more frequently during the summer months, and are always aggravated by exposure to heat or a vertical position of the limbs.

The **diagnosis** is easy, the condition being really a symptom rather than a disease. It may occur in the course of hemiplegia and in some organic diseases of the spine, and these should be excluded.

The **prognosis** is favorable; often, however, the disease will recur at irregular periods for a number of years. The attack can usually be cut short by plunging the limb into ice-cold water.

Treatment.—This should always be tonic, and employed during the intervals; massage, hot and cold douches, and the faradic current may be used upon the affected extremities. The pain may call for anodynes.

ACROPARESTHESIA.

(*Spastic Vasomotor Neurosis of the Extremities.*)

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The **pathology** and **etiology** are not understood. Possibly the condition is due to some disturbance of the peripheral nervous system. It occasionally occurs after injury or as a result of prolonged exposure to cold, hence is common among laundresses. It is more frequent among women than men, and usually develops in middle life.

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The **treatment** is rather unsatisfactory. Laundresses should be advised to adopt some other vocation. Local stimulation with the faradic brush has sometimes been of value, and hydrotherapy may also be employed. At the same time, the patient should be given tonics, particularly if anemia is present. Alkaline washes are almost a specific for the tender toes. Saturated solutions of sodium bicarbonate should be employed.

MERALGIA PARÆSTHETICA.

(*Bernhardt's Disturbance of Sensation.*)

Definition.—A disease characterized by paresthesia and disturbance of sensation on the outer side of the thigh, in the region supplied by the external cutaneous femoral nerve.

Pathology.—Nawretsky has examined one case, and found chronic interstitial neuritis. There is reason to believe that this is not always present.

Etiology.—This is very various; some of the cases have been preceded by injury, excessive exercise, or infectious disease. Alcoholism, constipation, and pregnancy are also common predisposing causes; cold douches have been blamed in several instances. The exposed situation of the nerve is supposed to render it more liable to this peculiar disturbance.

Symptoms.—These are of two varieties: First, the *paresthesiæ.* There may be burning, tingling, or stabbing pains that are severe enough to disable the patient; or there may be only a feeling of cold or numbness. Second, the *sensory disturbances.* These vary from slight hyperesthesia to total anesthesia. The different senses are not always equally involved; pain, temperature, and electro-cutaneous sensibility being usually more profoundly affected than the others. Frequently both thighs are affected. There is often a tender point just inside the anterior superior spine of the ilium.

The **diagnosis** is easy.

The **prognosis** is doubtful. Some of the cases recover rapidly, but the majority become chronic.

Treatment.—But little can be done. Locally, the dry brush seems to do good in some cases, and the general health should be improved if possible. In aggravated cases a portion of the nerve may be excised.

PART IX.

DISEASES OF THE MUSCLES.

MYOSITIS.

RHEUMATIC myositis and the suppurative form observed in pyemia, and rarely in other acute infectious diseases, have been appropriately described in connection with the diseases to which they are secondary manifestations. There remain to be discussed two rare forms of the disorder.

INFECTIOUS MYOSITIS.

(*Acute Polymyositis*).

Definition.—A primary acute or a subacute inflammation of the voluntary muscles due to an unknown microbic agent.

Pathology.—The disease is a true inflammation of all the voluntary muscles, involving chiefly the muscular fibers, and to some extent, also, the interstitial connective tissue. Beginning with marked hyperemia, there next occurs an exudation of leukocytes. The muscles are firm, fragile, and later undergo fatty degeneration. Serous infiltration occurs and there is a slight hyperplasia of the intermuscular connective tissues. Hueppe records a case that showed nothing definite beyond a hyaline degeneration of the muscular fasciculi.

Etiology.—We are no less ignorant of the predisposing influences than of the specific exciting agency, though, perhaps, young males are most often the victims of this malady.

Symptoms.—As a rule, first the muscles of the extremities, and later of the trunk also, become swollen, firmer than normally, and stiff, rendering locomotion somewhat difficult and painful.

The involved parts may also be tender to the pressing finger, and a slight edema may be noticed that is at first more or less localized, but finally becomes generalized, and extends even to the face. An erythematous eruption then appears, which is irregularly disseminated over the skin-surface, and may tend to more or less pigmentation. Moderate pyrexia and splenic enlargement are among the early and constant symptoms. In the advanced stage the muscles of deglutition and of respiration become involved, rendering the act of swallowing difficult, and inducing marked dyspnea.

Among the *complications* may be enumerated bronchitis and bronchopneumonia, the latter often being a terminal condition.

Diagnosis.—Taken in the aggregate, the symptoms are of little diagnostic importance and the previous history is invariably negative.

Trichiniasis must be discriminated, since this disease produces an identical clinical picture. The distinction may rest upon the examination of an excised piece of affected muscle, which will not only discover the trichinæ, if present, but also enable the microscopist to detect the positive evidences of polymyositis. In a recent supposititious case of infectious myositis of my own, a portion of muscle, examined for me by Dr. Babcock, showed neither trichinæ nor the histioid changes of myositis. *Multiple neuritis* presents neither swelling nor edema.

Course and Prognosis.—The course of the disease may either be comparatively rapid (two or three months), or it may be slow (chronic) and continue over two or three years. It usually terminates in death, which is caused, in the immense majority of cases, by paralysis of respiration. Occasionally, since the heart-muscle has been rarely found to be implicated, the end may be preceded by cardiac failure.

The **treatment** is simply palliative and supportive.

PROGRESSIVE OSSIFYING MYOSITIS.

Definition.—Myositis, either general or local, in which the affected muscles undergo progressive ossification.

Pathology.—Following the changes that ordinarily characterize myositis (swelling, leukocytic exudation, etc.), a calcification that is often complete takes place. The process may extend to and involve the heart.

The **etiology** is obscure, though males are especially the subjects of the complaint, which usually begins about the time of puberty.

Diagnosis.—The muscles are represented by plates of bony hardness, leading to more or less complete ankylosis of the joints and vertebræ.

The **course** of myositis ossificans is very slow, and **treatment** has afforded only negative results.

PROGRESSIVE SPINAL MUSCULAR ATROPHY.

(*Amyotrophia Spinalis Progressiva; Type of Duchenne-Aran.*)

Definition.—A disease of the peripheral motor neurons and the muscles they supply, usually beginning in the cervical region.

Pathology.—There is atrophy of the anterior cornua of the cord, affecting chiefly the ganglion-cells, degeneration of the nerve-fibers and of the muscles. Occasionally there are small areas of sclerosis that may involve the pyramidal columns for a short distance.

Etiology.—The disease appears to be hereditary in a few cases, and in these may develop in childhood. A commonly accepted predisposing cause is prolonged severe muscular exertion. It is most common in males, and most frequently appears during the third decade of life.

Symptomatology.—The first changes usually appear in the *thenar and hypothenar eminences* of the hands. These become flat and soft; there are loss of power, some stiffness, and inability to perform delicate coördinated movements; the thumb assumes a position parallel to the other fingers (*ape-hand*); the interossei muscles waste and grooves appear between the metacarpal bones. The degenerative changes do not ascend by continuity; the deltoid usually being affected immediately after the muscles of the hand. If the two hands have not been affected simultaneously, the other now begins to show characteristic changes. In the lower limbs the quadriceps femoris is usually the first muscle attacked. The disease gradually involves one group of muscles after another until a large part of the muscular system is affected. All the muscles exhibit the fibrillary twitchings, the reactions of degeneration, and the wasting. Hypertrophy never occurs, and the *paralysis* is nearly always flaccid. The fibrillary twitchings are characteristic, but not pathognomonic. They are not constant, but may be developed by slightly irritating the muscle. There is usually quantitative diminution to the faradic, and qualitative alteration to the galvanic current. The *diplegic reaction* consists of the development of contractures in the opposite arm when the anode is placed in the carotid fossa and the cathode over the spine. It is most common in this disease, but may occur in other conditions. The *reflexes* diminish in proportion to the atrophy of the muscles, and ultimately disappear completely; the patients gradually become almost incapable of voluntary motion; but for a time they learn to overcome their disabilities by the compensatory use of other groups of muscles. In the late stage the diaphragm becomes paralyzed and bulbar symptoms appear; usually the patients die from inspiration-pneumonia. Rare and probably accidental symptoms are disturbances of the pupillary reflexes and increase in the secretion of sweat.

Differential Diagnosis.—In *chronic antero-poliomyelitis* groups of muscles are affected without any particular order, and total paralysis is a very early symptom; in *amyotrophic lateral sclerosis* the spastic symptoms are present; in *syringomyelia* and *pachymeningitis cervicalis hypertrophica* disturbance of sensation, pain, and trophic lesions occur; in *Pott's disease* affecting the lower cervical region there are tenderness over the spine and sensory disturbances; in *peripheral neuritis* the fingers are unequally affected and the deltoid does not waste; in *arthritic atrophy* joint-symptoms are present; and in the peculiar *muscle-atrophies* following excessive use of certain groups of muscles, rapid improvement occurs when the cause is removed.

Prognosis.—This is unfavorable as to cure. The course is exceedingly slow, and the patients often live for a number of years after the first symptoms have appeared. They are, however, exceedingly liable to pulmonary complications, particularly a fatal form of bronchitis.

Treatment.—Prophylactic measures, such as the avoidance of prolonged excessive work, are rarely possible. Retardation may possibly be obtained by the systematic use of electricity, massage, and gymnastics. Gowers advocates the hypodermic injection of strychnin nitrate in ascending doses, commencing with $\frac{1}{100}$ gr. and rapidly increasing to $\frac{1}{40}$; one injection should be given daily.

PROGRESSIVE NEURAL MUSCULAR ATROPHY.

(*Charcot-Marie-Hoffmann Type; Peroneal Type, Gowers.*)

Definition.—A degenerative process, apparently commencing in the nerves, and characterized by muscular degeneration, with subsequent contractures, marked sensory disturbances, and a loss of the reflexes.

Pathology.—Degenerations have been found in the muscles, the peripheral nerves, and the spinal column. In the former the muscle-cells show degenerative changes. The nerves exhibit a chronic interstitial neuritis with proliferation of the connective tissue, and destruction of the myelin-sheaths and axis-cylinders. In the spinal cord degeneration has been found in the posterior columns. The nature of the disease seems to be akin to that of neuritis, the changes in the muscles and spinal cord being secondary to those of the nerves.

Etiology.—Heredity seems to play an important part in the causation of the disease, which may either occur in successive generations of a family or affect several members of the same generation. Sporadic cases occasionally occur for which it is impossible to trace any ancestral influence, though, as the disease has been known to skip a generation, it is not impossible that such cases are still hereditary. Males are much more frequently affected than females, and the disease almost invariably commences between the ages of ten and twenty years.

Symptoms.—As the name implies, muscular wasting usually begins in the muscles of the feet or hands, either the peronei, the common extensors of the toes, or the small muscles of the foot itself, or else in the muscles of the thenar and hypothenar eminences and the interossei. Usually the atrophy is symmetric. In the feet it leads to an early development of club-foot, which is most pronounced when the extremity is at rest. Very early the atrophy of the small muscles causes the toes to assume the claw position, and the atrophy of the peroneals causes foot-drop, so that in walking the foot is dragged along the ground. In the later stages the foot becomes permanently fixed in a position of equino-varus or valgus. The hands have the characteristic appearance given by a flattening of the ball of the thumb and middle finger. The interosseal grooves also become deeper and the fingers gradually assume the claw-like position ("*main en griffe*"). The disease extends slowly upward, involving the muscles of the calf and thigh or the forearm and arm. The affected muscles usually show distinct fibrillary twitchings that may be so severe as to give rise to an irregular tremor of the fingers. Spontaneous spasmodic contractions may also occur. When electrically examined the muscles either show a marked diminution in reaction to the galvanic and faradic currents, or distinct reactions of degeneration can be elicited. Similar electric changes are also found in the nerves. Mechanic excitability of the muscles is considerably diminished, these changes being found also in the muscles that are apparently healthy. The tendon-reflexes are usually absent, although in the early stages, when the muscles of the thigh are still unaltered, the knee-jerk may be merely diminished. Sensation is sometimes unaltered, but ordinarily there is considerable diminution to touch. It is possible that some cases show an alteration in the pain and temperature sense; often there are paresthesiæ, and occasionally, pains of considerable intensity. The general condition of the patient, however, remains excellent. The vegetative organs are unaffected and nutrition is therefore intact.

The **diagnosis** can be made from other forms of progressive muscular atrophy (particularly the type "Duchenne-Aran") by the sensory disturbances; from *locomotor ataxia* by the absence of sphincter disturbances; and from the *cerebral palsies of childhood* by the fact that it begins late in life and is distinctly progressive, showing also a diminution of the tendon-reflexes and reactions of degeneration in the muscles.

The **prognosis** is good as regards life, but unfavorable as regards cure or even improvement. The course of the disease is extremely slow.

The **treatment** employed in the other forms of amyotrophy may be tried, but so far nothing has succeeded in staying the course of the disease.

A type of disease closely allied to the preceding has been described by Déjerine under the name of "*infantile hypertrophic and progressive interstitial neuritis.*" The muscular symptoms were the same, but there were in addition ataxia, lancinating pains in the limbs, considerable sensory disturbances, Romberg's sign, myosis, with slow or absent pupillary reflexes and nystagmus. In addition to these a peculiar symptom in his case was the enormous hypertrophy of the nerve-trunks, which could be felt under the skin as large, firm cords. Pathologically the muscles showed degenerative changes and the nerves a pseudo-hypertrophy due to the enormous proliferation of the connective tissue and degeneration in the posterior columns of the spinal cord. The disease appears also to be due to old hereditary influence, the first 2 cases described being a brother and sister.

PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS.

Definition.—A disease characterized by a progressive loss of power in the muscles without disturbance of their electric reaction, while at the same time one or more of them increases in size and firmness.

Pathology.—Microscopic examination of the muscles shows that the hypertrophy has been produced by the hyperplasia of adipose tissue in the perimysium internum. The muscle-fibers may either be normal, atrophied, or hypertrophied, and there may be a relative increase in the connective tissue. The motor nerves are invariably intact.

The **etiology** of the disease is obscure. It appears to be transmitted by females chiefly to males. Occasionally it has been associated with mental disturbances that appear to indicate that it may be indirectly a nervous condition. Consanguinity, according to Gowers, may be a predisposing cause if it continues through several generations. The disease usually develops in early life, and those forms that occur after puberty are more common in females.

Symptoms.—The enlargement usually affects the muscles of the calves of the legs, although various muscles in other parts of the body may be involved, as the infraspinatus and masseter, or the muscles of the arms and thighs, giving the patient the appearance of an unequally developed athlete. Fibrillary contractions may sometimes be seen, but are not frequent. The electric reactions show no qualitative alteration, but are quantitatively diminished in proportion to the loss of power. This loss of power is manifested first in the gait, which is uncertain and waddling; next, by the difficulty the patient has in arising from the ground. He first gets on his hands and knees, then lifts his knees from the floor and, placing his hands first on his ankles, climbs up his legs until he assumes a more or less upright position (Fig. 76). In the later stages of the disease the volume of the muscles becomes less than normal. At this period contractures may occur leading to the development of club-foot or of lateral deviation of the spine. Lordosis may also be produced by weakness of the muscles of the back, and the spinal column, being no longer properly supported, may topple to one side or the other. Ultimately the patient may lose all power in the affected limbs and pass into a cachectic state, in which he dies. Few ever reach adult life. Some of the cases, however, seem to be milder in character, and may amount to nothing more than a slight weakness, which persists throughout life but does not seriously inconvenience the patient. Often signs of intellectual disturbance are present, the patient learning more slowly and showing an impaired intellectual coördination. At other times epilepsy may be present. A peculiar variety is known by the French as *forme fruste;* this is characterized by a rapid atrophy of the hypertrophied muscles, and consequently the course of the disease is more severe.

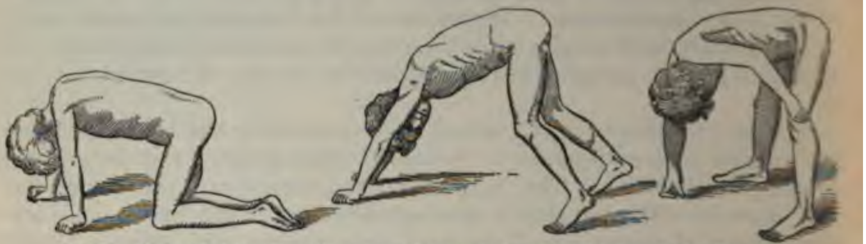

FIG. 76.—Mode of rising from the ground in pseudo-hypertrophic paralysis (Gowers).

The **diagnosis** is relatively simple, a typical case being easily recognized. In those cases, however, in which hypertrophy is slight, the disease may be easily confounded with progressive muscular dystrophy, a disease of which, perhaps, this is only a variety.

The **prognosis** is unfavorable, few of the cases living to adult life.

Treatment.—No tonic or alterative drug has exhibited the power of arresting the progress of the disease, and electricity is equally valueless. Gowers believes that persistent, systematic exercise and massage sometimes retards the course, but never leads to arrest. This should be tried in every case.

DYSTROPHIA MUSCULORUM PROGRESSIVA (*Erb*).

(*Scapulo-humeral Type; Juvenile Form of Progressive Muscular Atrophy.*)

Definition.—A primary myopathy, commencing usually in the muscles of the shoulder-blades and appearing about the period of puberty.

Pathology.—There are irregular hypertrophy and atrophy of the muscle-fibers, disturbances of the striation, and multiplication of the nuclei, with a relative increase of the entire fascicular connective tissue. The motor nerves, even in their finest terminations in the muscles, show no alteration, nor are changes found in the cord.

Etiology.—Heredity plays an important part, although occasionally the disease may occur sporadically. As in the other forms, it sometimes develops from emotional disturbances, exposure or fatigue. The sexes are affected about equally. The first symptoms usually appear at puberty, or not later than the twentieth year, although in a few cases the condition has developed in early adult life.

Symptoms.—The muscles first affected are usually the pectorals and the latissimus dorsi. From these the process rapidly extends to the muscles in the neighborhood—*i. e.* the serrati and the muscles of the back. The muscles of the arm, particularly the flexors in the lower arm and the long extensors, are usually most involved. In the thighs, the glutei and quadraceps femoris are particularly subject to the atrophic process. The muscles that are most likely to escape are the sterno-mastoid, the spinati, and the deltoid in the upper part of the body; and the sartorius and the muscles of the calves of the legs in the lower part. The muscles gradually waste, and the wasting is accompanied by a corresponding loss of power, a diminution in the reflexes and of the electric reactions. Reactions of degeneration are not present. Certain peculiar appearances are produced by the atrophy of certain of the groups of muscles. As the shoulder-blades are no longer supported, they stand out from the back, giving rise to the so-called "winged" appearance, and as the result of the weakness of the muscles of the back lordosis is exceedingly common. Finally, weakness of the muscles of the back, and particularly of the glutei, causes the patient, when he rises from the stooping posture, to go through the same actions that are carried out by children suffering from pseudo-muscular hypertrophy—*i. e.* climbing up his own legs. Finally, if the diaphragm is involved, dyspnea may develop to a greater or less extent, and may even lead to death. In certain rare cases the muscles of the face also show slight paresis, manifested by an inability to whistle, a disturbance of speech, and an imperfect closure of the eyelid. Motion is affected proportionately with the degree of atrophy. The gait is disturbed and becomes waddling, due to the alternate lifting of the sides of the pelvis in order to clear the foot of the ground. Sensation is never disturbed. The sphincters are not involved and bulbar symptoms do not appear, even late in the disease.

The **diagnosis** must of course be made from the other forms of progressive muscular atrophy. The differential diagnosis is essentially the same as that for the *infantile type*, excepting that from the latter it can be distinguished by the different order of invasion and the period of life at which it occurs. It is exceedingly difficult, sometimes, to make a differential diagnosis if the muscles of the face are also involved. In certain rare cases, such as one described by Oppenheim, there may be a congenital absence or a weakness of certain groups of muscles, and particularly of those most likely to be involved in this disease. In such instances the differential diagnosis may be made upon learning that the weakness has existed since birth and has not increased.

The **prognosis** is hopeless as regards improvement. The course of the disease is slow, but progressive, though life is usually not threatened unless the diaphragm is involved. The patient ordinarily lives for twenty or thirty years after being first attacked.

The **treatment** consists of systematic gymnastics, massage, and electricity. At the same time the general health of the patient must not be neglected. Apparent results are, however, rarely attained.

DYSTROPHIA MUSCULORUM PROGRESSIVA (*Déjerine-Landouzy*).

(*Facio-scapulo-humeral Type; Infantile Type of Progressive Muscular Atrophy.*)

Definition.—This form is characterized by the development of muscular atrophy of the face, shoulder, and arm, giving rise particularly to the *facies myopathica.*

Pathology.—The wasted muscles show in all respects the histologic changes found in the type of Erb, and essentially those found in the pseudo-hypertrophic form of muscular paralysis.

Etiology.—The disease is distinctly hereditary, and occurs ordinarily about the third and fourth years with equal frequency in both sexes, although a curious predisposition to one sex or the other is noted in certain families. As many as ten generations in a family have been recorded in which one form or other of myopathy developed. In a few cases some acute infectious disease or disturbance of general nutrition has preceded the muscular wasting, but there is no reason to believe that the connection is other than accidental.

Symptoms.—The disease usually begins in the muscles of the face. Of these the muscles of the eyelids and mouth first undergo degeneration, giving rise to a peculiar expression in which the eyes cannot be closed, the upper lid covering only half of the eyeball; the under lip drops forward and downward; the upper lip is wasted and expressionless; all wrinkles disappear, and the patient has a peculiar and strikingly stupid expression. The ordinary movements of the face are considerably affected. Whistling cannot be accomplished, speech is imperfect, and when the muscles of the eyeballs are involved ocular fixation is impossible. The shoulder-muscles next undergo atrophy. The earliest to be affected are usually the *cucullaris*, the rhomboids, and the pectoral muscles; finally, the disease extends to the arms, where we find the deltoid, biceps, triceps, and some of the extensors involved. Ordinarily certain groups of muscles seem to escape, among these being the muscles of mastication and the muscles of the forearm and hand. As the result of these changes, the shoulder-blades become more freely movable and stand out from the back and the shoulders, and when the patient is lifted by placing the hands under the arms, the shoulders show an abnormal degree of upward movement. The nutrition of the muscles is only affected in proportion to the atrophy. Electric reactions remain normal qualitatively, but are diminished quantitatively. Fibrillary contractions occur with extreme rarity; power is diminished in proportion to the wasting. Sensory disturbances do not occur.

The **diagnosis** is to be made from the *spinal* and *neural forms* of muscular atrophy and from the *congenital absence* of certain groups of muscles. From the two first-mentioned forms it can readily be distinguished by the fact that the hand becomes involved, if at all, in the last stages of the disease; also by the absence of the reactions of degeneration and of muscular twitching. It is also diagnosed from the neural type by the absence of disturbances of sensation. From the congenital absence of certain groups of muscles the diagnosis is sometimes difficult, for, curiously enough, the groups of muscles affected are usually the same as those affected by the myopathy. A distinction can be made partly by the history, partly by the more efficient and perfect compensatory hypertrophy of the muscles that remain.

The **course** of the disease is slowly progressive, only occasionally exhibiting a temporary arrest.

The **duration** is variable, but patients may live thirty or forty years after the first symptoms appear.

The **prognosis** is of course hopeless as regards cure or improvement. As regards existence, however, it is the most favorable of all the forms of progressive muscular atrophy—a fact that is probably due to the ability of the patients to walk until the very last stages of the disease, so that they are able to maintain a better physical condition.

The **treatment** is the same as that for other forms, and consists of electricity, massage, and especially of systematic gymnastics.

HEREDITARY MUSCULAR PARALYSIS (*Leyden*).

THIS commences in children, and usually between eight and ten years of age. It affects the muscles very much as they are affected in the pseudo-hypertrophic form, except that there is no increase in size. The disease is markedly hereditary in type.

ARTHRITIC MUSCULAR ATROPHY.

Pathology.—It has frequently been observed that after inflammation of a joint the muscles that move it have undergone a certain degree of atrophy. This usually occurs in the extensors, and is severe in proportion to the duration of the inflammation. Microscopic examination of the muscles shows a rather uniform diminution in the breadth of the fibers, as well as a slight proliferation of the nuclei and occasionally an indistinctness of the striation. The nerve-trunks and cord have been reported to be normal.

The **etiology** of the condition is not clearly determined. It has been supposed to be due to disuse, but if such were the case all the muscles moving the joint would be equally affected. Moreover, it sometimes occurs too rapidly to render this explanation acceptable. It has also been supposed to be due to the extension of the inflammation either to the nerves or directly to the muscles, but the other symptoms of neuritis are rarely present. Finally, Vulpian has suggested that it is of reflex origin, and this hypothesis is most generally accepted.

Symptoms.—The wasting usually occurs very rapidly after the onset of the joint-affection. The muscles show a diminished contractility to faradism and galvanism, but the reactions of degeneration do not occur. Occasionally there is fibrillary twitching. The mechanic irritability of the muscles is greatly increased, and the reflexes show a corresponding exaggeration, ankle-clonus being frequently observed when the knee- or ankle-joints are affected.

The **diagnosis** may be readily made upon the existence of the joint affection, the local character of the muscular atrophy, and the absence of degenerative reactions with increased mechanical irritability.

Prognosis.—Ordinarily, as soon as the joint has recovered, improvement commences in the muscles and progresses rapidly to complete restoration of function. In some cases, however, atrophy persists, and in a few instances secondary contractures take place.

The **treatment** consists, first, in the removal of the cause by the cure of the articular condition; secondly, in gentle massage and electric stimulation of the muscles. As a rule this should not be commenced until the joint is well.

MUSCULAR ATROPHIES.

These may also occur as a result of other conditions, such as direct injury, fracture of the bones, or prolonged work with a single group of muscles, but they scarcely demand separate description.

MUSCULAR HYPERTROPHY.

This occasionally occurs as an idiopathic affection. In these cases microscopic examination shows an increase in the size of the fibers, although sometimes there are slight degenerative alterations, such as the presence of vacuoles or indistinctness of the striation. The cause of the disease is unknown. It occasionally appears in those of a neuropathic heredity, and one case is recorded that developed in an idiot. The symptoms consist of enlargement of the muscles, which usually exhibit increased power, but, at the same time, great susceptibility to fatigue. Occasionally the power is diminished.

The *diagnosis* from pseudo-muscular hypertrophy is sometimes difficult. The *prognosis* is unfavorable for any improvement in the condition. No *treatment* that has any influence upon it is known.

THOMSEN'S DISEASE.

(*Myotonia Congenita.*)

Definition.—An hereditary disease of the muscles in which the groups that have been contracted by a voluntary influence remain for a short time in a state of contraction, and then relax slowly.

Pathology.—Certain authors have described alterations in the terminal nerve-plates in the muscles, but it is difficult to determine whether these alterations are artificial or an actual part of the disease. The peripheral nerves are normal. The muscles themselves exhibit the following alterations: The muscle-fibers are, on the average, of an increased transverse diameter—*i. e.* the smallest are the size of ordinary muscle-fibers and the largest about twice the size. There is also a distinct and considerable increase in the number of nuclei. The protoplasm is not so clear as in normal muscles, but shows a fine granular cloudiness, rendering the striation less distinct. Occasionally, the muscle-fibers are vacuolated. The connective tissue between the muscle-fibers is normal.

Etiology.—Hereditary influence is the most important factor in the causation of the disease. Thomsen, who was himself a victim, has been able to trace the disease for five generations in his own family. Occasionally a generation is skipped. Other factors that have been supposed to act as predisposing or exciting causes are prolonged exertion (1 case having developed in a man without myotonic antecedents after two years of severe exertion) and emotional disturbance of the mother during pregnancy. Exposure to cold, and fright, and a neurotic temperament have also been accused of exerting a predisposing or exciting influence. The disease is somewhat more frequent in males than in females, usually develops in early life, is often associated with manifestations of mental disturbance, and occasionally occurs in those whose ancestors have exhibited lesions of the nervous system other than myotonia.

Symptoms.—The chief symptom of the disease is the so-called myotonic contraction. If the patient, after a period of rest, attempts to set a certain group of muscles in action, the first contraction is made, but is not followed by relaxation for a considerable interval—sometimes as much as a half minute; during this period the muscles remain in a state of tonic contraction. Thus, if the patient attempts to shake hands, he clasps the other hand strongly, and the clasp persists. When he lets go, it is seen that a slight degree of tonic contraction still exists, for it is impossible for him to straighten out his fingers immediately. Upon a repetition of the movement the tonic contraction recurs, but not so strongly, and if the repetition is continued, it disappears entirely, so that the muscular system of the patient behaves in all respects like that of a normal person, and long walks or other severe muscular exertion may be undertaken. In some cases practically the whole muscular system is affected, although, excepting the muscles of mastication, the muscles of the face usually escape. In others the disease is limited perhaps to the upper, perhaps to the lower, extremities. In the former condition the patient may, upon an attempt to make a vigorous motion after resting, suddenly become rigid and fall to the earth with considerable force, often injuring himself severely. He will then lie upon the ground perfectly conscious, but incapable of relaxing his muscles. When the disease, as is more frequently the case, is limited to the lower extremities, the chief disturbances observed are in walking. The first step is accomplished, whereupon the patient halts, both legs having become fixed; after a time they relax and another step is taken. The period of delay is now much shorter, and after a few more steps disappears entirely. The severity of the contraction is diminished by moderate exercise, heat, and tranquillity of the spirits, and is increased by excitement, cold, and fatigue. The muscles of deglutition and the sphincters and the muscles belonging to the non-striated muscular system are never involved. Pain is not present, except perhaps a slight sensation of cramp, nor are there disturbances of sensation. Mental disturbances are frequent, and have been ascribed to the anxiety occasioned the patient by the disease. They consist of irritability, the avoidance of society, and sometimes of melancholia. The reflexes show various modifications; the knee-jerks may be either normal, increased, diminished, or absent. The most important pathognomonic symptoms are the alterations in the electric reactions of the muscles. The changes are as follows: Mechanic irritation of the motor nerves is normal or diminished; the mechanic irritation of the muscles is increased, and so modified that the contraction instead of being sudden is slow, with a long tonic after-contraction. The faradic irritability of the nerves is normal, and faradic excitation of the muscles produces a tonic contraction of long duration. The galvanic irritability is quantitatively increased and qualitatively altered; that is to say, ACC is equal to and sometimes even greater than KCC. All the contractions are slow, tonic, and of long duration. Finally, the application of the constant galvanic stream gives rise to rhythmic contractions that pass along the body of the muscles in slowly moving waves at the rate of about one to three per second. Occasionally qualitative galvanic alterations have been observed in the nerves. Finally, the appearance of the patient is of some value. The muscles are developed almost as much as those of an athlete, without a corresponding increase of power.

The **diagnosis** is usually easy, and particularly if it be possible to examine the electric reactions. The condition might possibly be confounded with *pseudo-hypertrophic muscular paralysis*, in which the muscles are also considerably developed; but instead of being normal they manifest greatly diminished power and fail to give a myotonic reaction. From *tetany* the condition may be distinguished by the absence of Trousseau's sign, by a briefer period of tonic contracture, and an absence of severe pains. From *spastic paraplegia* and *Little's disease* it may be distinguished by the fact that in these diseases the spastic conditions are permanent and do not disappear after exercise. From *occupation-neuroses* it may be distinguished by the fact that the cramps only appear upon the performance of a certain peculiarly coördinated movement. From *hysteria* it is differentiated by the absence of stigmata and the care an hysteric patient exhibits to avoid injury to himself, and by the peculiar electric reaction.

The **prognosis** is hopeless. The disease commences in early life and continues until death, with more or less frequent remissions and exacerbations. It is possible that these remissions may be permanent, and one case has been reported of a young woman whom marriage greatly benefited. The disease is rarely dangerous to life, excepting in so far that those who suffer from it are much more liable to injury.

Treatment is exceedingly unsatisfactory. Practically nothing can be done, although in a few cases systematic stimulation of the muscles has produced some mitigation. The patients often learn methods by which they can at least diminish the unpleasant symptoms. Certain movements seem to prevent or shorten the period of tonic contraction. Of course exposure to cold or emotional disturbance should be avoided as far as possible.

PART X.

THE INTOXICATIONS; OBESITY; HEAT-STROKE.

THE INTOXICATIONS.

ALCOHOLISM.

(*Alcoholic Inebriety.*)

Definition.—An acute or chronic intoxication due to the abuse of alcohol. It is a general degenerative condition, particularly of the brain and nervous system, characterized by a moderate (often progressively increasing) or excessive, continuous or periodic, craving for alcohol, leading to drunkenness. Alcoholism is often simply a variety of *inebriety* or *narcomania*, a congenital or acquired brain- and nervous disease, characterized by a resistless, permanent desire for alcohol (*alcoholic inebriety*). *Mania-a-potu*, or "crazy drunkenness," is an acute maniacal condition occurring in an alcoholic drinker of a neurotic constitution. *Delirium tremens* is an hallucinatory manifestation that occurs in habitual drinkers of alcohol, either as the direct consequence of the long-continued action of alcohol on the brain, or because of its sudden withdrawal in an inebriate. *Dipsomania* is an alcoholic insanity in which an intense maniacal "drink-impulse" occurs in a periodic drinker (usually of spirits).

Pathology.—In cases of death from **acute alcoholism** the brain and kidneys are found to be greatly engorged with blood. The gastro-duodenal mucous membrane is also markedly congested, injected, and covered with a thick, sticky, blood-tinged mucus.

Chronic Alcoholism.—Since alcohol is physiologically a poison, and not a food, and essentially a drug, and not a drink, the effects of its habitual ingestion are directly to produce degeneration of nearly all of the bodily tissues, and indirectly to increase the liability to many diseases by lessening the systemic powers of resistance, thus favoring fatality from such disease. The degree of pathologic change depends upon the innate vigor of the tissues, the age at which indulgence in alcohol is commenced, and upon the kind, degree of concentration, and the quantity of alcohol habitually taken. Ethylic alcohol is less deleterious than the "fusel oil" that is sometimes used as an adulterant in spirits.

The chief effects of chronic alcohol-poisoning are seen in the nervous and digestive systems, and in the kidneys. Fatty changes are prominent in the malt-liquor intemperates, while a connective-tissue overgrowth predominates in spirit-drinkers. The mucosa of the stomach presents the appearance of chronic gastric catarrh. Dilatation of the stomach is common in free drinkers of beer, ale, and porter. The liver shows the changes of chronic congestion, of fatty infiltration or degeneration, or of cirrhosis and contraction. The renal changes are analogous to those of the liver, the chronic congested ("pig-backed") and fatty kidneys occurring mostly in those who have drunk excessively of malt liquors, while the small, sclerosed, and fibrous kidneys (chronic interstitial nephritis) are seen in those who have been spirit-habitués. The heart is often loaded with fat, and the muscular structure may reveal fatty degeneration, being pale, flabby, friable, and dilated. The blood-vessels are atheromatous, thickened, tortuous, and sometimes varicose, and sudden death has been caused in inebriates by the rupture of small aneurysms of the middle cerebral artery. In the brain the various stages of sclerosis, with shrunken, narrow, and flattened convolutions, often appear. Chronic pachymeningitis, with slight hemorrhages, is not infrequent. The pia-arachnoid membrane also may be opaque and thickened, and serous effusions into the subarachnoid space and into the ventricles have been noted. The nerve-cells, nerve-centers, and nerve-fibers show degeneration, hardening, and atrophy. Alcoholic neuritis is especially prominent in many cases.

Etiology.—An impaired personal health and vigor, as well as the "personal equation" and a deficiency of will-power, self-control, conscience, and conviction, are among the *predisposing causes.* Drunken or inebriate parents frequently transmit to their offspring a morbid impulse or desire for alcohol, and an environment of depraved morality and of depressing and corrupting social influences are usually potent disposing influences, particularly in those who are illy prepared, by heredity or training, to resist the temptation and insidious activities of such evil surroundings. Although some assert that poverty predisposes to intemperance, it is more likely that, in a great majority of instances at least, intemperance is the cause rather than the consequence of poverty, both of individuals and communities. The *exciting cause* is the persistent misuse of alcohol as a beverage, in the form of distilled liquors or spirits, wines, and fermented or malt liquors.

Symptoms.—The symptoms of acute alcoholism range from mild intoxication to an acute delirium or a profound stupor and coma. It begins with the stage of *vascular relaxation* and of feelings of warmth and exhilaration, due to the depressing and paralyzing effects of the alcohol upon the vasomotor tone. The second stage is one of *partial functional paralysis of the nerve-centers,* marked disturbance of the faculties, muscular incoördination, and delirious speech. In the third stage, of "dead-drunkenness," there are acute coma, stertorous breathing, a bloated and congested face, a slow and full, but weak, pulse, a cold and clammy skin, a heavy alcoholic odor of the breath, and, sometimes, incontinence of urine and feces. It frequently happens that unconsciousness is not so profound but that the patient may be aroused, though replies to questioning are stupid and incoherent. Ordinary acute alcoholism seldom passes beyond a stage of exhilaration, ending in mild narcosis. Sometimes, however, the irritant action of the alcohol predominates over its narcotic action, giving rise to acute alcoholic gastritis or nephritis.

Acute mental disorders ("*acute alcoholic insanity*") are not infrequently met with. *Mania-a-potu* may come on quite suddenly in debauchees, or in those who have drunk hard during a short time, as in a night's carousal. The mental excitability increases until a violent maniacal storm not unlike the mania of epilepsy possesses the drinker. While in this state of infuriated delirium homicide may be committed. Tremors are absent. Acute *alcoholic melancholia* develops suddenly in some cases, with a suicidal tendency. *Delirium tremens* is more common in alcoholic inebriates, and is also seen at times in those who drink greatly to excess, but are not habitués. Convulsive seizures have been noted in some cases, interrupting the coma ("acute alcoholic epilepsy"); these may or may not be accompanied by mania. An acute *alcoholic paralysis* from multiple neuritis (occasionally with ataxic symptoms) may attack hard drinkers, and may last for several weeks or months.

Chronic alcoholism (alcoholic inebriety) I consider a true disease. While acute alcoholism may also be an occasional manifestation of the chronic affection, it is often a vice which, if indulged in to an excessive degree, or if too frequently repeated, becomes a disease, though it is difficult to determine at what point the transition occurs. Again, it is not always easy to learn whether the early acute alcoholic excesses are really vices or morbid, diseased cravings for alcohol in hereditary narcomaniacs. The disease of inebriety (alcoholic) is a condition in which, as some one has said, it is not whether one "cannot" or "will not;" but in which one "cannot will" to resist the desire for alcohol.

The steady, so-called "moderate drinker" who saturates his blood and tissues every day for years is much more apt to suffer from chronic alcoholic poisoning with its attendant degenerations than one who goes on a "spree" once a month for a day or two, and during the intervals is free from the toxic influence of alcohol. The *symptoms* develop very gradually, and are usually marked for some time by the deceptive sensation of stimulation, warmth, and well-being, due to the vasomotor paresis and the anesthetic effects of the alcohol. Impairment of digestion is early noted. There are a coated tongue, foul breath, vomiting before breakfast, and gastric distress after eating. Constipation alternating with diarrhea is common. Muscular tremors gradually develop and often progress into an ataxic gait. Insomnia, mental impairment, and blunting of the moral sense come on. "Alcohol dims the perception, confuses the judgment, paralyzes the will, and deadens the conscience" (Kerr). In his distress and degradation the inebriate seeks to relieve himself by taking more of the alcohol, only to find, on awakening from his narcosis, that body, intellect, will, and emotion are still more depraved. In fact, the brain- and nerve-disorders are more grave, permanent, and extensive in the majority of instances than those of the viscera. This is owing to the delicacy of the nervous mechanism and to the ready degeneration under the influence of the altered blood, and the consequent impaired cellular nutrition, directly due to the toxic action and deficient normal pabulum, and indirectly to the lessened elimination of waste-products.

Dementia is often the terminal state of the chronic inebriate. Delusions of persecution are quite masked in alcoholic insanity. The depurative organs manifest various symptoms due to the long-continued irritating action of alcohol. The liver is either fatty and enlarged, or cirrhotic and contracted, and jaundice, dropsy, and hemorrhoids, along with physical hepatic signs, are correspondingly observed. The watery eye, the injected conjunctivæ, the swollen eyelids, the bloated and flabby or pallid and shrunken face, the dilated capillaries of the nose (*acne rosacea*) and cheeks, may now be seen. The urinary examination will show in many cases the deranged function of the kidneys and point to the nature of structural impairment. On account of the weak and flabby heart there are palpitations, dyspnea, and precordial distress, and occasionally sharp pains. Chronic valvular endocarditis may be discovered. The pulse is soft and weak in beginning fatty degeneration of the vessels. Thickened arteries are common in old cases, and the pulsations are often increased in tension and usually rapid. Muscular capacity and endurance are greatly diminished.

Delirium tremens occurs in the majority of cases in inebriates or chronic drinkers during or after a debauch, and particularly from the use of spirituous liquors. It may occur, also, during abstinence from alcohol, on account of some mental perturbation, or fright, accidental shock, or acute inflammatory illness. It may either come on suddenly, or be preceded (often for a day) by some slight premonitory symptom, as anorexia, restlessness, or depression of spirits. The patient usually awakens at night with a tremor, becomes sleepless, wants to get out of bed to do some imaginary thing, talks constantly and incoherently, looks about uneasily and fearfully, and breaks gradually into a cool perspiration. Hallucinations of sight, hearing, and smell develop. The patient sees terrifying and loathsome reptiles, and tries to escape from them, or to clutch them in order to cast them away. The "horrors" may become so great that suicide may be attempted, as by falling out of the window. Auditory hallucinations may take the form of enemies, policemen, or the roar of wild animals. The muscular tremors increase, the pulse becomes frequent and weak, and the tongue coated with a thick white fur. There is moderate fever, which, if the delirium is prolonged, takes on a typhoid character, the tongue becoming tremulous, dry, brown, and fissured, with the onset of subsultus tendinum, carphologia, coma-vigil, and muttering delirium. In favorable cases improvement begins on the third or fourth day, from which time the symptoms gradually subside. Convalescence may be said to be established when restful sleep can be obtained; this is followed by a desire for food. In unfavorable cases the patient may pass from a typhoid state into exhaustion and death, or may die suddenly either during a paroxysm of cardiac failure or from some complication, as cerebral hemorrhage or pneumonia.

Diagnosis.—The condition of persons found dead-drunk is seldom mistaken for any other. The reverse more often happens, and in this way *apoplectic* and *uremic comas* may be diagnosed as alcoholic coma. Cases picked up in the street in a state of apparent unconsciousness should be carefully tested in this regard. Instances in which, as the *postmortem* examination subsequently has shown, cerebral hemorrhage has followed a drinking-bout, render the diagnosis more difficult; in such the patient should be given the benefit of the doubt and handled as though the case were one of apoplexy. An important early step is to ascertain whether the coma is complete, or whether the patient can be roused by shouting in the ear, by applying ammonia to the nostrils, or, better still, by pressing, with gradually increasing firmness, over a sensitive spot, as the supraorbital notch; if the unconsciousness is alcoholic, he will come to his senses, if only for a moment. Abstemious apoplectics have been known to stagger and talk thickly, like drunken men (Kerr), and have been arrested and taken to a police-station instead of to a hospital. *Congestion* and *lobar pneumonia* affecting the bases of the lungs should be looked for, as they are common causes of death in drunkards. A table giving the principal points in the differential diagnosis will be found under Uremia (*vide* p. 965).

The diagnosis of chronic alcoholism is made from the history, and from the muscular tremors (worse in the morning), vomiting, mental restlessness, "mendacity," and involuntary "lying" (Kerr). The condition may resemble general paralysis, and if the habits of the patient are kept secret it may be very difficult to differentiate these affections. A prominence of disorder of the digestive tract usually points to alcoholism. Nervous excitement, tremors, fear, wakefulness, and the distinctive physiognomy are more evident in chronic alcoholism, even when general paralysis has been caused by alcohol, which is apparently the case. *Paralysis agitans*, *locomotor ataxia*, *epilepsy*, and *nervous dyspepsia* may also be mistaken for chronic alcoholism by the unwary.

Delirium tremens is distinguished by the history, by the restlessness, delirium, hallucinations, tremors, and terrors. *Mania-a-potu* differs from the preceding mainly in its usual association with acute alcoholism in neurotics, in the muscular contractions, the furious mania, and convulsive movements. The delirium of *apical pneumonia* that obtains in some cases (as well as in meningitis) must be thought of in the diagnosis of delirium tremens. The diagnosis of *alcoholic neuritis* from other conditions simulating it will be found elsewhere (*vide* p. 1033).

Prognosis.—In acute alcoholism the prognosis is favorable in private, manageable cases. Many of the cases brought into hospitals are affected also with pneumonia, and usually die. The tissue-changes in chronic alcoholism are so profound, and they affect such delicate and vital tissues, that when the alcohol-habit thus becomes fixed permanent recovery never takes place. The treatment appropriate for the inebriate and forced abstinence from alcohol relieve many of the symptoms and some of the debility, but relapses are all too common and are almost certain to occur. Insanity and paresis are not infrequent terminations of chronic alcoholism. Many complications are apt to supervene, as Bright's disease, epilepsy, melancholia, fatty heart, pneumonia, and thrombosis. Alcoholic neuritis often clears up upon withholding alcohol and stimulating the peripheral nerves both by appropriate drugs and external remedial measures. Recovery from delirium tremens is dubious in cases of severe injury, inflammatory troubles, or infections.

Treatment.—In cases of acute drunkenness, which are only too commonly met with, nothing special is required except to prevent the ingestion of any more alcohol and to allow the patient to sleep until the elimination of the poison is more or less complete. The effects of the intoxication, in the general depression, headache, anxious and irritable stomach, and various functional visceral and nervous disorders, may need careful corrective and sustaining treatment for a week or more. The diet should be light and nutritious. Aperient waters, hot baths, with liquor ammonii acetatis frequently repeated, and a combination of dilute mineral acid and bitter tonics (nux vomica, gentian), are also indicated.

In profound cases of alcoholic coma, convulsions, or mania-a-potu no alcohol should be given. Trite though this injunction may seem, it is important to emphasize this statement, so that the physician may be sure to counteract a popular impression that the giving of more alcohol will cause a mania to subside *permanently*, and to guard against the smuggling of liquor to the patient by his misguided friends. It is often necessary to empty the stomach at once when collapse is imminent by the use of the stomach-tube or -pump, washing out the organ with hot water, to which ginger or cinnamon has been added. To this end emetics may be used—viz. ipecac or apomorphin, hypodermically (gr. $\frac{1}{8}$ to $\frac{1}{6}$—0.008–0.0108). The external application of warmth, friction, artificial respiration, faradism to the phrenic nerve, ammonia- or amyl-nitrite-inhalations, and hypodermics of atropin, strychnin, and digitalis, may all be tried. Hot rectal enemata or a calomel purge if the stomach will tolerate the drug should be used early. The maniacal attacks may be treated by hypodermics of morphin and hyoscin, and by such sedatives as chloral, bromids in large (ʒj—4.0) doses, and rarely such hypnotics as paraldehyde, trional, chloralamid, and the like. Indeed, it is very important to secure sleep as soon as possible. An excellent formula in cases of medium severity is:

℞. Sodii bromid., ℥j (32.0);
Tr. capsici, ʒj (4.0);
Tr. digitalis, ʒss (2.0);
Elix. simplicis, q. s. ad ℥ij (64.0).—M.
Sig. ʒj (4.0) every two or three hours, in water.

As soon as some quietude and sleep have been obtained, it is in order to administer concentrated food in an easily assimilable form.

The treatment of *chronic alcoholism* is more often best conducted in "homes" for inebriates, in hospitals, and similar institutions. At the outset there must be an "unconditional surrender" in the use of alcohol. Its withdrawal should be enforced at once in many cases, and very rapidly in all others, according to the judgment of the physican as to the psychic and physical condition of the patient. Substitutes for alcohol are the strong fruit-juices, as hot lemonade or hot ginger, and cardamom tea often is useful. Coffee, milk, cocoa, and hot broths are also to be recommended. The diet should be carefully increased in nutritive strength as the gastric irritability diminishes. Sometimes such sedatives to the stomach as the bismuth-preparations, effervescent alkaline drinks, and lime-water may be indicated. Peptonized food is often well borne at first in cases in which gastric distress is marked. Nutrient enemata are seldom required, but should be resorted to in the gravest cases, particularly during the states of alcoholic dementia. The general health must be looked after by placing the patient in the best of fresh air, exercise, cold and warm bathing, by mental and social occupation, and by diversion. When the craving for alcohol is hereditary and intense, seclusion in an inebriate-house or some similar institution is often necessary for a long time, inasmuch as the danger of lapsing into the former drink-habit is so common in these cases.

The insomnia of chronic alcoholism may be met temporarily by the use of large doses of bromids, chloral, hyoscin, or sulfonal. Morphin may be indicated at times, but should be used with great caution in order to avoid adding the morphin-habit to that of alcohol. Perhaps the best single agent to use in counteracting the symptoms of chronic alcoholism is strychnin, either as the nitrate or sulphate, hypodermically and by the mouth; iron, arsenic, the hypophosphites, dilute phosphoric acid, quinin, gold and sodium chloride, avena sativa, and the like are often useful adjuvants in the tonic treatment. Atropin, hypodermically, may also be recommended when vascular dilatation and weakness are prominent. A "substitute" for alcohol, both for its local and mental effects, may be prescribed to meet the occasional cravings. Tart fruits (as oranges and lemons), coffee, hot malted milk, ginger, gentian, and capsicum infusions may be tried. Tinctures should not be given in this form for obvious reasons. Sweating and purging the patient, and the administration of bromids, chloral, and gelsemium for a day or two in advance, may avert a "drink-storm" or the periodic cravings for alcohol that may be expected by prodromal manifestations. Sometimes, however, as in the sudden outbursts of dipsomaniacs, there is no time to institute their treatment. It is claimed that hypnotic suggestion will abolish effectually the ardent desire for alcohol in a certain number of neurotic cases of alcoholic inebriety. Temperance revivals may be said to do permanent good only in those similar neurotic cases that are fortunately impressionable to appeals by total-abstinence orators, but, in order to maintain the reformed drunkard's pledge, it is often necessary that interested persons continue to watch, guide, and inspire him, in order that a weakened will may not precipitate a cyclic lapse into his old habits.

In all cases the treatment will be incomplete unless the highest part of the patient's nature receives due attention throughout. The reason must be enlisted in the treatment, and this is best attempted by sound teaching concerning the fallacy of the prevalent belief in the virtues of alcohol as a beverage.

No pains should be spared to impress upon the patient the need of a persistent abstinence from all intoxicants as long as he lives. All the influence of culture, music, and the fine arts, of high-toned morality and pure, undefiled religion, should be enlisted to strengthen self-respect and to fortify volition and inhibition. Moral regeneration may thus in certain cases check indirectly the physical and mental degeneration, but it cannot efface the consequences of the alcoholic poisoning which it represents.

Delirium tremens requires firm but tactful isolation and vigilant nursing. All alcohol should be withheld. If stimulation is needed, aromatic spirits of ammonia, strychnin, and atropin, with bland hot drinks and broths, may be administered. Easily digested and nutritious food should be given to support the strength. Sleep must be procured by such means as are mentioned above in the treatment for *mania-a-potu*. The dosage required, however, is usually not as great, but must be kept up longer than in the maniacal condition. Cardiac weakness may need such stimulants as digitalis, strophanthus, and the ammonium salts. After the attack subsides, tonic doses of strychnin, chirata, gentian, asafetida, calumbo, and iron, together with graduated exercise out of doors, are to be employed. Turkish baths, industrial occupations, and the like are indicated to conserve the patient's strength and thus fortify him against yielding to temptation and a morbid appetite.

GINGER AND COLOGNE-WATER INEBRIETY.

Habitual drinkers of alcoholic ginger, capsicum, and lavender preparations, and eau-de-Cologne are practically alcohol-habitués or inebriates. They drink these liquids for the alcohol that is in them. The so-called essence of ginger (Jamaica ginger), which contains considerable alcohol in some of its preparations, is often used primarily for relieving an attack of "cramps" or "colic," and if frequently repeated, can readily induce a morbid habit of "ginger-drinking." In other cases the craving for alcoholic indulgence (often hereditary), may have been aroused by a social glass of wine, but, from a sense of shame the desire has been kept secret, and gratified by drinking eau-de-Cologne, lavender essence, or even tincture of capsicum. Perhaps many more such cases exist, and especially among neurotic women in good circumstances, than are usually recognized.

MORPHINISM.

(*Opium-inebriety.*)

Definition.—A chronic intoxication, due to the habitual use of morphin or of opium in some other form (*opiumism*).

Pathology.—In cases of death from acute or chronic opium- or morphin-poisoning there is nothing distinctive in the pathologic appearances. In acute cases vascular congestion of the brain and membranes has been noted; but even in chronic cases the tissue-degeneration and fatty and connective-tissue proliferations that are characteristic of alcoholism, are practically absent. Decided lesions are usually traceable to associated affections. The principal anatomic changes are those due simply to malnutrition. Thus, we have the emaciation and the shrunken appearance of cerebral anemia, and pallor and atrophy of the cardiac muscle and of the vascular walls. The dried and wasted structures, due to tissue-starvation, are quite a contrast to the fat-infiltrated or degenerated, cirrhotic, and inflamed tissue of alcoholic inebriety. Direct destruction of parenchymatous cells is more evident in the later.

Etiology.—The climate, country, and nationality have a certain disposing influence in the development of opiumism and morphinism. In the opium-growing parts of Asia, as in China, India, and Persia, where the climate is warm, enervating, and conducive to physical and moral abandonment during the greater part of the year, and in Turkey also, opium-eating-and-smoking habitués are as numerous as alcohol-habitués are in Europe and America among the Caucasians. Morphinism is more common here than is opiumism, except among the poverty-stricken.

Women are more commonly the victims of morphinism than men, except physicians and druggists as a class. Mattison has found 70 per cent. of his opiate patients to be medical practitioners. Many contracted the habit by using morphin for severe chronic neuralgia, insomnia, and the like. Indeed, pain and sleeplessness have been the principal source of this drug-habit.

Ennui and an idle spirit of irritation and adventure among the sensation-loving and luxurious sometimes sow the seeds of an indulgence in narcotics that bring forth fruitage in the form of a fixed, morbid, and uncontrollable craving for constant satisfaction, and a consequent physical, mental, and moral decline.

The incautious prescribing of morphin and the too ready hypodermic use of the alkaloid by physicians in treating various cases of pain are not infrequently the cause of morphinism. Overwork of the brain, great business or social strains, prolonged worry and anxiety either with or without work, insomnia, remorse, idleness, and secret vices, are the most common predisposing agents of the morphin-habit.

Paregoric, laudanum, chlorodyne, and "soothing-syrup" are drunk to a frightful extent in large cities among the poor and miserable, and cause great disturbance of the health of the habitués.

Symptoms.—These may be in abeyance for some time, while the habit is forming and the doses are still slight. As the craving increases, the dose and its frequency increase to keep pace with the desire. Anemia gradually develops, with sallowness of the skin, wasting of the features and body, languor, weakness, functional deterioration, mental depression, anorexia, restlessness, insomnia, tremors, irritability, shyness, dilatation of the pupils (except when under the influence of the drug), and a characteristic propensity to lying. Cardialgia is often complained of by those who use opium pretty constantly. The associated vices of opiumism are less violent and inflammatory than those of alcoholism, and more secretive and speculative, such as gambling and sexual perversions. Itching is frequent, and especially after taking the opium or morphin. Attacks of chills, followed by pyrexia, with delirium and transient albuminuria (renal congestion) occur in some cases. Diarrhea and dysentery have been observed in some instances. There may be also disturbances of the visual muscular apparatus. Sufferers from painful carcinoma in whom opium or morphin is required for steady use do not become, except in rare cases, true morphinomaniacs.

The *course* of morphinism is that of a progressive asthenia, in which cardiac palpitation, dyspnea, abdominal and muscular cramps, trembling, fear, sleeplessness, mental confusion, melancholy, slovenliness, and moral obtuseness come on. Some women, known to be kleptomaniacs, have been found to be secret opiumists. Sexual impotence in the male, and amenorrhea and abortion in the female, are common results. The skin is wrinkled, dry, and harsh, and may show numerous needle-scars and abscesses in those addicted to the hypodermic use of the drug. The *termination* is the direct result of the extreme debility or marasmus or of some intercurrent affection.

The **diagnosis** must be made from the history. When the latter is wanting because of a lack of veracity or deception, *chronic alcoholism* may have to be differentiated from opiumism. The more open and often periodic habits of the alcoholic habitué, and the general aspect of the physical and mental and complicating conditions, usually show marked differences between the two drug-intoxications.

Prognosis.—The likelihood of a cure is exceedingly remote. On the other hand, under proper conditions much relief may be given the morphinomaniac, and although the habit may be suspended only for a time, life may thus be prolonged for years.

The **treatment** is manifestly difficult and unpromising. Institutional isolation, rest, diversion, watchful care, regular and studied feeding, baths, and graduated exercise in the open air as far as possible, but under surveillance in order to prevent the smuggling of opium, morphin, or compound preparations containing either, are the most efficient measures. As to the manner of withdrawing the narcotic, much care, judgment, and tact form a *sine quâ non* in the treatment. A sudden and absolute stoppage of the use of the drug sometimes leads to great distress, and even to collapse ("abstinence phenomena"); it is, therefore, not to be recommended, as in chronic alcoholism. On the other hand, the too gradual withdrawal is torturing. A middle course, the "rapid-gradual method" of Erlenmeyer, is usually resorted to, in which the reduction of the quantity of morphin or opium to nothing occupies but a week or ten days. Various substitutes have been recommended that generally prove not to be substitutes at all, but simply act in a symptomatic way, and may lead to another habit as bad if not worse. Such drugs as cocain, hyoscyamus, belladonna, bromids, and chloral have thus been used.

In the symptomatic treatment of the morphin-habit moderate doses of bromids, with cannabis indica and some such vegetable bitter as gentian, may prove useful in allaying the nervous irritability and restlessness at night. Sulfonal is a good hypnotic in these cases. Cathartics, stomach sedatives alternating with tonics, concentrated foods, massage, hot and cold bathing, electricity (general galvanization), and "complete control over the patient" are usually indispensable adjuncts in the treatment after the withdrawal of the opium or morphin. Cardiac stimulants, strychnin and physostigmin salicylate (gr. $\frac{1}{100}$—0.0006) hypodermically, have been recommended recently as important in counteracting the functional depression of these habitués. Industrial activity, and mental and social diversion, aid in maintaining any improvement made and in rendering the patient less liable to a relapse.

PLUMBISM.

(*Chronic Lead-poisoning; Saturnism.*)

Definition.—A chronic intoxication due to the slow absorption of lead, either industrially or accidentally.

Pathology.—The principal lesions are found in the muscles, peripheral nerves, liver, kidneys, and mucous membranes. The affected muscles are wasted, pale-yellow in color, and, in advanced cases, show a marked fibroid growth. The vessels in the muscles also reveal arteriosclerosis. The peripheral nerves are affected with a parenchymatous neuritis, and are especially involved, with degenerative changes in the nerve-endings in the muscles. The nearer we approach the spinal cord along the course of an affected motor nerve, the less marked are the changes, although in some cases a very slight involvement of the anterior nerve-root cells has been noted. The cord is usually normal.

In the brain, the pathologic changes scarcely warrant us in attributing lead encephalopathy to them. Aside from a slight meningitis and arteriosclerosis of the cerebral blood-vessels here and there, with a corresponding connective-tissue growth and capillary hemorrhages, the evidences of lead-poisoning are practically *nil*. Cerebral symptoms are most probably the outcome of functional disturbances. The liver and kidney show parenchymatous atrophy and cirrhosis.

Etiology.—(*a*) *Personal susceptibility* to lead-poisoning is greater in some people than in others, all other things being equal. (*b*) Plumbism is more common in *adults* than in *children*, because of greater exposure. (*c*) *Sex.*—Women are more susceptible than men. (*d*) *Occupation* is the most frequent cause of lead-intoxication. Workers in white lead (plumbic carbonate), red lead, and litharge, all of which substances are used as paints, are especially to be mentioned as liable to saturnism. Among the most common industrial causes are the following: painting, plumbing, lead-mining, rolling sheet-lead, pottery-glazing, type-founding and setting, shot-making, dress-making (in which lead-dyed silk thread is used and the ends bitten off), lace-making, glass-grinding, and calico-printing. (*e*) An accidental source of lead-poisoning is found in the contamination of food and drink. Men employed in the manufacture of white lead and eating lunches in dusty work-rooms may also suffer from plumbism in this way. Drinking-water stored in lead-lined cisterns and passed through lead pipes is frequently contaminated, especially if the water contains a slight amount of acid. Flour, bread, biscuit, candy, butter, and milk may cause poisoning by adulteration with lead chromate, used to give a rich, yellow tint to these articles; and tobacco wrapped in lead-foil has, less commonly, resulted in symptoms of saturnism.

The *absorption* of the lead takes place mainly through the gastro-intestinal tract, especially through the lungs, and much less through the skin. It may be deposited in most of the soft tissues and viscera, but especially in the nerves, muscles, and liver. *Elimination* takes place through the kidneys, and probably, though in very slight quantities, with the bile and saliva, and through the skin.

Symptoms.—Depending upon individual susceptibility, it may be months or years before the first manifestations appear. *Anemia* is an early and marked symptom (*saturnine cachexia*). There is a moderate reduction of the corpuscles and of hemoglobin. The general nutrition is poor.

The characteristic *blue line* at the borders of the gums is rarely absent, especially in those who are not scrupulous in their attention to the teeth. It is, as a rule, most distinct at the roots of the lower canines and incisors, and is formed by a deposition of lead sulphid. Bluish patches may also be met with.

Colic is very common and is also characteristic. The pains center around the navel, and are quite severe and griping. They are associated with retraction and rigidity of the abdominal walls, and with obstinate constipation. The pains are paroxysmal, may be referred at times to the epigastrium, and may be accompanied by vomiting. Between the paroxysms a dull pain usually exists over the whole abdomen. During the attacks of colic the pulse-tension is increased and cardiac action lessened.

Paralyses are common symptoms, and may either be acute, subacute, or chronic in nature. Although usually localized palsies, they are sometimes generalized. The most characteristic lead-palsy is that known as *wrist-drop* (see also Multiple Neuritis, p. 1033).

Both fine and coarse *tremors* occur, though not so commonly as in chronic mercurial poisoning. They usually begin in the hands and arms, are rather constant, and are aggravated by *voluntary effort* and emotional excitement.

Cramps in the affected muscles and about the joints (*lead-arthralgia*) are occasionally noted. Slight anesthesia, especially in cases of wrist-drop, is sometimes detected here and there, but may in certain instances be due to saturnine hysteria.

The *cerebral symptoms* are important. The phrase "lead encephalopathy" includes such manifestation as delirium and coma, neuro-retinitis, aphasia, convulsions, hemiplegia, amaurosis, hysteria, and insanity. The delirium and coma are the commonest brain-symptoms, and may come on suddenly with tremors and hallucination. Epileptic convulsions are often severe. Hemianopsia has been observed. Mania and melancholia occur in cases of mental unbalancing, and hysteric outbreaks are seen in girls. Intense headache is not uncommon. "Saturnine gout," so called, is described as a result of chronic plumbism. The kidneys are contracted, the heart is hypertrophied, and arteriosclerosis is marked, with a diminution in the excretion of urea and uric acid. The pulse-tension is increased. These evidences show a similarity to gout, and favor the development of uratic deposits in the joints, but they are the effects of "mineral," and not of essential or true gout. Lead may be discovered in the urine by laying a strip of magnesium in it and noting the deposit of metallic lead if present (Von Jaksch). Abram asserts that the addition of a solution of ammonium oxalate (1 gm. to 150 c.c. of water) facilitates the test.

Diagnosis.—The history of exposure to lead-poisoning is usually clear in those working the metal in its various forms. *Accidental origins* of saturnism are often obscure and very difficult to trace, although if the characteristic wrist-drop, the blue gingival line, colic, and cachexia be present, the diagnosis is readily made.

Alcoholic paralysis of the lower extremities may be differentiated by the history, the greater prominence of sensory symptoms, and by the absence of the blue line on the gums.

Prognosis.—In the absence of the graver nervous, arterial, and renal symptoms, the prognosis is good. When there is profound paralysis, with reactions of degeneration, and especially in primary atrophy of the muscles, the prognosis is generally bad. In the severe encephalopathic forms, and in cases in which marked arteriosclerosis and renal cirrhosis are manifested, the prognosis is unfavorable, but depends upon the extent of damage done.

Treatment.—The prevention of plumbism is difficult in lead-working establishments, owing to the carelessness and indifference of both employers and employees, and to the lack of any adequate antidote during exposure. Rigid cleanliness is absolutely necessary, especially of the hands and nails and before eating. Means to allay dust should be regularly and constantly employed. Milk and sulphuric-acid lemonade have been recommended for use by workers in lead, for their supposed antidotal effects. As perfect ventilation as possible should be secured, and respirators are in use in some lead-works, being worn as "snouts." Potassium iodid should be given in chronic plumbism, beginning with small doses (gr. iii–v—0.1944–0.324), given preferably in milk, after meals.

In *lead colic* hot applications to the abdomen and hypodermic injections of morphin and atropin are often indicated. Efficient doses of Epsom or Glauber's salts are used to combat the constipation. Given in combination with dilute sulphuric acid (in order to form an insoluble lead sulphate) and with belladonna, the best and speediest benefits may be obtained thereby.

Iron for the anemia, strychnin and galvanism for the paralysis, lithia-water for the renal deterioration, and nitroglycerin or sodium nitrite for the arteriosclerosis (enough to relieve increasing tension) are the symptomatic items of treatment that are usually indicated. Rarely, hopeless cases of saturnine encephalopathy need to be sent to asylums for the insane.

ARSENICISM.

(*Chronic Arsenic-poisoning.*)

Definition.—A chronic intoxication resulting from the gradual absorption of arsenic.

Pathology.—The peripheral nerves show a degenerative neuritis, and the anterior horns of the spinal cord may be similarly affected.

Etiology.—The causes of arsenicism may be habitual, industrial, medicinal, or accidental. The individual predisposition to arsenic-intoxication varies in different persons. A neurotic diathesis usually underlies the habit of "arsenic-eating" in those who crave the drug for its alleged exhilarant or narcotic effects (*arsenic inebriety*). Not a few women suffer from chronic arsenicism as the result of the ingestion of arsenic "to improve the complexion and brilliancy of the eye." Men employed in arsenic-works of various kinds often suffer from the chronic poisoning. For example, miners and smelters of arsenic pyrites, dyers and wall-paper workers using Scheele's or Schweinfurth's green, artificial-flower makers, shot-makers, glass-workers, and taxidermists, are all liable on account of their occupations. Sometimes the medicinal use of moderate doses of arsenic, as in Fowler's solution, even for a short time, may in very susceptible persons induce arsenical paralysis (Putnam; Osler). Accidental arsenicism may come from living in rooms where wall-paper, carpets, colored paper ornaments, toys, or curtains are contaminated with arsenic anilin dyes; this does not occur so frequently as years ago.

Symptoms.—There are anemia, loss of flesh and strength, dryness and irritation of the mucosæ, of the eyes, nose, throat, and upper respiratory tract. Anorexia, nausea, and diarrhea indicate the presence of a gastro-intestinal catarrh. In some cases, milder than others, the fat is well preserved. Slight puffiness of the eyelids or eyebrows may occur, and some epigastric distress may be complained of. Marked conjunctivitis, occasional dysenteric attacks, loss of the hair, and numbness and tingling in the extremities form a commonly observed symptom-group. Cutaneous symptoms may appear, as pigmentation ("arsenic-bronzing"), and eczematous, herpetic, urticarial, and pemphigoid manifestations. Albuminuria with casts and blood mark the renal irritation that sometimes occurs.

The most characteristic evidence of chronic arsenic-poisoning is seen in the gradual increasing diffuse or multiple neuritis. Differing from lead-palsy, the leg-extensors and the peroneal group of muscles are involved first, although the arms may also become affected later (*vide* Multiple Neuritis, p. 1034). Contractions in the lower and a fine tremor of the upper extremities are apt to occur. Arsenic-poisoning may also cause headache, vertigo, melancholia, and hysteria. The drug is eliminated by the kidneys and may be found in the urine. Sometimes a great toleration of arsenic is observed in workmen and habitués, the only evidences being a clear, sallow, waxy complexion, a gloomy expression, and some dyspepsia, perhaps, as in the well-known Styrians.

Diagnosis.—This is not difficult, when once the source of the poisoning is determined. The clinical appearances are distinct from *lead-intoxication*, especially in the mode of progress of the paralysis, and in the more marked sensory symptoms combined with the motor-disturbances of arsenicism.

The **prognosis** is favorable in most cases in which removal from the exposure to the influence of arsenic is possible. A few cases die from the great general debility.

Treatment.—Abstention from the use of arsenic for cosmetic purposes, avoidance of its influence in the arts, care in its medicinal administration, and prophylaxis as regards the possible or discovered sources of contamination, form the first considerations in the treatment. Elimination of the arsenic may be promoted by the use of potassium iodid and purgatives. Gastro-intestinal and other irritations must be met by appropriate sedative remedies. The neuritis and palsies require —as soon as the tenderness and pain subside—massage and electricity. Judicious and wholesome alimentation and tonics are indicated.

MERCURIALISM.

(*Chronic Mercurial Poisoning.*)

Definition.—A chronic intoxication caused by the habitual ingestion, or combined industrial absorption of mercury, in susceptible individuals.

Pathology.—No marked pathologic changes have been noted in human beings, aside from the evidences of oral, gastro-intestinal, and renal irritation and inflammation. It is not improbable that the cerebral cortical areas suffer more from metallic irritation than do the spinal or peripheral nerve-tissues.

Etiology.—Some persons are much more easily mercurialized than others. (*a*) *Salivation* and *stomatitis* from the therapeutic use of mercury form a variety that is not infrequent in these days. (*b*) *Industrial origin.* The chief cause of chronic mercurialism is the inhalation of the vapor of the metal by artisans in the industries in which it is used. Thus miners and smelters and those engaged in making mirrors, barometers, thermometers, amalgams, felt hats, vermilion-pigment, and artificial teeth sometimes suffer from chronic mercurial poisoning. It should be pointed out here that mercury is volatile at ordinary temperatures, and is absorbed into the blood through the lungs, digestive tract, and skin. Calomel vapor-baths have caused poisoning in a few cases. (*c*) Purely *accidental* mercurialization also occurs. (*d*) Women and children are more susceptible to the action of mercury than men. In all cases the mercury exists in the tissues as an albuminate.

Symptoms.—There are anemia, emaciation, gastro-intestinal disorders, stomatitis, salivation, maxillary necrosis, ulceration of the gums, loosening of the teeth, fetor of the breath, marked tremors, and paralysis. The oral symptoms are not as prominent, however, as in acute mercurial poisoning. The hair falls out, the nails become brittle, and pigmentation of the skin is seen.

The *tremor* is characteristic. It is first felt or noticed in the tongue and lips, is usually fine, later coarse and choreiform, and spreads gradually throughout the muscular system. It is aggravated by voluntary effort, and may cease during sleep in mild cases. Speech is altered. Hysteric tremors may also exist. Great irritability and restlessness are common. Aphasia, hemiplegia, hemianesthesia, and peripheral neuritis with palsies, occur. There is no atrophy, nor are the reactions of degeneration present in the paralyzed muscles. Severe pains may be present in the extremities, including the joints, and grave cerebral symptoms occasionally develop (stupidity, headache, loss of memory, insomnia, hallucinations, delirium, coma, convulsions, and confusional insanity). Albuminuria with anasarca may occur. The effects of chronic hydrargyrism in women upon their offspring are also important, the children being rachitic, weak, sickly, and prone to tuberculosis.

Diagnosis.—The history, the characteristic tremors, paresis, and mental irritability are significant. In the absence of a history of exposure to mercury, the differentiation from *progressive general paresis*, *disseminated sclerosis*, or *paralysis agitans* may be more or less difficult.

Prognosis.—Recovery is common upon the removal of the source or on removing the patient from the source of the poisoning. Fatal terminations rarely ensue, and then in cases of mercurial encephalopathy of a grave type and with a tendency to idiocy.

Treatment.—Prevention of further poisoning is imperative, elimination is to be promoted, and the symptoms are to be met as they arise. Potassium chlorate, with the tincture of myrrh, and astringents are useful for the occasional stomatitis and salivation. Potassium iodid, and also sulphur baths, may be used to aid in the elimination of the mercury. Iron, cod-liver oil, good food and fresh air, and a free activity *of the emunctories are of positive value. Electricity may be resorted to for the paresis.*

FOOD-INFECTION AND PTOMAIN-POISONING.

In recent years there have been reported an increasing number of cases of serious illness that have been traced to infected and contaminated food. Undoubtedly many such instances are now brought to notice that in former times were attributed to other causes, or that were not diagnosticated because of a lack of knowledge. On the other hand, the increased consumption of canned and preserved meats has certainly augmented the liability to poisoning from these products, as the reports of cases show. Lack of care in the inspection and selection of the meats, uncleanliness, and sometimes unscrupulousness, in their handling and preparation, must result in infection, putrefaction, and toxicity. The infection of the food may be due to (1) disease of the animal or plant from which the food is derived; (2) microbic inoculation of the food after derivation and before ingestion by human beings; (3) infection by toxicogenic bacteria, and the presence of ptomains or toxalbumoses. The transmission to man of such affections in animals as tuberculosis, anthrax, glanders, and pleuro-pneumonia, by eating the infected meat, has been sufficiently proved. Again, meat and milk may become infected, before being ingested by the patient, by pathogenic micro-organisms, as of typhoid fever and diphtheria, or from the production of toxins owing to the action of non-pathogenic putrefactive micro-organisms. A great many instances of food-infection, particularly of meat and milk, have been shown to be due to the presence of saprophytic germs, this happening even when the articles of food have been obtained from healthy stock and have been kept free from specific pathogenic bacteria. It is not, however, the saprophytes themselves in all cases, but the poison developed in the food before it is eaten or formed in the body afterward, that produce the symptoms and sometimes death. According to Novy, some of the saprophytic bacteria with which food is infected outside of the body, under certain conditions, are capable of living in the body as parasites, especially on dead matter, and there become toxicogenic.

The chronic poisons or ptomains resulting from the action of the saprophytes in foods are called "putrefactive alkaloids;" those bacterial products of a proteid nature are called "toxalbumins" or "toxalbumoses." The latter, according to Vaughan, are more frequently present in infected foods. They are all absorbed from the digestive canal.

Poisoning by Infected Milk and Milk-products.—It is now well known that the cause of the high mortality-rate among infants in hot weather is traceable directly or indirectly to the "summer diarrheas" in children fed artificially, wholly or partially, with milk infected by numerous varieties of saprophytic germs and thus poisoned by ptomains, such as tyrotoxicon. This special chemical poison has been isolated by Vaughan, and discovered by him in cheese. It has also been found in ice-cream, frozen custards, and cream-puffs, and has caused poisoning-symptoms mainly of acute gastro-intestinal inflammation, "constriction of the fauces," nausea and vomiting, sharp, griping intestinal pains, headache, thoracic oppression, chilliness, dizziness, and sometimes purging, followed by relief in mild cases. In the severe and long-continued forms, however, exhaustion may supervene, with subnormal temperature, coma, collapse, and death in the graver cases. No chemical or physiologic antidote is known. Elimination may be assisted, and stimulation is needed. Irrigation may be employed for the former in both stomach and bowels. Strychnin, nitroglycerin, atropin, and the aromatic spirits of ammonia are most effective as stimulants.

Meat-poisoning.—Various tainted meats, as mince-meat "warmed over," veal pie, carelessly-kept chicken salad, badly-preserved and canned meats, partially-decayed sausages (*botulismus*) have caused violent symptoms of poisoning. Diseased raw and partially-cooked meat has also been eaten with disastrous results. It should be borne in mind that even prolonged cooking fails to destroy the toxic action of certain ptomains in infected meats; also, that meat that has been cooked and kept under certain conditions may become infected with bacteria as well as when it is raw. On the other hand, bad, putrid meat has been known not to cause toxic symptoms.

The *symptoms* caused by the poisoning are—"(1) those due to a true infection; (2) those due to simple poisoning" (Mann). Cases of the former group run the usual course of an infectious disease, often simulating typhoid fever. Those under the second division manifest the symptoms of a violent gastro-enteritis, with vomiting, intense colicky pains, purging, fever, accelerated pulse, nervous prostration, great muscular weakness, and cramps in the calves of the legs. Often a subsequent subnormal temperature, extreme depression, convulsive movement, vertigo, dimness of vision, dyspnea, somnolence, great soreness of the mouth, collapse, and sometimes death supervene. The mortality-rate varies from 15 to 55 per cent. of all the cases.

Differential Diagnosis.—Arsenic-poisoning may have symptoms similar to those of ptomaïn-poisoning. But, as Harrington[1] points out, there are three chief points of difference: in arsenic-poisoning there is swallowing because of pain; in ptomaïn-poisoning the pupils are usually dilated and the muscular prostration is almost as extreme as a palsy.

The *treatment* is largely eliminative, symptomatic, and supportive. The prophylactic measures, private and public, are generally obvious.

Poisoning by Fish (*Ichthysmus*) **and Shell-fish.**—Many instances of this serious form of intoxication have been produced. The fish may contain certain poison-glands, ovaries, etc. Especially is this true of certain species known in Japan, one of which is believed to cause the disease called "Kakke," which prevails during the summer months in Tokio. A certain species of fish (*Clupea venenosa*) inhabiting the West Indian waters is supposed to be always poisonous, although the source or true character of the poison is doubtful. In Russia, many cases of ichthyismus have resulted from eating both the fresh and preserved sturgeon and salmon meat that are affected with an infectious disease peculiar to the fish. In Germany and other parts of middle Europe a severe form of gastritis called "Barbencholera" follows the eating of sick barbels.

The use of tainted preserved and canned fish, eels, oysters, mussels,

[1] *Boston Medical and Surgical Journal,* Dec. 14, 1899.

crabs, lobsters, and the like, is more frequently the cause of symptoms of poisoning, however. Brieger's *mytilotoxin*, the active poison formed in some mussels, and the eating of which at Wilhelmshaven caused several epidemics, is probably developed only under certain favorable conditions of saprophytic infection. Devilled crabs, lobsters, and salad have also caused severe gastro-enteritis because of contamination with germs producing ptomains. Oysters have been accused of conveying typhoid infection (*vide* p. 26). The *symptoms* of fish- and shellfish-poisoning are variable. Sometimes marked cerebro-spinal manifestations predominate, with convulsions and paralysis. Dryness and constriction of the throat, dizziness, labored respiration, disturbed vision, jerky speech or aphonia, perhaps rapid pulse, loss of coördination, numbness, coldness of the extremities, dilated pupils, paresis, collapse, and death within a few hours, may ensue.

Other cases have a pronounced gastro-intestinal or choleraic group of symptoms, with nausea and vomiting, pain, tenesmus, and mucous and bloody stools. In some of them marked cutaneous irritation is shown by erythema, great heat and itching, urticaria, and swelling. Dyspnea, lividity, and sometimes delirium, have also been noted. The *prognosis* is grave in many instances. The *treatment* is similar to the above—namely, emetics, purgatives, enemata, and lavage. The indications are to be provided for as they arise.

GRAIN- AND VEGETABLE-POISONING.

Ergotismus.—Epidemics of ergotism have resulted from the continued use of meal made from contaminated grains grown on virgin soil. The parasite (*claviceps purpurea*) is a fungus that infests rye and other grains; it does not, however, grow readily where the soil is well cultivated, and epidemics of ergot-poisoning are much less frequent than formerly, if we except certain places in Spain and Russia. According to Kobert, three poisonous substances are found in the ergot: ergotinic acid, sphacelinic acid, and cornutin. The first of these is not poisonous when taken into the stomach; the second is supposed to cause gangrene; and the last produces grave effects on the nervous system, and is found only in fresh ergot, hence the greater prevalence of nervous manifestations in sickness that breaks out soon after harvest.

The nervous symptoms are remarkable for their convulsive characteristics (*ergotismus convulsivus*). Prodromes of weakness, tingling in the extremities, and headache may exist for several weeks before the spasms come on. The formication increases, and cramps and contractures, with flexed wrists and extended feet and toes, seize the patient. In severe cases epileptoid convulsions occur and may prove fatal. Delirium and, in very chronic cases, dementia may supervene. Recovery is slow, and the contractures may persist for some time, with muscular atrophy and anesthesia. In some interesting instances there may appear nervous symptoms resembling locomotor ataxia ("ergot tabes"), owing to posterior spinal sclerosis. Abortion results in pregnant women.

Gangrenous ergotism (*ergotismus gangrænosus*) is characterized by dry gangrene of the hands and feet, usually of the fingers and toes. Before the gradual blackening appears, there may be formication, pain, spasm, numbness, and coldness. As mortification and the line of demarkation progress, the parts drop off bit by bit, and fever may attend the sphacelation. Pneumonia (septic) may sometimes complicate this malady. The fatality has been considerable in some epidemics. The *treatment* of ergotism is entirely symptomatic.

Maidismus or Pellagra.—This is a chronic nutritional disturbance due to poisoning from eating contaminated corn-meal bread. The disease prevails extensively among the poorer classes in Lombardy, Spain, and southern France. The origin of the infection of the maize is said to be bacillary, the latter causing putrefactive or fermentative changes in the fresh, moist corn-meal, with the production of ptomains.

The *symptoms* at the beginning are languor, debility, indigestion, anorexia, restlessness, and occasionally diarrhea. This is soon followed by erythema, pain, and roughness of the skin. Exfoliation of the latter reveals a suppurating surface. In severe cases, paresthesiæ, spasms, paraplegia, headache, backache, delirium, and a suicidal mania may occur. Idiocy and profound cachexia may result from numerous attacks.

Structural changes have been found in the cord, and fatty degeneration and ulceration in the viscera.

Prophylaxis by thorough drying and careful storing of the meal is to be aimed at. The symptoms are to be met as rationally as possible.

Lathyrismus is an intoxication caused by the seed (used in the form of meal) of three varieties of vetch or chicken-pea, viz. *Lathyrus cicera*, *L. sativus*, and *L. clymenum*, or, respectively, red, German, and Spanish vetch. The meal is generally mixed with that obtained from other cereals. Its use for several hundred years has been observed to cause leg-stiffness, passing into a transverse myelitis, with sensory and motor paraplegia. Spasticity and exaggerated tendon-reflexes may remain for some time after the paralysis subsides. Slight fatty degeneration was noted by Cautain in excised bits of muscle. Very chronic cases may die in paralysis, from the toxic effects of the poison, which, thus far, has not been separated.

Mushroom-poisoning.—Though not so common as formerly, poisoning from eating non-edible mushrooms occurs now and then, owing to ignorance or carelessness in gathering, keeping, and cooking them. *Fresh morels* are poisonous, while those that have been dried and boiled are not so, because of evaporation or solution of the contained poison.

The *red agaric* (*amanita muscaria*), on account of the poisonous alkaloid muscarin that it contains, may cause very severe symptoms. These are nausea, vomiting, diarrhea, hemoglobinemia, hemoglobinuria, and jaundice (*probably hepatogenous*) in the case of fresh morel-poisoning (Strümpell). Tetanic and epileptiform convulsions give a slow pulse, dilated pupil, disturbed vision, salivation, coma, and death in the gravest cases of red-agaric intoxication, in addition to the symptoms of gastro-intestinal irritation.

The *treatment* is symptomatic. Emetics, purgatives, stimulants, and, in red-agaric poisoning, atropin, for its physiologic antidotal effect, are usually indicated.

OBESITY.

(*Polysarcia Adiposa; Lipomatosis Universalis.*)

Definition.—Corpulence, or the presence of an excessive amount of bodily fat, may be said to begin to take the form of a disease when it becomes an inconvenience or impairs the bodily functions.

Pathology.—The chief alteration is the marked and, in some instances, colossal increase in the fat deposit throughout the body. Not only is the adipose tissue greatly increased in localities where it is normally found or "preformed," as under the skin, but the various internal organs and tissues that are normally quite or nearly free from fat, may in obesity show a decided fatty infiltration.

The round, fat face, "double chin," broad and deep chest, large waist, thick and prominent, sometimes overhanging, abdominal *panniculus adiposus*, and bulky, cylindric, and apparently shortened extremities, are familiar appearances *postmortem* as well as *antemortem*.

There may be differences in the number and size of the fat-globules in the histologic elements. Thus, in the plethoric form of obesity the cellular fat-globules are larger than those of the anemic or hydremic form. Qualitative differences in the fat may also occur.

The *blood* in cases of obesity is increased in specific gravity to as much as 1065 or 1070. In a majority of cases the hemoglobin-percentage is also increased (plethora).

The *heart* is overlaid with fat, and the intermuscular tissue shows a decided fatty infiltration. Hypertrophic dilatation is frequently present.

The *arteries* may show fatty changes in the intima and media, and in the older cases chronic endarteritis with thickening and sclerosis of the vessels. The *veins* are often affected with varicosities.

Passive congestion and edema of the *lungs* are secondary to the cardiac weakness that is so common in advanced cases. The *liver, lungs*, and *kidneys* may be enlarged owing to fatty infiltration. Chronic interstitial nephritis may form a late complication of obesity.

The *stomach* may be dilated, and often shows a catarrh of the mucosa. Catarrhal enteritis of mild type also occurs sometimes.

Pathogenesis.—Obesity is probably dependent on a disturbance of cell-activity, and this disturbance of metabolism may be transmitted through heredity (*vide infra*). The overuse of carbohydrates leads directly to fat-increase. The consumption of proteids may also result in a fat-forming non-nitrogenous residue, which if not oxidized may produce fatness (see also Etiology).

Etiology.—Among the chief *predisposing* conditions are heredity, climate, habit, occupation, temperament, age, and sex. Among 202 of my cases, in which the family history was noted, heredity was distinctly traceable in 60 per cent. Gout was either in association or occurred among the antecedents in 45 per cent. of these cases and the same was true of "rheumatism" in 30 per cent. It followed typhoid fever in 6 per cent. of the totality of cases. The disease dated from child-birth in 14 per cent. of the cases and from marriage (apart from child-birth) in 7.3 per cent. among 151 females. The menopause has little if any influence. Corpulence is much more frequent among the inhabitants of hot, moist climates, and of low countries of the temperate and arctic regions. Thus it is commonly observed among Orientals, Dutchmen, South Pacific Islanders, Southern Italians, and certain African races. Sedentary habits and occupations form common predisposing factors. The sluggish, luxury- and rest-loving, phlegmatic temperament also favors an abnormal fat-deposition. As regards the age, polysarcia generally makes its appearance in persons of advanced middle life, between forty and fifty years, while hereditary obesity dates from infancy and early childhood; in women, it may appear at puberty and between thirty and forty years of age. Women, and especially Jewesses, seem to be more subject to corpulence than men. Congenital anomalies and monstrosities (idiots, cretins, acephali), also anemics and hemiplegics, are often excessively fat.

The *exciting* causes of obesity are especially the ingestion of too much fat-making food, the intemperate use of alcoholic beverages, especially beer, ale, and porter, with or without deficient exercise. The fat may be derived from an excess of albumin, fat, or carbohydrates in excess. An excessive diet of starches and sugars acts indirectly as a fat-producer by lessening the oxidation of the ingested fat and of the fat formed from proteids, because the carbohydrates themselves are so readily oxidized.

Symptoms.—Obesity is not accompanied by any bodily symptoms at first. Except some inconvenience, and a sense of burdensomeness during walking or working, nothing may be complained of for years. With the progressive development of the disease, however, and particularly with the involvement of the viscera, subjective manifestations increase in number and intensity. Usually the earliest troublesome symptom is *breathlessness* on exertion, due to a weak heart and to the hampering of respiration by heavy chest-walls and the upward-crowded diaphragm. In *plethoric* individuals the face is red and congested, as are also the mucous membranes (conjunctivæ, labiæ). In *anemic* subjects (usually women) the skin is pale, the muscles are flabby and weak; the pulse is small and compressible, and dyspnea, palpitation, inclination to rest often and sleep much, and dizziness (symptoms of anemia and chlorosis) are manifested. On the other hand, in plethoric, corpulent subjects (usually men) the muscles are firm and strong, and the pulse and heart-beats vigorous; later, however, the latter becomes weak and irregular. Brachycardia is not infrequent. The signs of fatty heart (*vide* p. 656) are obtained on physical examination. Muscular power may diminish very rapidly, the appetite often fails, and, oddly enough, great, fat men may consume very small quantities of food. Intercurrent acute infections (typhoid fever, pneumonia) are badly borne, and hyperpyrexia is usually associated with them.

The *liver* may show enlargement. The passive congestion of the *respiratory mucous membrane* is often signalled by cough and distressing dyspnea and attacks of asthma. Profuse sweating is common. There may be *polyuria* or *oliguria*, according to the activity of the skin and kidneys at the same time. Uric acid and the urates are usually found to be increased.

Symptoms of *gastric catarrh* and *gastrectasia* may occur. Great thirst and bulimia are noted in some instances. Constipation may be followed by chronic diarrhea. Sexual desire is often abated, and azoospermia is not rare. Corpulent women often suffer from uterine displacement and prolapse. Amenorrhea, sterility, endometritis (congestive), leukorrhea, and an aggravated climacteric are seen in obese women also. The skin is often irritated (intertrigo) by the excessive sweating, and by the friction of cutaneous surfaces in the folds of fat, as under the breast, at the abdominal and inguinal folds, and around the scrotum and labia. This may be followed by eczema. Painful excoriations, pruritus, acne rosacea (in alcoholics), and alopecia, are also not uncommon.

Complications.—Hernia, cardiac asthma, bronchitis, pulmonary congestion, edema, arteriosclerosis, albuminuria, glycosuria, anginal attacks, Cheyne-Stokes respiration, cerebral hemorrhage, and coma may manifest themselves as the precursors of the final stage.

Diagnosis.—This is not difficult in most cases. Care must be exercised in detecting associated conditions, complications, and sequelæ.

The **prognosis** will depend upon the peculiar features of each individual case, the cause and its removability, and upon the variety, degree, symptoms, and prevailing complications.

Treatment.—**Prophylaxis** is important in the earlier years of those showing an hereditary predisposition to corpulence. The fat-forming (farinaceous) substances must be diminished in the dietary. The proportions of fat and proteid in the food must be regulated according to the amount of muscular activity, and the latter should be encouraged in fresh air, along with cool bathing. At middle life, in those predisposed to polysarcia, all imprudences in eating and drinking should be cautioned against, and the quantities of various articles of food and the time of eating regulated. Outdoor sports and gymnastics should be also gauged accordingly.

The **dietetic** treatment of confirmed obesity is all-important. Inseparable from this is the stimulation of the bodily forces that oxidize and destroy the fat. These two means are utilized in the principal methods of treating obesity, and that method must be selected which invigorates, while at the same time it involves neither injury nor weakening of the patient.

The principal systems of dietary are those known by the names of Banting, Ebstein, and Oertel.

In "Bantingism," sugars, fats, and starches are greatly reduced in the diet-list; water, however, is not restricted, and vinous and spirituous liquors are permitted. In those of a rheumatic or gouty diathesis Banting's heavy proteid and alcohol dietary is not to be recommended. It is best, I think, to exclude alcohol in most cases, owing to its effect in diminishing tissue-oxidation and in retarding cell-metabolism. This method fails to secure elimination of waste products.

In Ebstein's diet-list more than double the amount of fat and carbohydrates is permitted as compared with Banting's list, whilst the albuminous substances are diminished. Fat is freely allowed, as this does not increase stored fat (?), but tends to impair the appetite, while sugar and potatoes only are strictly forbidden.

Oertel of Munich also allows more fat than Banting, but less fat and more (about double the quantity) proteids and carbohydrates than Ebstein. The amount of *free* water permitted daily is only one pint; about one pint additional in other food is allowable.

Oertel[1] writes: "The body stores up fat if more than 118 grams of albumin and 259 grams of fat, a total of 277 grams (2894 calories), are taken in. On the other hand, 110 grams of albumin and 600 grams of starch, a total of 710 grams (2944 calories), may be given without producing a deposit of fat. With a mixed diet the limit lies near 118 grams of albumin, 100 grams of fat, and 368 grams of starch, a total of 586 grams (2923 calories)." His diet-table for obesity is appended:

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[1] *Twentieth Cent. Pract. of Med.*, vol. ii. pp. 698, 699.

	Albumin.	Fat.	Carbohydrates.	Calories.
Minimum	156	25	75	1180
Maximum	170	45	120	1608

Oertel gives a special diet-list in circulatory disturbances.

On the basis of Voit's laws, Strümpell recommends in the average cases 125 gm. (4 oz.) or more of albumin, 40 gm. (1¼ oz.) of fat, and 150 gm. (462 oz.) of starch. Schwenniger's rule differs from Oertel's merely in the forbidding of liquids with the meals and in permitting their use only after two hours have elapsed. Yeo's diet-list is also a useful guide.

In plethoric obesity a judicious rearrangement of the food (some increase of the albuminoid substances), coupled with sufficient muscular exercise (walking, horseback-riding, bicycling, rowing, swimming, gymnastics) accomplish successful reduction, as a rule.

Increasing weakness of the heart with an impeded circulation naturally diminishes the excretion of water by the cutaneous and renal routes. In such cases the circulatory system must receive unusually careful attention and the consumption of fluid must be limited. If evidences of anemia be present, the amount of liquid may be also much restricted and the fat-forming dishes rigidly excluded. "The hydremic form must be opposed by the ingestion of an abundance of albuminoid material, of fat-producing substance, and the hydrocarbons" (Oertel).

Under any system of dietetic treatment the patient should be weighed accurately and frequently, and the food-limit be diminished or modified according to the results. The food may be weighed and measured at first, but the patient soon learns to estimate by bulk the requisite quantity of each substance.

The following dietary illustrates what may be ordered in some cases:

Morning Meal.—Fine wheat-bread, 1¼ ounces (40.0); a soft-boiled egg; milk, 1 ounce (32.0); sugar, 77 grains (4.9); coffee, 4¼ ounces (136.0).

Noon Meal.—Soup, 3 ounces (96.0); fish, 3 ounces (96.0); roast or boiled beef, veal, or game or poultry, 6 to 8 ounces (192.0–256.0); green vegetables, 1½ ounces (48.0); bread, 1 ounce (32.0); fruit, 3 or 4 ounces (96.0–128.0); no liquid (or only 4 or 5 ounces—120.0–148.0 cc.—of very light wine).

Afternoon Meal.—Sugar, 77 grains (4.9); coffee, 4 ounces (128.0); milk, 1 ounce (32.0); occasionally bread, 1 ounce (32.0).

Evening Meal.—Caviare, ⅓ ounce (10.6); one or two soft-boiled eggs; beefsteak, fowl, or game, 5 ounces (160.0); salad, 1 ounce (32.0); cheese, 1 dram (4.0); bread, rye or bran, ½ ounce (16.0); fruit or water, 4 to 5 ounces (120.0–148.0).

The mechanical treatment (to increase oxidation), by exercise, is to be used in conjunction with the dietetic. The form of the exercise, and also the time and frequency, must be adjudged for each case (*vide supra*).

When cardiac dilatation and myocardial degeneration (fatty) are the cause of symptoms of precordial distress, dyspnea, and palpitation, resort may be had to Oertel's system of graduated walking on the level or climbing along "health paths" (*vide* Fatty Overgrowth, p. 661). Or, the well-known Nauheim or Schott treatment may be used. Great care must be exercised in prescribing the mechanical treatment in obese persons who have atheromatous vessels.

The **medicinal** treatment is neither satisfactory nor successful. Causative or associated conditions—*e. g.* gout—may present special therapeutic indications. The juice of the phytolacca berry may reduce the weight, but is harmful.

Recently, the use of thyroid extract has come into favor. Leichtenstern, Wendelstadt, Ewald, and others have reported success in a number of cases, especially in those exhibiting the anemic, flabby, "myxedematoid" form of obesity. The loss of weight was from 2 to 3 pounds (1–1.5 kgms.) in one week, and as high as 20 pounds in two to four weeks. In two of my own cases belonging to this category the use of thyroid extract (desiccated) in small doses (gr. j—0.0648, t. i. d.) caused a progressive loss of weight at the rate of 4 and 6 pounds per week respectively, without injury to the general health. Thyroidin, the active principle of the thyroid gland, as shown by Baumann and Ross, and iodothyrin give results that are perhaps as good as those of thyroid-feeding. Jeozykowski treated 10 cases of corpulence by thyroidin in doses from 5 to 8 grains (0.324–0.518) *per diem.* In 1 case more than 40 pounds (18.1 kgms.) were lost in two months, and in another 30 pounds (13.6 kgms.) in three months. Symptoms of thyroidism are the signal for a reduction in the dosage of thyroid extract (*vide* Myxedema, p. 468). Hematinics are indicated in the anemic variety of obesity.

HEAT-STROKE.

(*Sunstroke; Insolation; Thermic Fever; Heat-exhaustion; Heat-prostration.*)

Definition.—A diseased condition the effect of exposure to excessive heat.

Pathology.—*Rigor mortis* is marked and comes on early. The high temperature of the cadaver accelerates the putrefactive changes, which also appear early. There is considerable venous engorgement of the brain and of the cerebral and spinal membranes; also of the lungs, spleen, and conjunctiva. The blood is fluid and dark, and the corpuscles are crenated and do not tend to form rouleaux. Ecchymoses and extravasations of blood are found in the skin, the serous membranes, and the cavities, around the superior (cervical) sympathetic ganglia and the vagus and phrenic nerves. Parenchymatous changes in the liver and kidneys may be found. Rigid contraction of the left ventricle is a notable feature, while the right ventricle is usually dilated with blood. Van Gieson's recent report of the cellular pathology of the cerebro-spinal system in 3 cases of sunstroke in New York shows an acute parenchymatous degeneration of the neurons of the whole neural axis similar to that of, and, Van Gieson thinks, here actually due to, "a species of auto-intoxication." He found the chromophilic plaques in the cortical cerebral and cerebellar (Purkinje's) cells and also in the cells of the anterior horns of the spinal cord, diminished in number, changed in shape and position, sometimes finally broken up, and even entirely absent. The nuclei stain more deeply than normally.

Etiology.—Anything that lessens bodily resistance to external high heat predisposes to heat-stroke. Thus, privation, unsanitary surroundings, fatigue of body or mind, emotional excitement, worry, and excessive fretfulness, overeating, indulgence in alcoholics (especially), and previous attacks of sunstroke, are all conducive to heat-stroke on exposure to high temperature. Males are affected more often than females.

Sunstroke occurs in persons (on land) working hard under the direct rays of the sun, in an atmosphere that is very hot and humid, still, and sultry. Soldiers on the march and heavily accoutered, masons, bricklayers, hod-carriers, roofers, drivers, farmers, and other out-door laborers are particularly subject to insolation.

Heat-stroke and **thermic fever** are terms more appropriately applied to those similarly affected in midsummer while working in places not exposed to the sun, but yet close, confined, and excessively hot, such as glass-works, foundries, ocean steamers, stoke-holes, boiler-rooms, steam laundries, sugar-refineries, kitchens, and the like.

Heat-exhaustion (*prostratio thermica*) is caused under similar conditions as the preceding, but manifests dissimilar, and sometimes almost opposite, effects.

The majority of the cases of sunstroke occur between 2 and 5 P. M., although heat-stroke and heat-exhaustion may occur at night as late as 10 or 11 P. M., as among bakers, night engineers, and hotel cooks.

It seems to be the consensus of opinion that the direct cause of the symptoms of sunstroke, heat-stroke, or heat-prostration is the action of the excessive heat upon the heat-centers, or upon the vasomotor center or nerves (H. C. Wood), the former of which, if paralyzed, produces "*thermic*" or "*heat-fever*," while the latter, if paralyzed, produces *heat-exhaustion*.

It should be stated, however, that Lambert and Van Gieson,[1] after a clinical and pathologic study of 805 cases of sunstroke occurring in New York City during 1896, hold to the not improbable view that the immediate basis of sunstroke is autotoxic, with heat only as a contributing cause.

Symptoms.—Two forms of heat- or sunstroke are usually met with: (1) The *asphyxial* or *apoplectic* form; (2) the *hyperpyrexial form*. Flint believes that the majority of the cases of sunstroke are combinations of apoplexy and exhaustion. Vallin puts all cases of insolation into two classes: the first, sthenic or asphyxial, corresponding to our hyperpyrexial or congestive variety; the second, asthenic or syncopal, corresponding to our heat-exhaustion. Mixed forms may occur quite frequently, the most prominent symptoms being referable to the organs suffering the most, as the cerebro-spinal system, heart, lungs.

Heat-apoplexy (*asphyxial sunstroke*) is probably the least frequent form. There may be sudden premonitions, or dizziness, chromatopsia, throbbing headache, cessation of sweating, or dyspnea. Sometimes the patient, while at work in the sun, suddenly falls unconscious, a few convulsions may occur, and in this state he may die with symptoms of cardiac failure. More often, insensibility is not so profound as complete coma, there is much restlessness, epigastric "cramp" may be complained of, also a sense of thoracic oppression, and occasionally there are nausea and vomiting. The headache may be intense, the face is flushed, the pulse is rapid and full, the temporal and carotid arteries are bounding, the breathing may be labored and stertorous, the pupils are contracted (except in grave cases), and urination is often frequent. The skin is hot and dry, and may show petechiæ. The tongue is coated with a whitish fur. A wild delirium has been observed in some cases. The temperature may be subnormal, and is not higher than 102° F. (38.8° C.) in many instances. In others, a mild degree of thermic fever may be associated with the apoplectic condition, the thermometer registering 104°–106° F. (40°–41.1° C.). In fatal cases the coma becomes deeper and deeper, the pulse more rapid and feeble, and Cheyne-Stokes respiration may precede the termination. A "mousey" odor about the body has been noted. In favorable cases the temperature falls to normal by lysis in three or four days, consciousness being rapidly regained at the same time.

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[1] *Med. News*, July 24, 1897.

The *hyperpyrexial* variety comprises the numerous cases of marked sunstroke that resemble the preceding type, with the addition of an intensely high temperature (*thermic fever*). The patient may suddenly become comatose and die in an asphyxiated condition, with a temperature as high as 110°–115° F. (43.3°–46.1° C.) or even higher.

Sometimes prodromes, as anorexia, progressively increasing physical weakness, cramp-like abdominal pains, irritability and restlessness, vertigo, colored and blurred vision, lack of sweating, a "bursting" headache, and an irritable bladder may exist for several days. A subconscious (automatic) state, in which the patient may be unaware of his surroundings, although walking or even working, may be noted for hours before he is stricken down. The onset is marked by hyperpyrexia; the skin is hot, burning, dry, sometimes flushed and red, and sometimes cyanotic and clammy; the eyes are suffused or "staring and filling," with pin-point pupils. There is a full, rapid, and non-

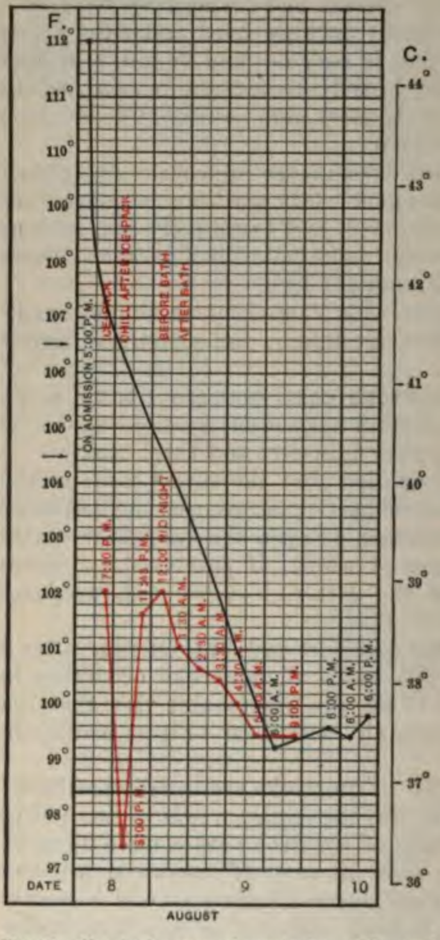

FIG. 77.—Chart of a case of sunstroke. J. D., aged forty years; steam-fitter. Recovery.

compressible pulse, and coma may be present. Clonic spasms may alternate with either muscular rigidity or flaccidity. Delirium, moaning, jactitation, and explosive expiratory sounds may occur. There is frequently incontinence of both feces and urine. The temperature is very high in most of the cases, varying from 105° to 112° F. (40.5–44.4° C.).[1] The pulse-rate varies with the temperature, from 90 to 160 beats per minute. The respirations are also increased to 24–50 per minute. Many of the alarming symptoms, including the high fever (*vide* accompanying chart, Fig. 77), unconsciousness, cyanosis, dyspnea, and convulsions, may greatly subside during and after the use of the cold bath. Secondary exacerbations occur for a few days before convalescence is established in the favorable cases (*vide* chart, Fig. 78).

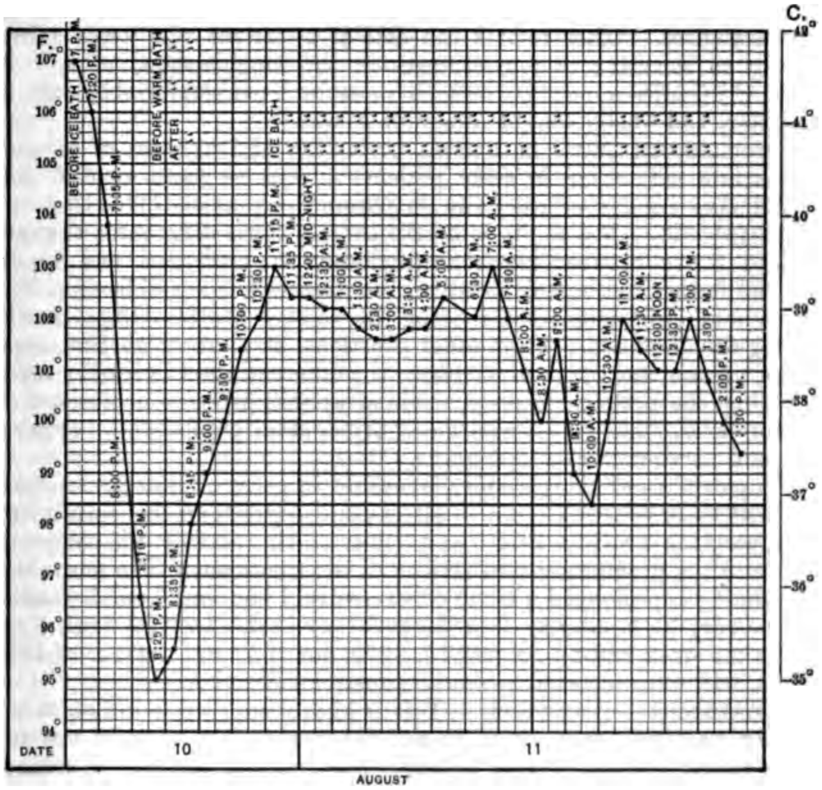

FIG. 78.—Chart of a case of sunstroke. C. B., aged twenty-nine years. Recovery.

Some patients never rally, and die in a state of asphyxia. Retention of urine (suppression) is observed at times, and particularly in those accustomed to the use of alcohol. Leukocytosis is noted, besides the crenation of the erythrocytes (degeneration of the red cells).

Fatal *complications* of sunstroke are pneumonia, meningitis, uremia, and cardio-respiratory paralysis.

Heat-prostration or *heat-exhaustion* may come on gradually or suddenly, with prodromal symptoms (dizziness, faintness, headache, nausea, thirst, drowsiness, yawning, epigastric or lumbar pains, numbness and tingling of the hands and feet). These are followed by coldness, clamminess, and pallor of the surface, marked muscular weakness and prostration, a small, febrile, rapid pulse, sighing breathing, syncope, and collapse in the graver cases. The temperature at first is subnormal (95° to 97° F.—35° to 36.1° C.), though mild thermic fever of from 100° to 102.5° F. (37.7°–39.1° C.) may be present. Consciousness is rarely completely absent and is regained early. Recovery usually takes place within one or two days, and in milder cases, under prompt and appropriate treatment the patient may be ready to go about in a few hours. In a few cases of extreme prostration in weakly persons death may ensue from cardiac failure.

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[1] Lambert (*loc. cit.*), reports a case in the N. Y. Hosp. of 117.8° F. (47.6° C.).

The sequelæ of heat-stroke are quite interesting and peculiar in some instances. Osler relates the case of a patient who "was subsequently so sensitive to temperatures in the neighborhood of *75° F.* (23.8° C.) (italics mine) that at such times he lived comfortably only in the cellar, and finally sought refuge in Alaska."

Chromatopsia, severe headaches, irritability and ugliness of temper, or delirium may occur in some patients as soon as warm weather sets in, and may be due occasionally to chronic meningitis (Wood).

Diagnosis.—Bearing in mind the characteristic differences that are outlined above between sunstroke (including the asphyxial and hyperpyrexial forms) and heat-exhaustion, the diagnosis is not difficult. The history and circumstances attending the seizure are also important in making the diagnosis. From other affections, as *acute alcoholism*, *meningitis*, *uremia*, and *cerebral apoplexy*, the differentiation is readily made by noting the previous history, mode of attack, presence or absence of thermic fever, state of consciousness, urine, skin, pupils, pulse, respiration, and nervo-muscular apparatus.

Prognosis.—This is usually favorable in cases of heat-prostration. It is less so in sunstroke, but in all cases it depends on the severity of the stroke, the previous health and habits of the patient, the complications, and the promptness and facility of the treatment. The mortality-rate during a prolonged period of excessively hot and humid weather may be very high, ranging from 15 to 50 per cent. In New York City, during the week ending August 15, 1896, out of a total number of 1810 deaths, 648 were reported as due to sunstroke (Lambert).[1]

Treatment.—**Prophylaxis.**—This is highly imperative in hot, sultry weather, particularly in cities, in which persons must work in the sun or in poorly-ventilated and highly-heated, closed places. Workmen should be taught and warned privately and publicly, as through the medium of the press and Health Board circulars, to take extra precautions during hot weather, to work and sleep in as well-ventilated rooms as possible, and to secure artificial ventilation, if necessary. They should live regular and temperate lives, avoiding alcohol and heavy eating; oat-meal water should be drunk, light-weight and light-colored clothing should be worn, and the direct rays of the sun should be avoided as much as possible. The condition of the skin should be watched and care taken that sweating continues freely. Shelter or rest should be sought at once if sweating stops. Cool wet cloths or green leaves should be worn inside a light straw hat, and sometimes it may be necessary for employers to shorten the hours of labor during the hottest part of the day.

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[1] *Loc. cit.*

Treatment of the Attack.—Cases of ordinary *heat-prostration* seldom require much treatment beyond the removal of the patient to the shade of a comparatively cool place, loosening all constricting clothing, spraying with cool water, the use of ammonia- or amyl-nitrite-inhalations, and of the aromatic spirits of ammonia or spiritus glonoïni by the mouth. If the temperature is subnormal and collapse threatens, a hot bath is advisable. Strychnin and digitalis may be used for a day or two to combat the nervo-muscular weakness.

Heat-stroke, especially the hyperpyrexial cases, must be promptly treated by the application of the ice-bath (ice floating in a tub of water), temperature about 40° F. (4.4° C.), or by rubbing, by the cold pack, or by the needle-spray with iced water.

In the *asphyxial* cases venesection is frequently indicated. The subcutaneous or intravenous injection of physiologic salt solution (F. A. Packard) may be a valuable procedure in many cases. External stimulation should be applied to the precordium by mustard and to the feet by hot bottles, and hypodermic injections of nitroglycerin, strychnin, atropin, brandy, camphor, or ether are useful. Ice should be rubbed over the head constantly. Care should, however, be taken to see that the temperature is not reduced too far. A temperature of about 102° F. (38.8° C.) should be the signal for cessation of the ice-bath, and for the removal of the patient to a cot, where he is to be rubbed dry and allowed to rest until an exacerbation of fever indicates the reapplication of the cooling measures. Ice-water enemata, with or without brandy, are often useful adjuvants. The needle-spray of cold water is an excellent nervous stimulant as well as antipyretic. It is given while the patient lies on a Kibbee or netting cot, or on a cot covered with a rubber sheet so arranged as to drain into a pail or trough. Internal antipyretics are seldom well absorbed, and their depressant action is so well known as to discourage their use in place of hydrotherapy. Hutchinson, Coplin, and Bevan recommend highly the use of morphin to control the convulsions of heat-stroke. Artificial respiration in the asphyxial cases, kept up until other measures and stimulants have time to act, may be the means of saving life.

After the reduction of the hyperpyrexia the patient should be lightly covered on a cot placed in a cool place. An ice-cap should be applied to his head, and small pieces of cracked ice may be given to allay gastric irritability, with calomel to open the bowels if necessary. Albumin-water, skimmed milk, buttermilk, unfermented grape-juice, junket, and the like may be given for several days preparatory to the ingestion of heavier food. If, as sometimes happens, free diaphoresis does not come on after the reduction of most of the fever and the stimulating treatment, a hot bath may be given, and perhaps aided by the hypodermic injection of pilocarpin in urgent cases. *Sequelæ* must be treated on general principles.

The increased susceptibility to repeated attacks of insolation (after the first attack) makes it necessary to avoid exposure to heat ever after, and, if possible, to seek a cooler climate during the hot months.

PART XI.

ANIMAL PARASITIC DISEASES.

PSOROSPERMIASIS.

Psorosperms belong to the lowest form of protozoa. They are also known as *sporozoa*, and, because of their parasitic relation to cells, as *cytozoa*.

The *amœba coli* of amebic dysentery belongs to the protozoa. Blood parasites (*hematozoa*), as the *plasmodium malaria*, are likewise closely related to the sporozoa.

Various coccidia may occur in man to produce the disease indicated by this heading. The *coccidium oviforme* of the rabbit is the commonest variety, being found also in rats and mice. It escapes from the livers of the latter animals and passes into the dejecta; it produces an hepatic disease in which there are numerous whitish nodules studding the liver. These range in size from a pinhead to a split pea, and on section disclose a bile-duct, the dilated portion of which forms the nodule. The ovoid coccidia are found in the epithelial cells of the walls of these biliary expansions.

The *coccidium perforans* and *coccidium bigeminum* are found in the cells of the intestinal villi instead of in the liver of the hosts mentioned above. Among veterinarians a common form of sickle-shaped organism is known that is found within an ovoid body in the sarcolemma of the pig's muscle—(*i. e.* the so-called Rainey's tube).

In man, hepatic disease similar to that found in the rabbit is produced by the *coccidium oviforme.* The tumors formed by the coccidia may be palpable, and the liver may be quite tender. Some chilliness and fever, malaise, and stupor passing into coma have been observed. Death was caused on the fourteenth day in a case admitted to St. Thomas's Hospital (Osler). The necropsy showed whitish neoplasms in the peritoneum, omentum, and kidneys.

In the intestinal variety of *internal psorospermiasis* nausea and vomiting, diarrhea, and the typhoid state may be manifested. Involvement of the kidneys has caused hematuria and frequency of urination.

External or *cutaneous psorospermiasis*, one form of which was formerly called *keratosis follicularis*, is characterized by lesions at first of a hard, crusty, papular type, later becoming confluent, and situated on the face, lumbo-abdominal, and inguinal regions. These papillomatous growths contain numerous parasitic sporozoa.

In carcinoma, epithelioma, and Paget's disease of the nipple coccidia are readily found in and between the pathologic epithelial cells, but whether they have an etiologic bearing upon these malignant affections is still a matter of uncertainty.

Prophylaxis consists in cleanliness and care in preparing such food vegetables as spinach, lettuce, cabbage, and other greens that may possibly be contaminated by the excreta of the lower animals liable to psorosperm-infection. The **treatment** of psorospermiasis is symptomatic, though rectal injections of a solution of quinin (1 : 5000 to 1 : 1000) may be tried.

DISTOMIASIS.

(*Trematodiasis.*)

VARIOUS forms of trematodes, including the distomata, may become parasitic in man.

Distoma Hepaticum (Liver-fluke).—Among the more common varieties of trematodes or flukes, is the distoma hepaticum or liver-fluke, a parasite found in animals (horse, goat, ass, sheep, rabbit) and accidentally ingested by man.

It is almost 30 millimeters (1.1 inches) in length, and inhabits the biliary passages of the animal, and from them is discharged into the intestinal tract and evacuated with the feces. Under certain conditions of temperature and moisture, a ciliated embryo escapes from the egg, and is ingested by a gasteropod or snail (*limnæa truncatula*), in which it undergoes development into a sporocyst, that in turn gives origin to *radiæ* or parent nurses. These give birth to daughter-radiæ or *cercariæ*, which leave the gasteropod or snail and attach themselves to aquatic plants, where they are in turn eaten by animals.

Symptoms.—When present in sufficient numbers in the bile-passages the liver becomes greatly enlarged, with the occurrence of jaundice and ascites that may prove fatal. Other symptoms may also be present; thus pain was prominent in 41 out of 100 cases reported by Kurimato in Japan, and heart-murmurs were present in 42 of those cases.

Late in the disease the liver may become nodulated and terminate in atrophy.

On inspection in well-marked cases, a peculiar barrel-shaped bulging is sometimes seen, extending over the hepatic area, with tense abdominal walls over the enlarged liver. This is a pathognomonic symptom of hepatic distoma. An endemic form occurring in Japan has been described; it is characterized by marked emaciation, diarrhea, hepatic enlargement, and often by ascites.

The **prognosis** of distoma hepaticum is absolutely fatal and the **treatment** is merely palliative.

Among other trematodes may be mentioned (*a*) *distoma lanceolatum* (found also in cattle); (*b*) *distoma crassum*, which is larger in size than the preceding; (*c*) *distoma sibiricum;* (*d*) *distoma pulmonale* (*D. Ringeri*); (*e*) *distoma spatulatum* (*endemicum*); (*f*) *amphistomum hominis;* (*g*) *distoma hematobium* (Bilharz). Two of these deserve extra, though brief, mention.

Parasitic Hemoptysis (*Distoma Pulmonale*).—This is caused by the *Paragonimus Westermanii* first described by Manson (1880) and Baily (1880) in man. The lung fluke worm has also been found in the tiger (originally by Kerbert), hog, dog, and cat. The disease is extremely prevalent in certain provinces of Japan and China. Elsewhere it is usually mistaken for pulmonary tuberculosis. Stiles and Hassall[1] have discussed the whole subject. The parasite is 8 to 16 mm. long, 4 to 6 mm. broad, and 2 to 5 mm. thick. It is found encysted, usually two individuals in each cyst, with eggs, and its habitat is the lungs of mammals. It enters its final host (man, etc.) either encysted or as a free-swimming *cercaria*. *The mode of infection.*—Eastern writers look upon the drinking water-supply as the source of infection, and this view has much in its favor.

Predisposition.—Most cases occur between the ages of 11 and 30 years. It is much more common in males than females, because the former sex is more exposed to infection. Out of 58 sufferers, 38 were farmers (Stiles).

Symptoms.—In the usual form (lung infection), *cough* is common but not constant; the sputa are similar to those of lobar pneumonia, although they may be absent from time to time. Free *blood-spitting* often occurs at intervals. Jacksonian epilepsy may supervene from metastasis to the brain.

Diagnosis.—This rests upon the detection of the microscopic worm eggs in the sputum; "as many as 12,000 eggs may be expectorated daily" (Stiles and Hassall).

The **prognosis** depends upon the number of the *parasites present*, *the age of the patient* (the young and the old bearing the disease badly) and the presence or absence of complications. Pulmonary tuberculosis is an unfavorable complication.

Treatment.—Prophylaxis embraces care regarding the drinking-water, and the collection and disinfection of the sputum as in pulmonary tuberculosis. The patient should be sent to healthy non-infected areas. There is no special *medical* treatment.

Distoma Hematobium (*Bilharzia hematobia; Blood-flukes*).—This hematode is a narrow worm with anterior abdominal sucking-disks. The male is shorter and thicker than the female; the former being 4–15 mm. ($\frac{1}{6}$–$\frac{3}{5}$ in.) long; the latter, about 20 mm. ($\frac{4}{5}$ in.). It prevails mostly in Egypt, Cape Colony, and other parts of Africa, and its entrance into the human body is now believed to be through the skin of those who bathe frequently in the African rivers, in many of which it abounds. It is not unlikely that, as formerly held, infection may also occur in many cases from drinking the impure water of the rivers. The parasites or their ova are found in the bladder, the pelvis of the kidney, and the veins (especially the portal and mesenteric).

The **symptoms** are hematuria, with some pain during urination. Pus, and some of the ova of the parasites, may also be found in the urine. No serious systemic disturbances occur in bilharziosis. Prophylaxis as regards drinking and bathing in African waters should be exercised. Fouquet affirms the value of the extract of male-fern internally in this form of distomiasis.

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[1] *Annual Report of the Bureau of Animal Industry*, 1899.

NEMATODES.

HELMINTHOLOGISTS include in this class the cylindric worms, certain varieties of which are among the most common entozoa that infest the human body and inhabit the intestines.

ASCARIASIS.

Ascaris Lumbricoides (*Round-worm*).—**Natural History.**—This species resembles the common earth-worm, and is the most frequent in occurrence of all the parasites. It usually appears in children between the ages of three and ten years. The round-worm inhabits the upper portion of the small intestine, and occurs singly or in numbers. Its body is round, fusiform, and marked with fine transverse striæ. It has a yellowish or reddish-brown color, and measures in the female from 7 to 14 inches in length (17.5–35 cm.), and from 4 to 8 inches in the male (about 20 cm.), its thickness being about that of an ordinary goose-quill. The cephalic extremity of the worm has three oval papillæ, furnished with fine teeth; the caudal extremity is straight in the female and curved in the male.

Lumbricoid worms develop from ova, which are about .05 to .06 mm. long, elliptic, dark-reddish in color, and have a thick, resisting envelope. There may be sixty million of them in a single female worm, and they sometimes occur in the feces in vast numbers. The development of the embryo and worm external to the body is not accurately known. The eggs obtain entrance into the human intestine most probably through drinking-water, and it has been held that abundant mucus, and the predominating starchy and saccharine diet of which children so often partake, offer a favorable nidus for the development of the ingested ascaridian eggs.

The round-worm sometimes, though rarely, migrates from the small intestine. It has been vomited up, and it has also crawled into the pharynx, mouth, and nares, and has been withdrawn thence by the patient's fingers. It has even passed into the larynx and trachea, causing fatal asphyxia or pulmonary gangrene. The Eustachian tube and biliary ducts may be invaded with such serious symptoms as perforation of the membranum tympani and hepatic abscess. The ascarides have also been found in the peritoneal cavity, *postmortem*, with intestinal perforation, due, most likely, to other causes. They may penetrate the pancreatic duct and enter fistulæ connected with the intestine.

Symptoms may be absent, and yet the worms be found repeatedly in the stools. Existing symptoms are indefinite, and point simply to an irritative condition of the bowel. Serious symptoms may, however, result from the migration of the worm, as into the biliary passages, Eustachian tube, or larynx. Fever is not a necessary concomitant. Lumbricoid worms may give rise to any or all of the following symptoms: colicky pains, nausea, vomiting, indigestion, diarrhea (sometimes), restlessness, irritability, anorexia, itching of and picking at the nose, disturbed sleep with grinding of the teeth, salivation, and nervous twitchings. The child's abdomen and face may be swollen. Very nervous children may manifest epileptiform convulsions, choreic movements, dilated pupils, vertigo, cephalalgia, mental disturbances, and even contractures.

Complications.—The development of jaundice will indicate obstruction of the bile-duct, in cases in which the worms have been found in the feces. Intestinal obstruction from coiled worms has occurred. So also, suffocative symptoms coming on, especially at night, in a child with worms, may be due to a migrating lumbricoid. Perineal abscesses and inflamed herniæ that have perforated externally sometimes discharge the ascaris lumbricoides.

Diagnosis.—This is positively determined only by discovering the worm or ova in the stools. In doubtful cases, judged symptomatically, the administration of a suitable purgative and inspection of the resultant passages will enable the physician to arrive at a diagnosis.

The **prognosis** is good, unless serious complications arise (*vide supra*), when the case should be guarded accordingly.

Treatment.—*Prophylaxis.*—The water used for drinking-purposes should be obtained from the purest sources. That from small streams, shallow wells, and the like is most likely to contain the ova of the lumbricoides, and should be avoided. The use of filtered water should be encouraged.

Before giving an anthelmintic, it should be borne in mind that no good result can be certainly obtained unless the gastro-intestinal tract be nearly deprived of food for from twelve to thirty-six hours, so that the toxic action of the drug used may be exerted directly upon the unprotected worm.

Santonin is at once the most efficient and the most easily administered remedy. It may be given in doses of gr. ¼ to 1 (0.0162–0.0648) of the crystals to a child, or from gr. ij to iv (0.1296–0.2592) to an adult, in the form of a troche, before breakfast. A little milk or other light nourishment may be allowed, the troches being continued once or twice daily for two or three days. This treatment is to be followed by a brisk purge, preferably gr. j to iij (0.0648–0.1944) of calomel. I have sometimes combined small doses of calomel with the santonin in a troche, and with good effect. Xanthopsia or yellow vision, spasms, and even convulsions, and saffron-colored urine may follow the use of santonin in cases of idiosyncrasy or overdose of the drug. Oil of wormseed (chenopodium) in doses of five to ten drops, in emulsion, capsules, or on sugar, may also be used with benefit. Another favorite remedy with some is the unofficial fluid extract of spigelia and senna, to be given in from 1- to 3-dram (4.0–12.0) doses. Finally, the fluid extract of spigelia alone (1 to 2 drams—4.0–8.0), followed by a brisk purge, may bring away dead worms.

Oxyuris Vermicularis (*Seat-*, *Pin-*, *Thread-*, *or Maw-worm*).—**Natural History.**—The *ascaris vermicularis*, as this worm is also called, inhabits the colon and especially the rectum. It is a small worm, as several of the commonly-used terms signify, and frequently it occurs in great numbers, sometimes agglutinated with mucus into feculent balls. It is most common in children, though found not rarely at any period of life. The female oxyuris is whitish in color and about ten or twelve millimeters (one-half inch) long, the male being about three or four millimeters (about one-sixth of an inch) in length. Oxyures develop from ova in about two weeks after the ingestion of the latter. The eggs are irregularly ovoid, about $\frac{1}{500}$ in. (0.05 mm.) in length, and tenacious of life. By the time the embryos have reached the cecum, they are sexually mature, and when the female arrives in the rectum, immense numbers of eggs are deposited that mature into great numbers of worms, the latter being discharged with the feces. Sometimes the worms crawl out of the anus.

Infection with the ova may take place through water and food (green, uncooked vegetables and fruit) that have come in contact with the hands of infected persons. Scratching the anus will permit of the reception of oxyuris eggs under the finger-nails (Zenker and Heller), and in careless, ignorant, and uncleanly persons the possibility of such an auto- or re-infection should be recognized and avoided.

Symptoms.—*Pruritus ani* (itching of the anus), sometimes burning pain, and tenesmus, with restlessness and disturbed sleep, are the commonest symptoms of the presence of this parasite. The itching is always worse at night, and may be paroxysmal. An herpetic or eczematous eruption around the anus should arouse suspicion, particularly in children, of the presence of the oxyuris in the rectum, and it accounts for the intense itching (Flint). Anorexia and anemia, rectal irritability, and "nervousness" may be associated. It is believed that the migration of the worms into the vagina of girls may set up pruritus and leukorrhea, and that habits of masturbation may be induced in both girls and boys by the sexual irritation caused by the worm. Inspection of the stools will reveal, in positive cases, the whitish, thread-like parasites.

Diagnosis.—The pruritus, indicating rectal trouble, will direct the physician's attention to the anus, where the oxyures may be seen; if not found, their discovery in the feces or the discovery of the eggs by microscopic examination will suffice.

The **prognosis** is good, and proper treatment is always effective.

Treatment.—The exhibition of anthelmintics and purgatives, such as recommended for destroying and removing the lumbricoid worm, may be effective against seat-worms also, but mainly in reaching those lodged in the bowel above the rectum. Attacking the oxyures directly, however, by means of enemata is the most useful and rational treatment.

The rectum should be well emptied of feces, so that the worms may be exposed to the action of the medicament injected, and for this purpose enemata of cold water, either simple or with salt or soap, may be resorted to. Injections containing the decoction of quassia (1 or 2 ounces—32.0 to 64.0—of the powder or chips to the pint—half liter—of water) are nearly always curative. Other useful remedies are carbolic acid, turpentine, tannin, vinegar, camphor, potassium sulphid, and the oil of eucalyptus. The injections should be repeated once or twice daily for at least ten days. It sometimes happens that killing the worms as directed above affords only temporary relief. The reason for this is obviously to be found in the fact that the oxyuris breeds in the cecum, and that only grown forms descend, reaching the rectum.

Rectal irritation may be allayed by injections of laudanum and starch-water (gtt. iij–v to the ounce—32.0). Anal itching is often amenable to carbolized vaselin, applied at bed-time, or to belladonna ointment, or the following, which has been highly recommended:

℞. Hydrarg. chloridi mitis, ℈ij (2.592);
Petrolati, ℥ss (16.0).
M. et ft. ung.
Sig.—Apply at bedtime.

Ascaris Alata.—This is another name for the *ascaris mystax*, a species of worm found in the intestines of the dog and cat, and occasionally in man. It is a slender worm, with a closely-rolled spiral tail and a wing-like projection on either side of the head. The female is about 6–7 centimeters (2.7 inches), the male about 4 centimeters (1.75 in.) in length. Scarcely ten instances, however, have been recorded in which this parasite has occurred in man.

Trichocephalus Dispar (*Ascaris trichiura*).—**Natural History.**—This worm measures about four or five centimeters (2 inches) in length, and is characterized by the very slender, hair-like appearance of the anterior two-thirds of its body, in contrast to the thick posterior portion, which is more or less straight and blunt-pointed in the female, but rolled into a spiral in the male. Its particular habitat seems to be the cecum, though sometimes it is also found in the colon. It may exist in great numbers. Europeans appear to be infected with the parasite more commonly than Americans. The trichocephalus has been found *postmortem* in many subjects dying with various diseases, as typhoid fever (Flint), meningitis (Barth), profound anemia (Osler), and beri-beri.

Propagation is effected by the microscopic eggs, which are ovoid, hard, nodular, brownish, and about 0.05 mm. ($\frac{1}{500}$ in.) in length.

Symptoms.—It is not certain that the parasite causes any symptoms, nor even that it aggravates those of an associated disease (*vide supra*). When occurring in great numbers the possibility of fecal accumulation may be mentioned.

The **diagnosis** may be made by microscopy. The ova may be detected in the feces.

The **prognosis** and **treatment** are not called for.

ANKYLOSTOMIASIS.

Ankylostomum Duodenale (*Dochmius duodenalis*).—**Natural History.**—This parasite belongs to the family of *strongylidæ* of the nematoid worms. It was discovered in Milan, in 1838, by Dubini. The length of the female is from 8 to 18 mm. ($\frac{1}{2}$ inch), and of the male from 6 to 10 mm. ($\frac{1}{3}$ inch). Its body is thread-like, with a conical-shaped head, and a large, bell-shaped mouth surrounded by a horny capsule, and possessing four hook-like teeth, ventrally situated, and two smaller, vertical teeth on the dorsal side, by which the worm fixes itself to the mucous membrane. A bulbous-like swelling exists at the tail end of the male worm. It inhabits the jejunum and duodenum. The eggs are found in muddy water, and there liberate the embryos. These develop into larvæ, which, when taken into the human bowel through drinking-water develop into mature worms. They do not multiply within the intestine.

Pathology.—The ankylostomum is nourished by the blood it sucks from the intestinal vessels. It is found *postmortem*, sometimes, in the mucous or even submucous coat, rolled up in a little blood-cavity. Ecchymoses, containing a central opening through which blood can ooze, are the usual result of the worm's action. Chronic catarrhal enteritis is usually associated. Hypertrophic dilatation of the heart is observed.

Symptoms.—The chief symptom of the condition is anemia (secondary). When the number of ankylostoma embryos introduced into the intestine is large, the anemia may develop acutely; when but a few are introduced, the withdrawal of blood is more gradual, and chronic anemia develops. I think, however, it may be safely affirmed that the anemia is not wholly due to blood-sucking. In some cases the impoverishment of the blood has been so profound as to simulate a pernicious anemia.

This parasite has been found to be the cause of the disease known as "Egyptian chlorosis," first described by Griesinger. Ankylostomiasis is not uncommon in tropical countries (Italy, Brazil). In Italy it has been termed *tunnel* or *mountain anemia;* in Belgium it is known as *brickmaker's anemia;* again, it occurs among workers in coal-mines—*miner's cachexia.* In this country it is rare, though alleged to have been seen in the Southern States. The importation of infected Italian, Hungarian, and Polish laborers may, at some future time, cause the propagation of the ankylostoma parasite in the United States. The anemia of ankylostomiasis is progressive, and it is noteworthy that no organic cause for it can be discovered. There may be in addition, slight gastro-intestinal disorder (anorexia, colicky pains, nausea and vomiting, and constipation alternating with diarrhea). In cases marked by an acute development of anemia considerable general weakness, dyspnea and sometimes dropsy may ensue. The areas of the apical cardiac impulse and of cardiac dulness are increased downward and laterally. Various murmurs—hemic—may be heard, and the pulmonic sound may be accentuated (*vide* Pernicious Anemia, p. 433).

Diagnosis.—This is made by finding the eggs or mature worms in the feces. The former are oval-shaped, about 0.05 mm. ($\frac{1}{500}$ inch) in length, and have a much thinner shell than the ova of the round-worm. They do not segment except within the intestine. In any case of pronounced anemia in which the cause is obscure the patient's dejections should be carefully examined for the ankylostoma parasite or its eggs.

Duration.—The disease may last for months or for several years.

Prognosis.—If left untreated, the affection may end fatally. Intense anemia, obstinate diarrhea, and profound nutritive disturbances constitute symptoms of grave import. Properly treated, the prognosis is quite favorable. A spontaneous cure may occur in some cases.

Treatment.—*Prophylactic.*—Workmen in mines, tunnels, and brick-yards, and in tropical localities especially, should be warned not to drink the water close at hand without previous boiling and then cooling.

Medicinal.—Anthelmintics to kill the ankylostoma and purgatives to remove it from the intestine are indicated as for other intestinal parasites. The oleoresin of male fern in ½- to 1-dram (2.0–4.0) doses, santonin, and thymol are very useful for the first-named object.

Cathartics or enemata are used to bring away the dead parasites, after which nourishing food, iron, and tonics are to be given.

TRICHINIASIS.

(*Trichinosis.*)

The parasite that gives rise to this affection is the *trichina spiralis.*

Natural History.—The mature male worm is 0.8 to 1.5 mm. ($\frac{1}{2}$ in.) long and the female 2 to 4 mm. ($\frac{1}{12}$–$\frac{1}{6}$ in.). The head is pointed and unarmed, and the neck is long and more slender than the body, which has a round blunt end. The worm is viviparous. It inhabits the intestines of such animals as the rat, dog, cat, hog, and man.

The embryo or muscle trichina is about 0.6 to 1 mm. ($\frac{1}{25}$ in.) long, and lies coiled up in a spiral form within an ovoid capsule in the sarcolemma-sheath of muscle-fiber. The life-history begins with the larval state of the trichinæ encysted in the muscles. When this flesh is eaten by another animal, or by man, the larvæ are liberated during the digestive process. Passing into the intestines, they reach the adult stage in from two to four days, being then sexually mature, and in five to seven days more they produce hundreds of living embryos.

The intestinal trichinæ become fully grown, and then usually die in from four to five weeks. The female trichina may bring forth several broods of embryos during her life-period in the intestine. The living embryos leave the intestine at once, and invade the muscles through various channels—principally along the connective-tissue routes—so that the symptoms of muscular irritation develop in from seven to ten days after eating the trichinous meat. The embryos attain to maturity (larval form) in about two weeks after entering the muscular tissues. Their presence causes a mechanical irritation that results in the formation of a fibrous capsule in from four to six weeks. In man it probably becomes encysted at a later period than in the lower animals, as shown by the accompanying illustration, taken from a case under the immediate observation of Dr. L. Napoleon Boston (Fig. 79). Usually but a single worm is found within one capsule, though occasionally three or four are seen. Leuckart found numbers of embryos free in the abdominal cavity of infected animals; they have also been found in the mesentery. The encapsulated trichinæ may live many years in the muscles. With increasing age the capsules become thicker and may be the seat finally of calcareous infiltration.

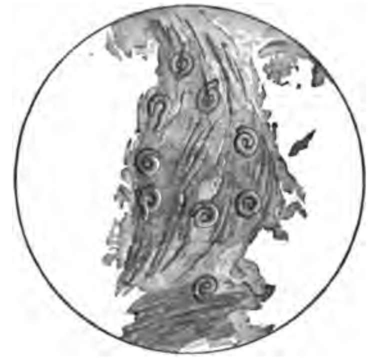

FIG. 79.—Trichina spiralis from the head of the right gastrocnemius muscle three weeks after the first symptoms appeared (Queen obj. $\frac{1}{4}$; eye piece No. II).

Pathology.—The diaphragm is most thickly infested with the larval trichinæ. Next in order are such trunk-muscles as the intercostals and abdominals, then the muscles of the neck, including the larynx, head, eyes, and extremities. Up to the seventh week of the disease the intestinal trichinæ may be very numerous, as many as a dozen being found in a drop of intestinal mucus. There may be some intestinal inflammation (catarrh) and the mesenteric glands may also be swollen and appear like those of typhoid fever. In cases that proved fatal during the second month, Cohnheim noted an abundance of fat in the liver, a granular state of the renal epithelium and of the heart-muscle, broncho-pneumonic areas (occasionally), and hypostatic pneumonia (frequently). *Microscopically*, the muscles show "the changes characteristic of acute myositis" (Fitz) after the fifth week. The trichinous cysts in the muscles may be seen with the naked eye as small, grayish-white, opaque, "oat-shaped" specks, longitudinally disposed in the meat-fibers.

Sources of the Trichina.—The trichina was first found in pork—the usual source of trichiniasis in man—by the late Joseph Leidy. It should be noted that some individuals may be dangerously infested with trichinæ and yet give no symptomatic evidence of the presence of the parasite. Recent investigations show that the live trichinæ may be found in the fatty as well as the fleshy portion of pork. The pig is infested by eating trichinous rats, trichinous pork, or human or porcine excrement containing the embryos of propagating intestinal trichinæ. The rat may be the original host of the parasites, or it may itself become infected by older rodents eating their fellows, or by eating trichinous pork or human or porcine excrement voided during the stage of intestinal infection.

As to the frequency of the infection of hogs, it may be said that about 2 per cent. were found to be trichinous, according to Salmon's report (1884), of nearly three hundred thousand examinations of American pork. Other examinations, however, show a variation of infection of from .05 to 6 per cent. of hogs. In Prussia, according to Eulenberg's statistics, the ratio is decidedly less varying—from 1 to 2160 hogs (1876) to 1 to 1817 (1889). "The dissecting-room and postmortem statistics show that from one-half to two per cent. of all bodies contain trichinæ" (Osler).

Of course, man, as a rule, becomes infected by eating raw or partially cooked pork containing living muscle-trichinæ (larvæ). Eating raw ham and sausages, a habit common among the Germans of Prussia (particularly during picnics), and in some parts of the United States where German emigrants have settled in large numbers, explains the comparative frequency of this disease in such localities. Trichiniasis has occurred in epidemic form in North Germany, France, Spain, Russia, the Scandinavian countries, and in several of the northwestern United States.

Symptoms.—*Postmortem* examination often reveals the presence of muscle-trichinæ, whereas no history of trichiniasis or of any disease resembling it has been obtainable. It is to be recollected that to the migration of the parasites the principle symptoms of trichinosis are due.

In well-marked cases of infection *gastro-intestinal disturbances* appear on the second or third day after the ingestion of the affected meat. Vomiting, diarrhea, and colicky pains in the abdomen may be present. The diarrhea sometimes takes on the characteristics of a choleraic attack or may be followed by obstinate constipation.

Extreme "muscular weariness" and bodily fatigue often occur for several days before the embryonic parasites can have begun to wander into the muscles. On about the tenth to the fifteenth day, when migration usually commences, *chills*, followed by a temperature of 101.5° to 104° (38.6° to 40° C.) and marked *myositis*, come on. The muscles are stiff, tense, painful on pressure and motion, and somewhat swollen. The flexors of the extremities are particularly sore and often firmly contracted, causing the knees and elbows to be acutely bent. *Mastication*, *deglutition*, and *phonation* may be difficult and painful because of the involvement of the muscles of the jaws, pharynx, and larynx. Intense *dyspnea* is frequent on account of the involvement of the diaphragm and intestinal muscles. The *temperature* shows marked remissions in most cases, and may even be subnormal. The *pulse* varies with the temperature.

Edema is characteristic in nearly all of the cases. It appears on about the seventh day after the infection, and begins in the face, usually being noted first in the eyelids, and extending thence to the extremities and trunk during the height of the muscular symptoms. It may last for several days, then disappear for several days or a week, and reappear. Ascites even has been observed. Edema of the larynx and bronchial catarrh, the latter rarely leading to broncho-pneumonia, may also supervene and add to the gravity of the dyspnea. Profuse sweating may last for several weeks. Miliaria, urticaria, acne, furunculosis, herpes, and pruritus may occur as skin-manifestations. Insomnia, headache, a temporary loss of the tendon-reflexes, and dilatation of the pupils (Rupprecht) have been noted among the nervous symptoms. Prolonged cases show a marked degree of emaciation and anemia. T. R. Brown[1] found a decided increase of the eosinophiles in the blood, amounting to 37 per cent. This discovery has been confirmed by Cabot, Gwyn, Atkinson, Stump, Osler, and others. W. T. Howard, Jr.,[2] was able to confirm Brown's observation on the presence of eosinophiles in the muscle-lesions, but failed to find an increase of these cells in the circulating blood.

Complications, as a typhoid state, hypostatic pneumonia, and pleurisy may appear. Albumin, with casts, and occasionally red and white corpuscles are found in the urine.

Recovery is effected in mild cases within two weeks; in the severe cases of infection from six weeks to several months may be occupied before convalescence begins.

Diagnosis.—The following symptoms are regarded as pathognomonic: sudden swelling of the face, coming on after the patient has suffered for several days from muscular soreness; loss of appetite, fever, and profuse sweats (Böhler); painful, tender, and "rubber-like" hardness of the muscles, with difficulty in movement; semiflexed extremities; gastro-intestinal catarrh, with a red, dry, coated tongue; dyspnea, diarrhea, and edema of the extremities following the subsidence of that first noticed in the face. Friedreich emphasizes hoarseness, and Packard rapidity of respiration without evident cause, as diagnostic symptoms.

Meat- and *sausage-poisoning* may be distinguished from trichiniasis by the more rapid course of the former, the dry throat and skin, jaundice, visual disturbances, and the absence of edema and muscular symptoms.

Direct examination of the passages and of the muscles may be resorted to. The discovery of the parasites in the pork a portion of which has been eaten by the sick of course establishes the diagnosis. A low-power microscope should be used to examine the intestinal mucus for the trichinæ. Light purgation should precede this endeavor. Harpooning such muscles as the biceps for the purpose of removing some muscle-fiber, or directly incising a small portion under Schleich's method of infiltration-anesthesia, may permit of a positive diagnosis in some cases.

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[1] *Johns Hopkins Hospital Bulletin*, 1897, vol. viii.

[2] *Philadelphia Medical Journal*, December 2, 1899.

Acute rheumatism, *cholera*, *typhoid fever*, and *acute polymyositis* (*pseudo-trichiniasis*) may at times resemble trichiniasis. Epidemics of the parasitic disease are more readily diagnosed than an isolated case.

Prognosis.—This depends upon the number of parasites ingested with the infected meat or sausage, and upon the number of embryos generated in the intestines by the matured worms. Marked early diarrhea is favorable. The prognosis should be guarded, as death may occur as late as from the fourth to the sixth week. Of 357 cases collected by Packard the mortality was 24.07 per cent.

Treatment.—Prophylaxis is of supreme importance, both as to the infection of the hog and the danger of eating infected pork. Care should be exercised in the feeding of swine, and the destruction of rats should be made as complete as possible in and about the styes. Pig-excrement should be removed and burned, and feeding with milk, bran, grain, and vegetables should be forced upon all keepers of swine.

Rigid inspection of the meat-supply, as is done in Germany, should be carried out by sanitary officers employed by the government. It is held to be an impracticable measure where immense quantities of meat are handled daily to examine microscopically simply a minute fragment of pork taken from each hog slaughtered. Decidedly the safest and most efficient way to prevent trichinosis is to thoroughly salt, smoke, and cook the pork that is to be used. Roasting should be particularly well done, in order that the heat may effectively reach the central portions of the meat. Putrefaction does not kill the parasites.

The treatment of those who have eaten trichinous meat should be by a prompt evacuation of the bowel, especially within the first twenty-four hours, as after the embryo young have been brought forth and have passed into the muscles no known treatment is successful in attacking them. Calomel is one of the best drugs, and active purgation usually follows its use in large doses, succeeded by salines; rhubarb, senna, sulphur, aloin, and large doses of oil or glycerin may also be tried. In combination with the purgatives some anthelmintic (male fern, santonin, thymol) should be used. The encysted or larval parasites are not accessible to treatment, although picric acid has been recommended. The symptoms to be met are the great muscular pains, insomnia, and weakness, which is often severe in protracted cases. Prolonged hot baths, anodyne embrocations, with hypodermics occasionally, may prove useful for the first; bromids, chloralamid, and the like for the second symptom; and a concentrated liquid diet, strychnin, peptonoids, and the like for the last. Massage, electricity, and stimulating applications, as chloroform liniment, may be required during convalescence and for some time thereafter to combat the muscular weakness, soreness, and stiffness.

FILARIASIS.

(*Filaria Sanguinis Hominis.*)

There are several varieties of filariæ that may be found in human blood. The two principal ones are the *filaria sanguinis hominis nocturna* and *filaria sanguinis hominis diurna.* The first is a white, opaline, thread-like worm, tapering toward the ends, which latter, however, are blunt. The male is 83 mm. (3.2 inches) long; the female 155 mm. (6.1 inches). The second worm is known only in embryonic form, and is distinguished by granulations in the axis of the body. Manson found them in the blood of Congo negroes, but only during the daytime. On the other hand, the nocturnal filaria is found only at night, or, if the host be either by habit, necessity, or choice, a day-sleeper, during this time, showing, then, that there is some condition of the body during quietude that is conducive to the appearance of the filaria in the blood (blood and chyle flow, Granville). This "filarial periodicity" is a curious and striking characteristic of these parasites.

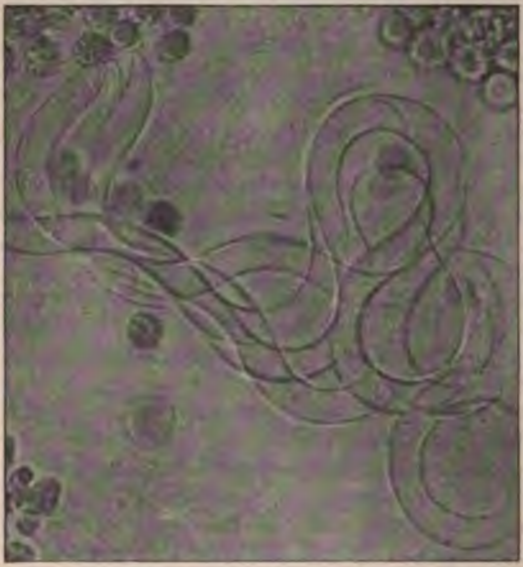

FIG. 80.—The movement of a single filaria during a series of four successive instantaneous exposures. The length of each exposure was one-fifth of a second, the entire series occupying less than five seconds. The magnification is to eight hundred diameters, with a Zeiss one-twelfth homogeneous immersion lens (F. P. Henry).

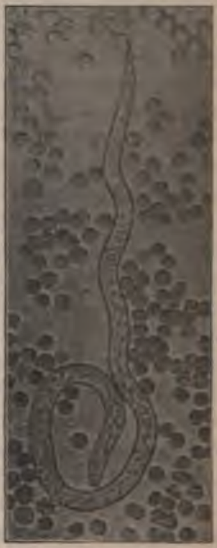

FIG. 81.—Filaria alive in the blood. Instantaneous photomicrograph. Four hundred diameters magnification. Four millimeters Zeiss apochromatic (F. P. Henry).

The embryos are produced by the female in great numbers, and are so small that they readily pass through the capillaries. According to Manson, who, in 1877, found the larvæ of filaria sanguinis hominis in the stomach of a female mosquito, it is probable that after filling itself with the blood of an infested man during sleep, the mosquito seeks stagnant water, dies, and the larvæ are set free. In this way it may happen that man takes in the embryos through the drinking-water. They find a permanent seat in the lymphatics of the human host, mature, and bring forth young, which may again infest the blood by passing through the lymph-ducts into the thoracic duct and general circulation.

The geographic distribution of the filaria is limited mainly to the tropics and sub-tropics. Filariasis is most common in Brazil, the West Indies, Mexico, the Southern States, Southern China, India, Egypt, a part of Australia, and the southern Pacific islands, where it is quite endemic.

The **symptoms** of filariasis are in abeyance until some obstruction of the lymph-channels is caused by the parasite. There are several conditions or endemic diseases produced. **Elephantiasis arabum** is believed by Manson to be the effect of these parasites in a certain proportion of cases at least. In specimens of night-blood from 88 Cochin Chinese he found filariæ in 21; 14 specimens came from patients with elephantiasis, and only 1 showed filariæ. This latter fact, he explains, is to be expected, since, in order to give rise to elephantiasis (due to an infarction of the lymphatic glands connected with the diseased areas), the adult filariæ must lie on the distal side of the glands, which makes it impossible for the young filariæ to pass into the general circulation. "Therefore the person least likely, in a filarial district, to have filariæ in his blood is one who is the subject of elephantiasis."[1]

Hematochyluria and Chyluria.—The patient passes a white, opaque, milky urine, occasionally bloody, with a clotty sediment. This may be intermittent, and normal urine may be passed for many weeks before chyluria or hematochyluria reappears. There may be at the same time a slight degree of polyuria. Under the microscope, fat granules and white and red corpuscles are seen. The lively, wriggling embryo filariæ may also be discovered in the urine, as well as in the blood at night. There is a dilatation of the lymph-vessels in the kidneys alongside of the tubules, and in the abdominal lymph-plexuses. Sometimes a little vesical irritation and straining during urination may be caused by the endeavor to pass chylous blood-clots. The thoracic duct above the diaphragm has been found impervious (Stephen Mackenzie).

Lymph-scrotum and lymph-vulva have been caused by the filariæ. The parts are greatly swollen, thickened, and contain distended lymphatics filled with a turbid and either milk-white, salmon-colored, or blood-red coagulable liquid that is discharged upon puncturing the varices. The filaria is not always found in the exuded lymph. The inguinal and femoral regions are often enlarged and doughy. An erysipelatous inflammation of the parts is not infrequent in these cases, and may be ushered in by a chill and high fever, lasting a day or two, and ending with a profuse sweat.

The filariæ have also been found in a case of ascites (Winckel), in one of hemoptysis (Yamane, Japan), and, by the same observer, they were found in the feces (chylous diarrhea).

Treatment.—Prophylaxis in regard to the drinking-water is essential in order to avoid filariasis. Filtering, boiling, and storing the water in mosquito-proof receptacles is sufficient. Thymol in from 1- to 5-grain (0.0648–0.324) doses, given for from two to eight weeks, has caused the disappearance of the larval filariæ in several cases. Methylene-blue appears also to have produced a cure in a case of chyluria reported by Flint, although Laveran and Henry believe that it is of little value. The latter states that he has "given this drug in larger doses than were used in the case reported by Flint, and for a much longer period, without the slightest effect upon the parasite."[2] The adult filaria seems to be beyond the reach of any known medication that will not prove dangerous, either directly or indirectly, to its human host.

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[1] *British Med. Jour.*, June 2, 1894.

[2] *Med. News*, May 2, 1896.

DRACONTIASIS.

(*Guinea-worm Disease.*)

The parasite is the *filaria* or *dracunculus medinensis* or *persarum*, common in the tropics of Asia, Africa, and America. It is only recently that the male guinea-worm has been found. It is usually solitary, and measures from 50 to 100 cm. (20 to 40 in.) in length and about 2 mm. ($\frac{1}{12}$ in.) in diameter. It is cylindric, whitish, with blunt papillated head, and a sharp, curved tail. The body is nearly filled by the uterus, which contains innumerable embryos. The live young dracunculus escapes from the intestines of an infested man, ox, horse, dog, or jackal, enters the body of a cyclops or small cray-fish, and there becomes a fully-developed larva. It is then taken into the stomach and intestines of man through the contaminated drinking-water. The female enters the intestines by way of the mesentery, and the male worm, after fulfilling its sexual functions there, probably dies, while the female brings forth its young, which pass into the connective tissues of its human host. The worm has an inexplicable affinity for the subcutaneous and intermuscular tissues of the feet and legs, where it attains full development.

Symptoms.—Wherever the parasite is situated, it may often be felt coiled up under the skin, which at that point becomes red, sore, and fluctuating like an abscess. When opened, either surgically or naturally by the worm, the head appears through the aperture. The favorite spot for perforation is the dorsum of the foot, though sometimes it extrudes from the legs, occasionally from the thighs, and very rarely from the thorax and abdomen.

Treatment.—Prophylaxis in regard to the drinking-water and as to bathing where the intermediary host of the dracunculus—the cyclops—has its habitat is essential for safety.

The active treatment embraces the surgical measures necessary to remove the worm entire and to promote the healing of the irritated tissues. The burrow should be opened, and the worm gradually coiled around a quill or a smooth, cylindric piece of wood until it can be withdrawn without being torn and allowing any embryos to escape into the tissues. Roth claims that after incision the application of compresses of carbolic acid (1 to 15) over the wound causes the worm to be removed in two or three days. Native Indian physicians commend highly the local application of the leaves of the "amarpattee" plant. Asafetida and sulphur have been recommended internally, but without any definite result.

OTHER FILARIÆ.

Among other filariæ that have been found in man are the following: The *filaria immitis*, which causes hematuria and has been found in the portal vein, whilst the ova were discovered in the ureteral and vesical walls; *filaria labialis*, found in a lip pustule; *filaria lentis*, found in a cataract; *filaria trachealis* and *bronchialis*, seen in the trachea, bronchioles, and lungs; *filaria hominis oris*, observed by Leidy in the mouth of a child;

filaria loa, noticed in the tropics among negroes, its habitat being beneath the conjunctiva.

OTHER AND UNCOMMON NEMATODES.

Eustrongylus Gigas.—This parasite is exceedingly rare in man, but has been found in many of the carnivora and in some herbivora. It is supposed that fish act as the intermediate host for the larvæ. The worm is enormous in size, the female being from 25 to 100 cm. (10 to 40 in.) in length and from 5 to 12 mm. ($\frac{1}{5}$ to $\frac{1}{2}$ in.) long. It is a red, cylindric parasite with blunt-pointed ends. Its most common seat is the kidney, which it may destroy, causing hematuria and, perhaps, the presence of the eustrongylus ova.

Strongylus paradoxus has been found in the respiratory organs of the pig and in the dejecta of a pork-dealer.

Anguillula stercoralis or **intestinalis** occurs in the stools of certain tropical endemic diarrheas. The parasites are oviparous, and the eggs may be taken through the drinking-water. They have been found in the biliary and pancreatic ducts, as well as in various parts of the intestines. Boiling the water as a prophylactic measure and the administration of thymol or male-fern are to be recommended.

Echinorhyncus gigas belongs to the *Acanthocephala* (thorn-headed worms) and infests the intestines of the pig. The larval host is the cockchafer or floral beetle grub. In the only case reported, that of a boy (Lambl), a small echinorhyncus was found in the intestines.

Echinorhyncus moniliformis occurs in rats, and one case, that of a Sicilian, has been reported by Calandruccio, in which the ova were found in the feces. The larval host is probably the *Blaps micronata.* The ethereal extract of male-fern causes the expulsion of the parasite.

CESTODES.

ECHINOCOCCUS DISEASE.

(*Hydatid or Bladder-worm Disease.*)

THE tænia echinococcus is also called *tænia nana* by Van Beneden, but should not be confounded with the tenia nana of v. Siebold, a brief description of which follows this article. It is the smallest tape-worm of our domestic animals, and lives between the villi in the small intestine, especially in the larger breeds of dogs, as the mastiff and Newfoundland. It has a length of from 4 to 9 mm. ($\frac{1}{5}$ to $\frac{1}{3}$ in.), and consists of only three or four sections, the last one of which is mature. The rostellum projecting from the small head has thirty or forty hooklets arranged in a double row. Hundreds and sometimes thousands of eggs are contained in the mature segment. The intermediary hosts for the larvæ are rarely man, the horse, and the sheep, and more often the hog and ox.

Life History.—The ova, embryos, or the proglottides even, of the adult tenia are voided by the dog, and in various ways, to be pointed out later, are ingested by man. The dog first becomes infected by eating the bladders or echinococcus cysts of some animal that harbors the larval form of the tenia, and the matured teniæ appear in from eight to ten weeks. The liberated six-hooked embryos burrow through the intestinal wall or enter the portal vein; they then pass into the solid viscera, as the liver, into the peritoneal cavity, the muscles, lungs, brain, etc. There they develop into the larval form and cause the formation of hydatid or echinococcus cysts. During the latter process the hooklets disappear.

In the development of echinococcus cysts, about four weeks after the ingestion of the bladder-worm eggs, small nodules appear, about 1 mm. ($\frac{1}{25}$ in.) in size. In about five months the cyst-walls consist of two layers, an external layer and an inner, granular, parenchymatous layer (or endocyst), containing a clear liquid. As the reaction to the irritation caused by the parasite and its cyst increases, a fibrous investment forms around them. At this time, also, small daughter-cysts, or vesicular buds, form the minor granular layer of the mother-cyst, and contain the heads of the larvæ. They are soon set free, and may themselves give rise to other or granddaughter-cysts in a similar way. These really become the breeding capsules of little cellular outgrowths that form the scolices or heads of future teniæ. They show the four sucking disks and a circle of hooklets. Each scolex, when taken into the intestine of the dog, develops into an adult bladder-worm or tænia echinococcus. This endogenous mode of cystic growth is common in man (*E. hydatidosus*); but in some of the lower animals, and rarely in man, the daughter- and granddaughter-cysts may develop between the two layers of the primary or mother-cyst, and then extrude (*exogenous* variety; *E. granulosus*). A third variety is the multilocular echinococcus (E. alveolaris, Buhl), affecting principally the liver. A large, hard tumor is seen that on section shows a firm connective-tissue framework surrounding alveoli that average a small pea in size. These alveoli contain small echinococcus cysts with thick, laminated walls. They may contain scolices or hooklets, and sometimes they are quite sterile. The echinococci may be situated in the lymph-channels and bile-ducts (Zenker).

The pure hydatid fluid is colorless, odorless, limpid, neutral in reaction, and has a specific gravity of 1005 to 1012. About 96 to 98 per cent. is water, and sodium chlorid, carbonate, and sulphate, traces of sugar (dextrose), and uric acid are found among the constituents.

Among the changes that an echinococcus cyst may undergo the commonest is that of the *death of the echinococci*, as from diminished nourishment due to intense proliferation of daughter- and granddaughter-cysts. The contents become thickened, putty-like, or granular, and even calcified. Remnants of these obsolete cysts, such as the chitinous substance of the old and outer wall-layer and hooklets, may be found. Traumatism or chemical irritation may also cause the death of the echinococcus and obliteration of the cysts. Sometimes *rupture* of the cyst occurs, with serious consequences to the patient; on the peritoneum daughter-cysts or free scolices may be disseminated and grow. Or *perforation* into the respiratory, digestive, or urinary tracts and discharge of daughter-cysts and hydatid fluid may take place. Lastly, *suppuration* and the formation of large hepatic abscesses may ensue, either spontaneously or on account of septic instruments used for tapping the cysts.

Etiology.—Carelessness in the feeding and the keeping of dogs is the primary source of hydatid disease, and the preparing of food where dogs are allowed to roam about, to be petted, and so on, accounts for the majority of cases. Females are more often affected than males, and children and young adults seem to be oftener affected than those older in years.

As regards the *geographic distribution*, echinococcus disease prevails most extensively in Iceland, where man and dog live closely together. In Australia, also, many persons are affected. It is not so common in Europe, Asia, or Africa, and in America it is rare.

Organs Affected.—The tenia echinococcus has an undoubted predilection for the liver. Next in order of frequency are the lungs, intestines, perhaps the urinary organs, brain, and spinal cord: The spleen, bones, muscles, the heart, and blood-vessels are involved with uncertain frequency.

Symptoms.—**Hydatids of the Liver.**—Unless the cystic tumors compress the portal area or the biliary passages, or invade the neighboring viscera, subjective symptoms may be entirely wanting. Not infrequently echinococcus sacs, partly calcified, have been found *postmortem*, not having produced any symptoms during life. Gradual but progressive loss of flesh and strength with the presence of a fluctuating tumor may be the only symptoms present until late in the disease. If the cysts attain a large size, a sensation of dragging, and of pain even, is often present; as a rule, however, pain is absent throughout the course of the disease. If the tumor displaces the diaphragm upward and compresses the lung, cough and dyspnea result. In some cases the sac has ruptured into the bronchi, and given rise to cough and to expectoration of the fluid and vesicles.

If the portal veins and bile-duct are compressed, splenic enlargement from passive congestion, ascites, and jaundice will occur, these symptoms being more common when the cysts are multilocular. Rupture may occur into the intestines (colon), into the pleura or pericardium, causing pyothorax or pyo-pericardium, or into the inferior vena cava, causing fatal pulmonary embolism.

Fever is usually absent throughout, unless the contents of the sac become converted into an abscess; then rigors or chills, fever (hectic in type), and sweatings occur, with jaundice (more or less intense) and rapid emaciation.

Not infrequently the cyst-wall becomes partly calcified and the contents are reabsorbed, with an entire absence of symptoms, the patient dying in after years of some intercurrent disease.

When rupture occurs, unless the contents be evacuated through the respiratory or alimentary tract or externally, symptoms of collapse develop and are followed by death.

The **physical signs** give on *inspection* fulness or bulging in the right hypochondriac region, especially if the cyst be single, of large size, and situated anteriorly.

Palpation confirms inspection and shows a fluctuating mass or masses. A trembling impulse is felt sometimes on deep palpation, aided by light percussion over the opposite side of the cyst, constituting the so-called "hydatid thrill." This sign cannot always be elicited, but when present is pathognomonic of the disease. The remainder of the liver shows uniform enlargement. The spleen is often palpably increased in size from passive congestion.

Percussion reveals, in addition to the hydatid fremitus, an increased area of dulness to the left or posteriorly, depending on the location and extent of the growths. If the left lobe be involved, the line of flatness may extend across the sternum to the left hypochondriac region. If the cysts are multiple and on the antero-inferior surface, the stomach may be displaced toward the left and dulness may extend across the epigastrium; if posteriorly, the pleural cavity may be encroached upon, causing an increased area of flatness upward in the postero-axillary line. Frerichs claims the line of dulness posteriorly in hydatid disease to be a curved one, whose convexity is upward.

Auscultation gives, according to Santoni and others, a short sharp booming sound when the tumor is percussed, that may be likened to one produced by striking a membrane stretched over a metallic frame.

Diagnosis.—In the entire absence of subjective symptoms and of characteristic physical signs, the diagnosis is impossible. If, however, the cyst be of sufficient size to give fluctuation and the liver be irregularly enlarged, with an absence of fever, pain, and marked emaciation, the disease may be strongly suspected. The only certain demonstration of the condition is the discovery of the characteristic hooklets in the aspirated or discharging contents of the cyst. Among the conditions that may be misdiagnosed for hydatid disease are—(*a*) Dilatation of the gall-bladder, (*b*) hydronephrosis, (*c*) right-sided pleurisy with effusion, (*d*) syphilis of the liver, (*e*) carcinoma, (*f*) abscess, and (*g*) cirrhosis.

Hydatid Cyst.	Dilatation of the Gall-bladder.
Previous history negative, except the companionship of dogs.	A previous history of having passed biliary calculi is often present.
Pain and jaundice usually absent.	Attacks of biliary colic followed by jaundice either are present or enter into the previous history.
Enlargement in any direction, depending upon the location of the cysts.	Enlargement is always in one direction—downward and posteriorly.
Hydatid thrill may be present.	"Hydatid fremitus" never present.
Less so.	The tumor is somewhat movable.

Hydatid Cyst.	Hydronephrosis.
The history is negative (*vide supra*).	There is a history of renal calculi or of vesical inflammation.
Urinalysis is negative.	Urinalysis reveals evidences of renal disease.
The tumor is most prominent over the hepatic area, and is associated with enlargement of the liver.	The tumor is most prominent in the flank and iliac fossa. If extending to the right hypochondriac region, it *does not* move with the liver.
The duration is indefinite and uremia rare.	The duration is short; a termination in uremia is common.

HYDATID CYST.	PLEURISY WITH EFFUSION.
The onset is slow; pain and fever are absent.	The onset is sudden, and violent pain is present, with fever and dyspnea.
The presence of a fluctuating mass in the hepatic area, *not changing with the position of the patient.* Hydatid fremitus is present, but no bulging of the intercostal spaces.	The presence of effusion, beginning at the base of the chest and gradually extending upward—*changing with the position of the patient* and accompanied by bulging of the intercostal spaces.
Aspiration reveals a clear yellow liquid of low specific gravity, containing no albumin, but chlorids and hooklets.	Aspiration gives a cloudy, turbid liquid, containing albumin and flakes of lymph with high specific gravity.
The disease invariably runs a chronic course.	The disease generally runs an acute course.

For a differential diagnosis from (*d*), (*e*), (*f*), and (*g*) I would refer the reader to the discussion of the several diseases (*vide* Diseases of the Liver).

Echinococcus of the Respiratory Organs.—The lung has been the seat of the larvæ quite frequently, and instances have been noted especially in North Germany and Australia. The right lower lobe has been the seat of predilection, though sometimes the pleura is the primary source of trouble. There are pain in the chest, cough, dypsnea, perhaps arching of the overhanging thoracic region, signs of a pleural effusion, a tympanitic note above the prominence, hemoptysis, and the pathognomonic expectoration of hydatid disease. The general condition may not be seriously affected. Perforation into the pleural sac by pulmonary echinococci may be followed by empyema, and, later, by perforation of the chest wall. The heart may be dislocated. Compression of the lung may produce gangrene.

The *diagnosis*, in the absence of the characteristic sputum, is to be made from phthisis and a pleural effusion. Their location at the base of the chest may serve to differentiate hydatid cysts from phthisis, as well as the absence of marked emaciation. The characteristic curved upper boundary of dulness in pleural effusion and the change of the boundary upon changing the patient's position will serve to distinguish this affection. Puncture of any bulging area will determine the character of the liquid. Pleural echinococci sometimes cause great compression of the lung and a barrelling of the chest on one or both sides. The pain may be quite sharp, and the respiratory murmur either distant or altogether absent.

Echinococcus of the Mediastinum.—Hare has collected 6 cases of hydatid disease among 520 cases of mediastinal tumors.

Echinococcus of the Heart.—Since most of the cases have shown involvement principally of the right side of the heart, the instances of sudden death that have been reported may be readily understood.

Echinococcus of the brain and spinal cord should not be confounded with cystic degeneration of the choroid plexuses. The symptoms of cerebral hydatids are those of tumor, peristent and intense cephalalgia, vomiting, psychical disturbances, convulsions, amblyopia, and "choked disk," and sometimes paralysis. Hydatid disease may develop inside the dura mater, or it may penetrate from without and destroy the vertebræ before they compress the cord to a great degree. The symptoms are those of a compression myelitis.

Echinococcus of the Spleen.—About 40 cases of involvement of the spleen have been described. The organ may become greatly enlarged and be mistaken for that due to malaria, leukemia, etc. The hydatid thrill may be detected.

Echinococcus of the Kidneys.—More than 100 cases have been observed, mostly in Germany and France. The cyst may be as large as in hydronephrosis. Many of the cysts are of the exogenous form of growth. As a rule, one kidney only is affected, and generally the left one. Abdominal and thoracic compression symptoms may be caused, and bulging is often present in the lumbar region in marked cases. This may be punctured as an aid in the diagnosis. Rupture into the pelvis of the kidney and the discharge of the smaller cysts may give rise to renal colic and to the discharge of the cysts with the urine. More rarely, rupture of a suppurating cyst may take place in the loin.

Echinococcus of the peritoneum is rare as a primary condition. Echinococci have also been located in the bladder, prostate, testicle, ovary, uterus, great omentum, mesentery, pancreas, arteries, lymphatics, thyroid gland, muscles, bones, joints, parotid gland, orbit, and mamma.

A **multilocular echinococcus cyst** may give rise to a very large, fluctuating, bossellated tumor below the liver; this may simulate colloid cancer, either of the liver or the gall-bladder. Icterus, marked and obstinate, with or without ascites, an enlarged spleen, and a long course without decided loss of flesh, are indicative of this form of hydatid. Fatal hemorrhage may supervene.

A peculiar **complication** of echinococcus cysts is the occasional development of urticaria. It has been noted especially shortly after the puncture of a cyst, and this is somewhat diagnostic when it appears.

The **prognosis** is generally grave both as to life and cure, although some cases of hydatid disease of the liver have lasted for more than ten years.

The character of the changes in the cysts and their mode of termination influence the prognosis. Thus, the occurrence of suppuration is to be dreaded. Spontaneous cures have been noted in a few instances.

Treatment.—As in most of the other parasitic diseases, prevention is more or less effectual, and a cure is difficult or impossible. Infection of the dog should be avoided by preventing its gaining access to possible sources of hydatid disease, as the raw flesh of animals, especially in the form of meat-scraps around slaughter-houses. In order that human beings may not be affected, dogs should not be carelessly handled or allowed to be where they may come in contact with food and drink in any way, whether meat or eggs, vegetables, fruits, or cereals. Cleanliness in keeping dogs and in the proper preparation of food are essential in regions where hydatid disease is prevalent.

Medicines cannot reach the parasites in man, situated as they are in larval form encysted in the various tissues and organs of the body. Whenever the cyst becomes large, accessible, and the cause of troublesome symptoms, surgical measures may be resorted to. Among these are, simple tapping, tapping with aspiration, and with the subsequent injection of various substances (as iodin and zinc-chlorid electrolysis), and incision with drainage. Excision of the liver cysts has been practised by Raggi, Pozzi, Tansini, and others, but its practical value is still undetermined.

TÆNIÆ OR TAPE-WORMS.

Natural History.—Tape-worms are found in the intestine of man, and are the matured or completely developed larvæ or cysticerci from the muscles and solid viscera of animals. Different varieties of cysticerci develop from the ova of the respective varieties of teniæ. These tape-worm eggs, after having passed out of the bowel, may be taken into the systems of various animals by various modes, entering the circulation, it may be, and becoming fixed within the solid tissues, especially the muscles. In about two or three months pea-sized cysts develop, and from the cyst-walls there gradually forms a new tenia-head, called a *scolex*, or nurse. The worm-cysts, popularly termed "measles," constitute the cysticerci. Remaining in the tissues, they die and become calcified in from three to six years (Strümpell). But, if taken into the stomach by the eating of raw or partially-cooked meat, a tape-worm develops from the scolex. The maturation of the segments of the tape-worm commences several months after the fixation of the scolex in the intestine. In the natural life-cycle of a tape-worm the usual order of lodgement may be reversed. Thus man instead of a lower animal may become the host of the tenia eggs, which in turn may find their way into the solid viscera and muscles to develop into cysticerci. Again, this same order may in some way be brought about by "auto-infection." The tape-worm, as its name indicates, has a ribbon-like form; although it has a number of segments and joints, giving it a link-belt appearance. When matured, these segments, or *proglottides*, develop male and female generative organs.

Varieties.—**Tænia Solium** (*Pork Tape-worm*).—This worm is seen much less frequently here than in Europe. It develops in the small intestine after the ingestion of raw or underdone "measly" pork. This worm does not necessarily exist singly, as its name would indicate, although such is usually the case. It ranges from 2 to 4 meters (6 to 13 feet) in length. The head is rounded, pin-head in size, and is succeeded by a thread-like neck and by gradually shortening and widening segments. Four suckers and a projecting circle of twenty-six long and short hooklets arm the head of the tenia. There may be as many as 800 segments. The mature ones become detached continuously, and are passed with the feces, several, as a rule, occurring together, and not singly, as in the case of tænia saginata. They are about 1 centimeter ($\frac{2}{5}$ in.) in length and from 6 to 8 millimeters ($\frac{1}{4}-\frac{1}{3}$ in.) in breadth, and about 1 meter (39.36 in.) from the head they are "approximately quadrilateral" in shape. These proglottides are bisexual. The female matrix occupies the middle of each proglottis, and is provided with from eight to fourteen irregular, tree-like branches on each side. The male generative organs are small vesicles in the anterior portion of the segment. The sexual opening is situated on one side, near the middle. The ovarian or uterine apparatus of a mature segment contains myriads of thick-shelled eggs, each one of which has an embryo with six hooklets.

Tænia Mediocanellata (*Saginata*).—The beef tape-worm is sometimes called the "unarmed tape-worm," since the head possesses sucking disks, but no hooklets. It is more common in this country and even in some of the European nations, as England. Longer than the tenia solium, being 4 to 6 meters (12 to 20 feet) in length, its segments are also thicker and larger, measuring from 16 to 18 mm. ($\frac{2}{3}$ in.) long, and from 8 to 10 mm. ($\frac{1}{3}$ in.) broad. The head of the worm as well as the ripe ovum is also slightly larger and proportionately thicker. The ovarian branches are more numerous (eighteen to thirty in number) and divide more dichotomously than those of tenia solium. Proglottides are also found in the stools, where they sometimes exhibit a crawling motion that has caused them to be mistaken for individual parasites. Cysticercus saginata has never been observed in man.

Bothriocephalus latus (*Fish tape-worm*, *Tænia lata*) occurs most commonly in Russia, Switzerland, Holland, and the German Baltic provinces. It is the longest cestode, measuring from 6 to 10 meters (20 to 30 feet). The head is club-shaped, unarmed, and has two lateral longitudinal grooves as suckers. The segments may be distinguished from those of the preceding varieties named by their marked breadth and shortness, also by the centrally situated, tortuous ovarian rosette, and the sexual orifice near the center of the abdominal surface of each proglottis. The ova are larger than those of the pork and beef tape-worms, though thinner-shelled and with a sort of lid at one end. They develop only in fresh water. From them is formed an embryo with vibrating cilia and six hooklets. Pike and other fish swallow these embryos, which develop into cysticerci in the muscles, peritoneum, and solid viscera. The eating of measly fish, raw or partially cooked, thus favors the development of this tape-worm in the human intestine.

Symptoms.—Contrary to what has been supposed in days gone by, there are no absolutely diagnostic symptoms of the presence of tape-worm that can be relied upon. Indeed, the existence of a tape-worm in the bowel may not be suspected even because of the total absence of indicative, subjective sensations. On the other hand, teniæ may cause considerable local distress and impairment of the general health. Because of this fact a knowledge of the existence of tape-worm in certain neurotic subjects leads to an inordinate description of symptoms that exist mainly in the workings of a morbid imagination.

Alimentary symptoms of tape-worm may be as follows: anorexia alternating with a voracious appetite, constipation alternating with diarrhea, colicky pains in the abdomen, indigestion, nausea, and vomiting, and sometimes salivation.

General symptoms of the teniæ may be added, as lassitude, inappetence, mental uneasiness, worry and irritability, depression of spirits, some physical prostration, and even emaciation. Various *reflex symptoms*, such as pruritus of the nose and anus, vertigo, migrain, tinnitus aurium, palpitation, visual disturbances (even temporary amaurosis), dilatation of the pupils, choreic movements, and epileptiform convulsions have been attributed to these parasites. But, on careful inquiry, adequate causes for some of these symptoms may be found in other associated morbid conditions.

Diagnosis.—This is always to be made by the discovery of tenia segments or ova in the underclothing or stools. The doubtful presence of suspected tape-worm may be cleared by the administration of a suitable purgative, which will usually suffice to bring away portions of the worm in the dejections. I would here add a special warning lest mucous casts or shreds or vegetable structures (as of onion) be mistaken for tape-worm.

The diagnosis of the variety of the tape-worm may also be made by a careful scrutiny of the segments. Those of the tenia saginata are larger and fatter than, and their generative apparatus is unlike that, of tenia solium (*vide supra*).

Hypochondriasis can be excluded by repeated examinations of the stools, especially after the exhibition of cathartics, and by the uniform failure to detect portions of tape-worm or tenia eggs.

The **prognosis** is favorable. Indeed, teniæ may exist at all ages and for years without any danger to the patient.

Treatment.—*Prophylaxis.*—The way to avoid acquiring a tape-worm is to use none but well-cooked meats; this applies to beef and pork in particular. The use of pure drinking-water is of no little importance also. The proglottides of the tenia should always be burned, and not thrown where they may be taken into the bodies of other animals, as the cow or hog, and then be allowed to propagate. Governmental inspection of the meat-supply in abattoirs should be rigidly carried out in all parts of the country.

Curative.—Before administering the chosen anthelmintic, the patient needs to undergo a "preparatory treatment." This has for its object the starvation of the parasite, so as to weaken, if possible, its hold upon the intestinal mucosa. This is specially necessary in the case of tenia solium, in which the cephalic hooklets are obstinately and firmly fixed to the membrane, and since a cure cannot be said to have been effected unless the head be dislodged with the dejecta. For about two days prior to giving the remedy the patient should be restricted in diet to milk, light soups, a little white bread, and the like. Meanwhile, the bowels should be purged gently once or twice, after a simple enema, to clear away accumulated fecal masses that might prevent the easy discharge of the worm.

In the evening preceding the day on which the drug is to be exhibited, a saline cathartic should be given to empty the bowel of fecal matter as completely as possible. The following morning no breakfast should be allowed, and before noon the selected anthelmintic should then be administered. Some authors assert that if the worm does not come away in a few hours, and an intense sense of pressure is felt in the abdomen, a brisk purge is indicated. To make assurance doubly sure, and if the patient be not too weak, it might be well to order a cathartic as routine practice, within a few hours at the latest.

There are several very efficacious anthelmintic drugs to choose from. Prominent among them is male fern. Given to an adult in doses of ½ to 1 dram (2.0–4.0) of the ethereal extract, and followed in several hours by a calomel and a saline purge, it usually succeeds in bringing away the tenia. Another valuable remedy is pelletierin, the active principle of pomegranate; the tannate may be prescribed, dose 1 to 1.5 gm, in capsules; or, a decoction of the pomegranate bark may be used, in combination with male fern, as in the Leipsic formula (Strümpell):

℞. Granati radicis corticis,	℥iv–v (128.0–160.0);
Aquæ,	Oij (1 liter).
Mix and macerate for twenty-four hours, and boil until reduced to	f℥v (148.0).
Add: Oleoresinæ aspidii,	ʒj (4.0).

Sig. To be taken in three or four doses, at short intervals.

Pepo in emulsion or in a sugary paste (about two ounces—64.0—and deprived of the envelopes) is at once a useful and harmless remedy.

Another effective vermifuge is kousso (Brayera anthelmintica). An infusion of half an ounce (16.0) of the flowers to one pint of water and mucilage of acacia is made, a wineglassful of which may be taken every half hour. The Germans recommend sometimes the agreeable, though more expensive, Rosenthal's "kousso tablets." Enough of these to make 15 grains (0.972) may be taken within one hour, with *café noir* or lemonade. Koussin (the active principle) in doses of 30 to 40 grains (1.94–2.592) has also been recommended, but should not be given to pregnant women, as abortion may be produced. Among other remedies of value as vermifuges may be mentioned kamala (1 to 3 drams—4.0–12.0—of the powder and hairs, in wine or water), oil of turpentine (½ to 2 ounces—16.0–64.0—in emulsion or milk), and thymol. The combined use of such drastics as croton oil renders the action of the anthelmintic drug more certain at times.

Although the head of the tenia may not be detected in the stools along with the body of the worm (and such is usually the case), a cure usually follows nevertheless, since, on account of its smallness, it may easily escape notice, and also from the fact that the head often dies and thus loses its hold upon the membrane, being carried away with the feces. On the other hand, if after the lapse of several months from the removal of a tape-worm, segments again appear in the stools, it may be inferred that the head was not dislodged or that another worm has developed. In cases where the tenia seems to redevelop with remarkable frequency and obstinacy it may happen that the head and neck are well protected beneath one of the valvulæ conniventes.

After the removal of the tape-worm—a weakening procedure, as a rule—the condition calls for supportive measures. The diet should not be too heavy for a time, but nutritious and easily digestible.

TÆNIA NANA.

This is the smallest tape-worm in man (v. Siebold). It varies from 8 to 20 mm. ($\frac{1}{3}$–$\frac{4}{5}$ in.) in length and from 0.5 to 0.7 mm. ($\frac{1}{50}$ in.) in width. The head has four suckers, a rostellum, and hooklets. The segments are yellowish, short, and broad. It is believed by some observers that, occurring in children, as it commonly does, this parasite is the cause of *epileptiform convulsions* and *enuresis nocturna*. Thousands of ova may be found within a cubic centimeter of fecal matter.

TÆNIA CUCUMERINA.

(*Elliptica; Canina.*)

A small reddish tape-worm found frequently in the intestines of the dog. It is 10 to 40 cm. (4–16 in.) in length. The larvæ or cysticerci develop in the louse or flea of the dog or cat. The parasite is more common in children than in adults, owing to the intimate relation of the former with the last-named pet animals.

TÆNIA FLAVOPUNCTATA.

(*Tænia Diminuta; Tænia Leptocephala.*)

Tænia diminuta is a very small cestode, 20 to 60 mm. ($\frac{4}{5}$–$2\frac{1}{2}$ in.) in length, with a small club-shaped head and nearly a thousand segments. The cysticerci inhabit such insects as the *asopia famialis* (caterpillar and cocoon); the *anisolabis annuli* (belonging to the orthoptera); and the coleoptera *axis spinosa* and *scaurus striatus*. Man has been infected a number of times, probably by taking food containing these infested insects.

Tænia Madagascariensis and *Tænia serrata* are other forms rarely found in man.

PARASITIC ARACHNIDA.

Pentastoma Tenioides.—This parasite in its adult form is an inhabitant of the nasal fossæ of the dog or horse, though it may also occur in man both in this and in the larval form. The ova are ejected during sneezing, and are then ingested by man. The larvæ are found in the liver, lungs, and kidneys.

Sarcoptes (*Acarus Scabiei*).—This insect produces the skin affection known as "the itch," or *scabies*, an affection more common in Europe than in America, where it constitutes only about 4 or 5 per cent. of all cases of skin disease. It is most prevalent among the poor and the unclean. The female is visible to the naked eye, and is about 0.5 mm. ($\frac{1}{50}$ in.) in length; the male is about 0.25 mm. ($\frac{1}{100}$ in.). Both are nearly as broad as they are long.

The parasite penetrates the skin and lives in a burrow or *cuniculus* that it makes for itself. The female lives in the end of the burrow, which may contain a number of ova, and appears as a minute, brownish-black, dotted, sinuous line, situated chiefly in the cutaneous folds, where the skin is mostly delicate, as between the fingers. Secondary skin lesions, due to scratching, are common. Sulphur ointment, well rubbed in after hot bathing, is usually quite efficacious.

Sarcoptes scabiei hominis is a variety of the preceding that infests other animals (cat, dog, cow, horse, wolf, goat, camel, etc.). Occasionally it may gain an entrance into man's skin, but dies simultaneously in the human host, although many invasions may occur.

Leptus Autumnalis (*Harvest Bug*).—The most common of several variation is a mite of a reddish color, having six legs armed with claws and sharp mandibles. It arises among low bushes and thus appears about the ankles and legs. It partially penetrates the skin, boring only far enough with its short, thick head to procure nourishment. Artificial dermatitis may be produced by the irritation of scratching. Mercury, sulphur, and naphthol ointments suffice to destroy the parasite.

Demodex Folliculorum (*Comedo Mite*).—This minute parasite may be expressed from swollen sebaceous follicles of the nose, cheek, and other parts of the face. It has a worm-like body with very short legs, and is only about 0.2 to 0.4 mm. ($\frac{1}{60}$ in.) in length. It is not known to produce acne, as was formerly supposed.

OTHER PARASITIC INSECTS.

PEDICULOSIS.

(*Phthiriasis.*)

Lice or **pediculi** live on and attack the skin. Three forms are found on man: *pediculus capitis*, *pediculus corporis*, and *pediculus pubis*.

The *pediculus capitis* is whitish or grayish in color, about 1 mm. ($\frac{1}{25}$ in.) long (male), and has six legs under the front part of the body. The oviparous female is nearly twice as long as the male, and lays from fifty to eighty eggs on the hairs within a week. These ova, or "nits," mature in from three to eight days. Itching is the most prominent symptom, and an eczematous eruption above and behind the ears and in the neck is often associated. "Plica polonica" was a phrase once used to designate the matted condition of the hair in extremely dirty, greasy, and long neglected cases of head-lice.

Pediculus Vestimentorum (*Corporis*).—This louse inhabits more often the clothing than the body itself. It is larger than the head louse, and, like the latter, moves slowly. The nits are found with difficulty on the fibers of the underclothing. It sucks blood through a proboscis inserted into the sweat pores, and after withdrawing leaves a minute hemorrhagic speck. Irritation of the skin is produced, and in old cases, as in dirty tramps (the great unwashed class), the skin becomes scaly and often pigmented (vagabond's disease). The effects of scratching are almost frantic, and after a cure is effected parallel white lines, the remains of scratch-marks, followed by atrophic changes, may be visible, as in a case that I observed.

Pediculus or Phthirius Pubis (*Crab-louse*).—This parasite is not limited to the pubis, but is also found in the hairy region of the axilla, on the chest, and may even invade the beard and eyebrows. It clings firmly to the [illegible] [illegible] six legs with strong claws are placed [illegible] the anterior part of the [illegible] body.

[illegible] The hair should be cut short where the head-lice are nits are abundant. Saturating the hair and scalp with kerosene oil for twenty-four hours usually kills the parasites. *Body-lice* may be destroyed by scalding the underclothing and hot-ironing carefully about the seams. A hot soap-and-water bath is sufficient for the body, and sedative and antiseptic ointments may be useful adjuvants. Mercurial and beta-naphthol unguents usually suffice in treating for *pediculus pubis*. Prof. J. V. Shoemaker[1] affirms that naphthol is a remedy that seems to meet the indications presented by the three forms of the disease; he prepares it as follows:

℞. Beta-naphtol, ʒj (4.0);
Cologne water, f℥iv–vi (120.0–178.0).—M.

Cimex Lectularius or Bed-bug.—This too well-known parasite is flat, brownish-red in color, and from 2 to 5 mm. ($\frac{1}{12}$–$\frac{1}{5}$ in.) in length. It infests beds and public vehicles, emitting a disagreeable odor. It is a blood-sucker, and causes considerable itching, local irritation, and urticaria even in some persons, while others are unmindful of their attacks. Sulphur fumigation and mercuric chlorid applications to the harboring places of the bed-bugs are effectual destructive agents. Saturated sodium bicarbonate solution will relieve the burning and itching.

Pulex Irritans (*Common Flea*).—This "ubiquitous" parasite is from 2 to 4 mm. ($\frac{1}{12}$–$\frac{1}{6}$ in.) in length, black or (when filled with blood) brownish-red in color, having six legs, the hind ones of which are relatively very large and powerful, enabling it to jump many times its own height. A flea's bite causes a sharp sting, and leaves a slightly raised red spot. Treatment is the same as for the preceding insect.

Pulex Penetrans ("*Jigger*").—This parasite, also called "sand-flea," is indigenous to the West Indies, South America, and the Southern States. The impregnated female penetrates the skin, and especially that of the feet, for purposes of ovulation. As the distention with the eggs occurs, swelling, pain, and even ulceration may appear. The sand-flea is a small, egg-shaped insect, about half the size of an ordinary flea, brownish in color, and exceedingly resistant to crushing force. *Prophylaxis* in regard to foot-wear is necessary. Essential and antiseptic oils may also be put on the feet or stockings.

Ixodes (*Wood-tick*).—There are several varieties of tick- or wood-louse that may attack the human skin, among which *ixodes albipictus* is supposed to be the most common. *Ixodes ricinus* and *ixodes bovis* are found on horses and cattle. They are blood-suckers, adhering to the skin very firmly, and wheals may be produced by them. A drop of turpentine, or of some such essential oil as anise or rosemary, will cause them to loosen their hold.

Dermanyssus Avium et Gallinæ.—These bird- and fowl-insects are small and grayish-white in color, and may attack the human skin and cause eczematous eruptions, owing to the scratching induced by the irritation.

Culicidæ (*Mosquitoes and Gnats*).—The blood-sucking *mosquito* (*culex auxifer*), so well known, may also transfer to human beings the filaria sanguinis hominis and perhaps the plasmodium malariæ.

The *gnat* (*culex pipiens*) is very troublesome during certain seasons, particularly along water-courses and in wooded districts. Its bite is quick, sharp, and stinging.

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[1] *A Practical Treatise on Diseases of the Skin*, p. 849.

The **hirudo** (leech) is a parasite that sometimes attaches itself to bathers. In the tropics it has been known to cause severe bites and inflammation.

The bites and stings of bees, wasps, spiders, and ants have been known to cause considerable inflammation, edema, and blood-poisoning.

Estridæ (*Bot-flies*).—These may become parasitic in man in the larval form. Species of the *hydoxerma* and *dermatobia*, that infest the skin of the horse, ox, goat, etc., have also been observed among the Central and South American Indians. They burrow beneath the skin of the abdomen, scrotum, and other regions.

Muscidæ (*Common Flies*).—Common flies affect the skin of man by depositing eggs in wounds. The ova hatch within twenty-four hours sometimes, and the dipterous larvæ may swarm to make the so-called "living" wound or sore (*myiasis vulnerum*). The larvæ or maggots do not penetrate the tissues, however. The principal flies that infest wounds are the flesh-fly (*sarcophila carnaria*), the blow-fly (*calliphora vomitoria*), the screw-worm fly (*compsomyia macellaria*), and the ordinary house-fly (*musca domestica*).

Internal myiasis may also be caused by swallowing the ova of these flies. The larvæ may thus be vomited or defecated.

Epidemic urticaria is often caused by the migration of the caterpillar (*cuethocampa*). Among other parasites that attack man and inhabit particular regions are the following: The *simulium reptans*, or creeping gnat of Sweden; the *seroot-fly* (*zimb*) of Abyssinia; the *ixodes carapato*, a virulent bed-bug in Brazil; the *hæmatopota pluvialis* (Clegg) of the West Highlands.

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